Brigham and Women's Hospital
Founding Member, Mass General Brigham

Important New Trials In Glomerular Disease

David Bruce Mount MD, FRCPC
Associate/Clinical Chief,
Renal Division BWH
Assistant Professor HMS

David B. Mount, MD, FRCPC

University of Toronto Medical School
Medicine Residency @Toronto General Hospital, Toronto, Canada
Renal Fellowship @ Brigham and Women's Hospital (BWH)
Associate/Clinical Chief, Renal BWH
Co-Director, Glomerular Diseases Clinic BWH
Director of Dialysis Services, BWH
Physician, Renal Division VABHS

Research Focus: Urate transport
Clinical Focus: Glomerulonephritis, Electrolyte disorders, Gout, Consultative Nephrology

Financial Disclosures

Author, peer reviewer – UpToDate, McGraw Hill
Consultant/advisory boards
– Gout: Allena Pharmaceuticals, Horizon Pharma/Amgen, Alnylam Pharmaceuticals, ANI Pharmaceuticals, Shanton Pharma
– ANCA-associated vasculitis: Amgen

OBJECTIVES

- Understand the circumstances when a referral for a clinical trial is appropriate for glomerular diseases.
- Review some recently approved therapeutics for glomerular diseases.
- Mention some recently published and ongoing clinical trials for glomerular disease therapeutics.

IgA Nephropathy Epidemiology Analysis

377,829

7MM Prevalence in 2020

69,191

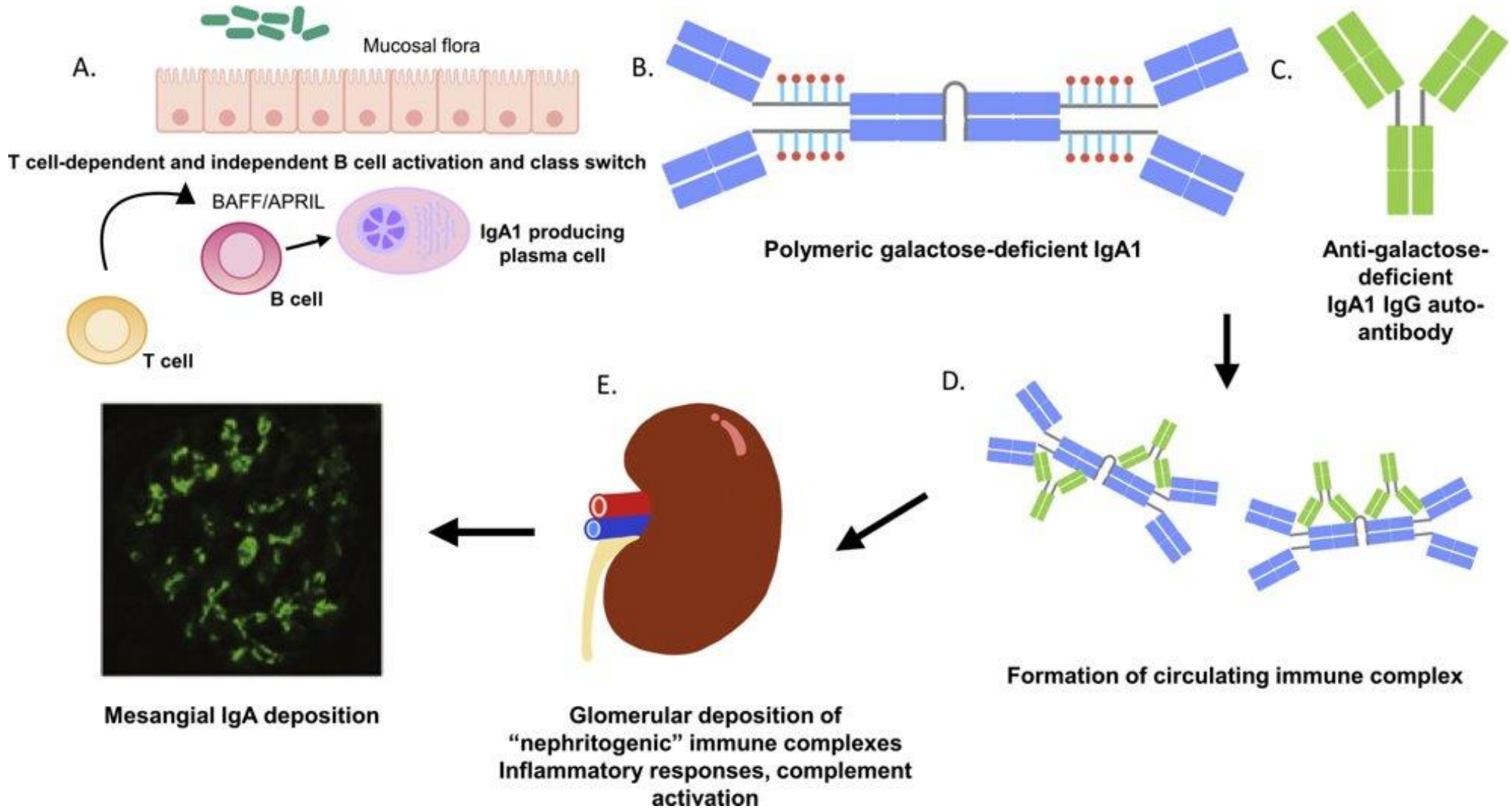
Prevalence in Males in the US in 2020

58,169

Prevalence in Females in the US in 2020

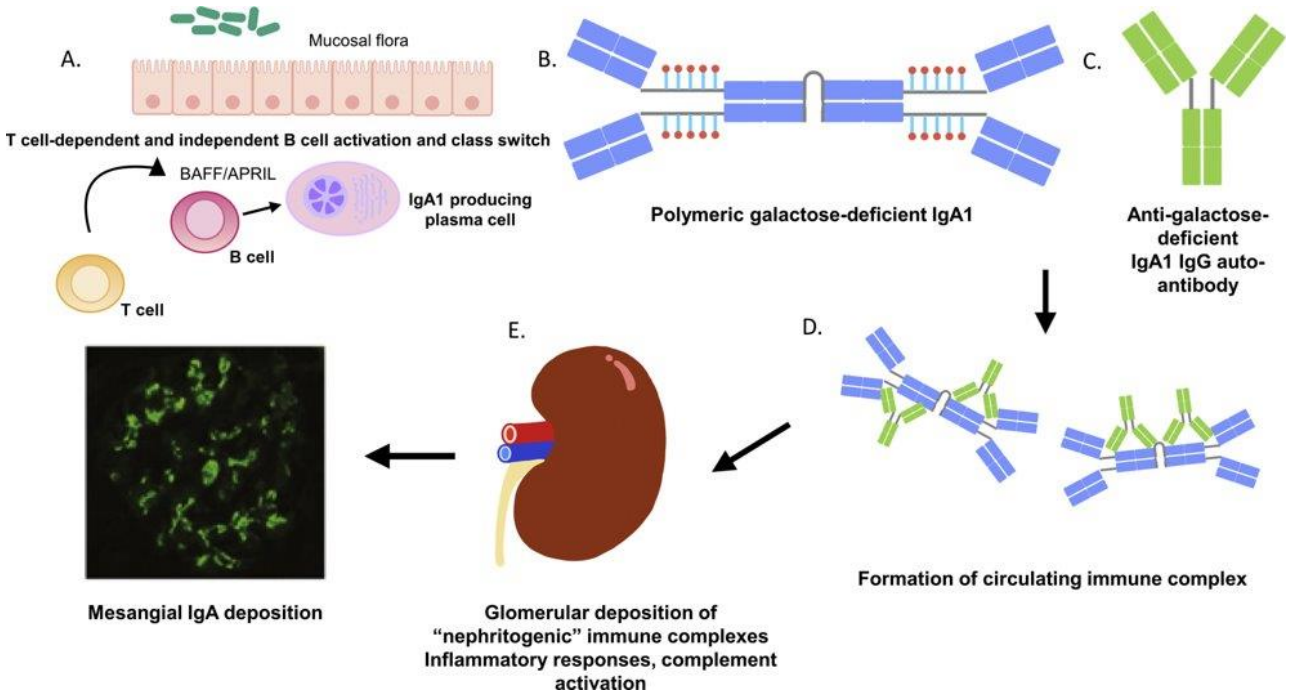
Source: IgA Nephropathy Epidemiology Forecast to 2030

IgA Nephropathy pathogenesis is complex.



American Journal of Kidney Diseases 2021 78429-441

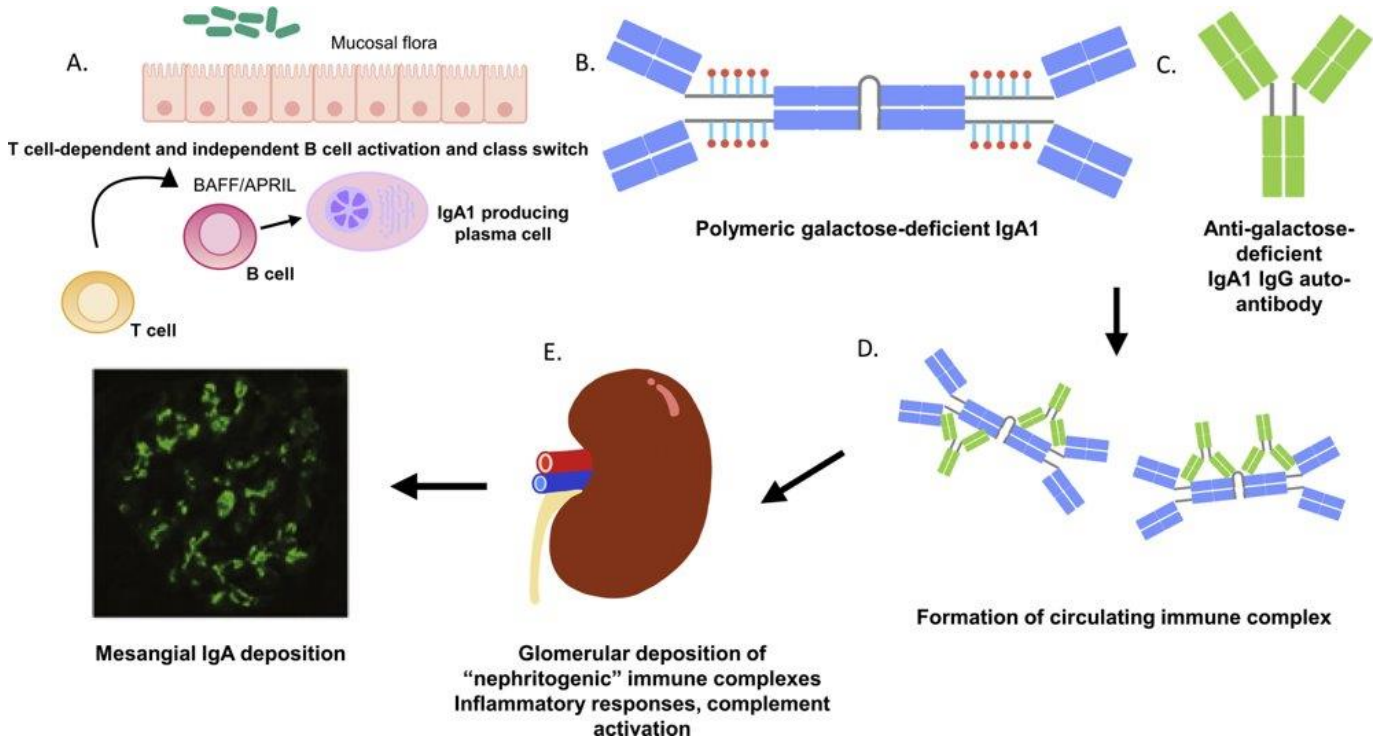
IgA Nephropathy pathogenesis is complex.



1st Hit - Production of poorly glycosylated IgA1 (Gd-IgA1).

American Journal of Kidney Diseases 2021 78429-441

IgA Nephropathy pathogenesis is complex.

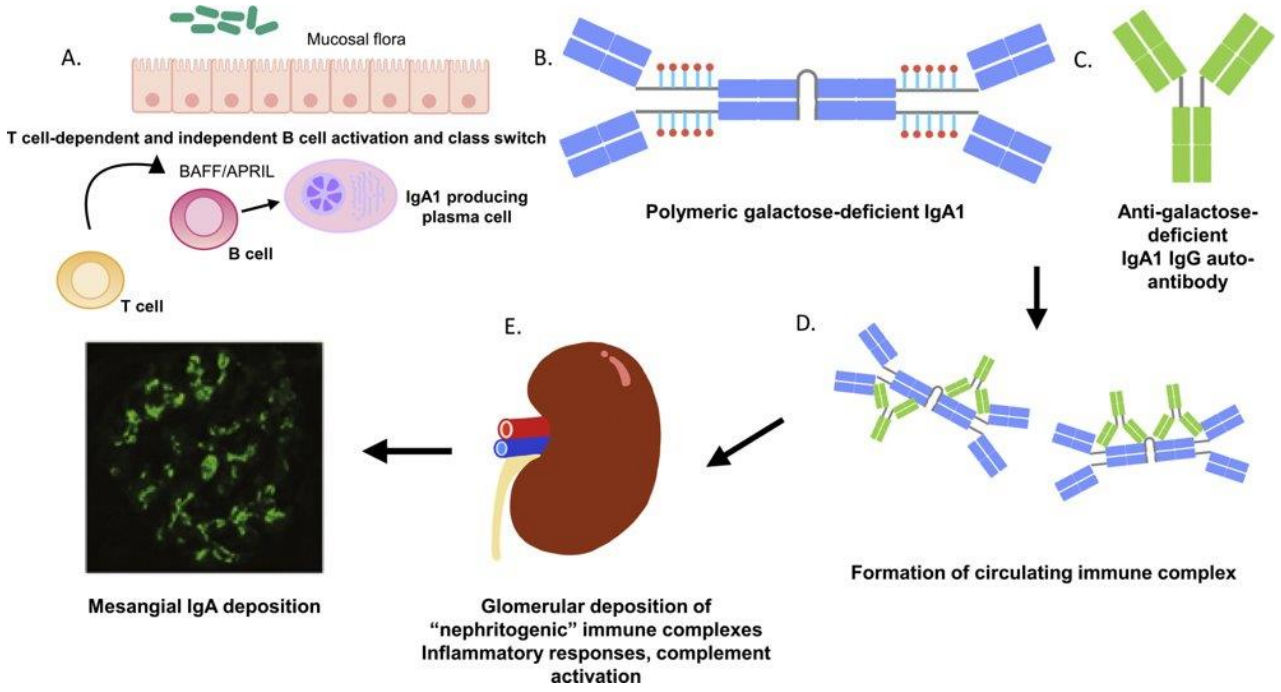


1st Hit - Production of poorly glycosylated IgA1 (Gd-IgA1).

2nd Hit - Production of anti-Gd-IgA1 antibodies (IgG or IgA).

American Journal of Kidney Diseases 2021 78429-441

IgA Nephropathy pathogenesis is complex.



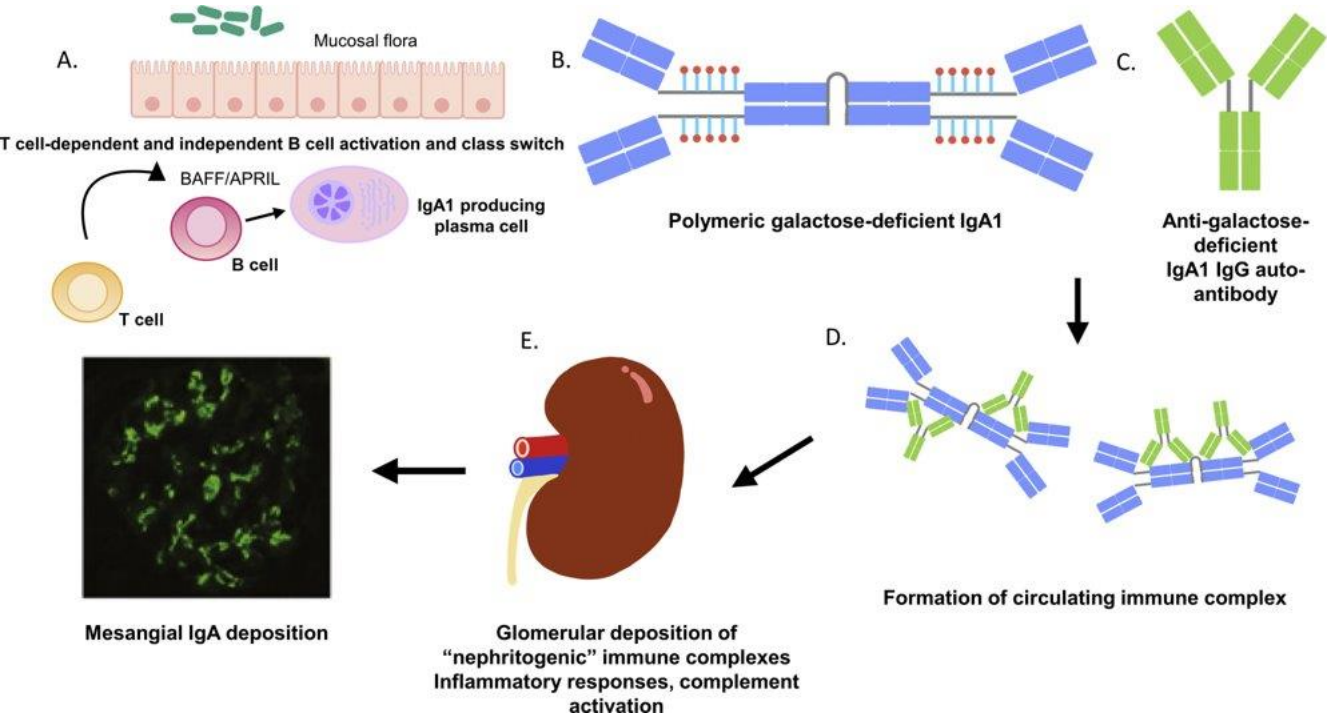
1st Hit - Production of poorly glycosylated IgA1 (Gd-IgA1).

2nd Hit - Production of anti-Gd-IgA1 antibodies (IgG or IgA).

3rd Hit - Formation of circulating immune complexes containing Gd-IgA1.

American Journal of Kidney Diseases 2021 78429-441

IgA Nephropathy pathogenesis is complex.



1st Hit - Production of poorly glycosylated IgA1 (Gd-IgA1).

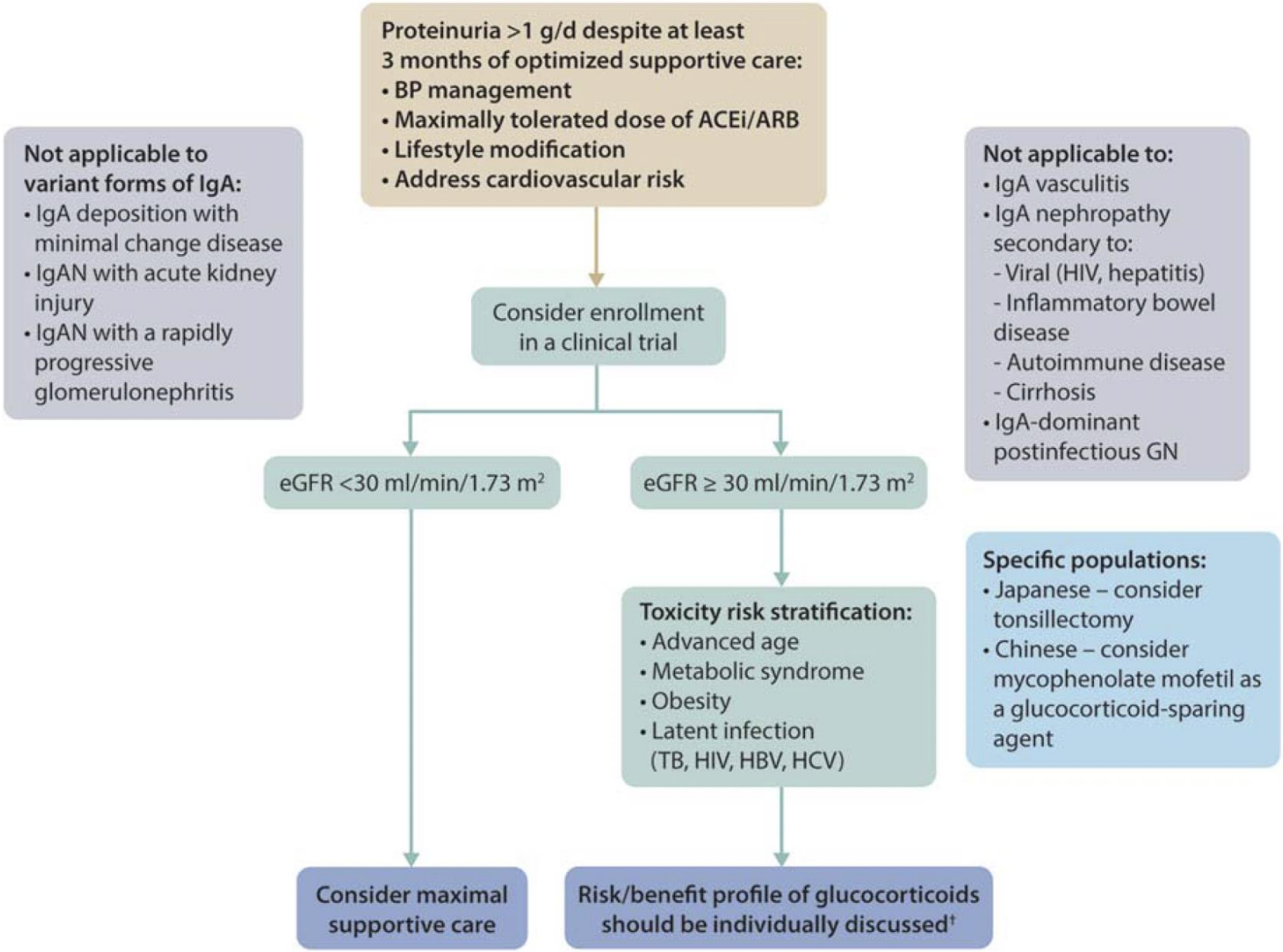
2nd Hit - Production of anti-Gd-IgA1 antibodies (IgG or IgA).

3rd Hit - Formation of circulating immune complexes containing Gd-IgA1.

4th Hit - Immune complex deposition and kidney injury

American Journal of Kidney Diseases 2021 78429-441DOI: (10.1053/j.ajkd.2021.01.024)

KDIGO includes consideration of trial enrollment upfront.



Recently completed and ongoing trials.

JAMA®

QUESTION What are the effects of oral glucocorticoids, compared with placebo, in patients with IgA nephropathy and proteinuria of 1 g per day or greater receiving optimal supportive therapy?

CONCLUSION Treatment with oral methylprednisolone significantly reduced the risk of the composite of kidney function decline, kidney failure, or death due to kidney disease in patients with IgA nephropathy, but the incidence of serious adverse events was increased.

POPULATION

305 Men
198 Women

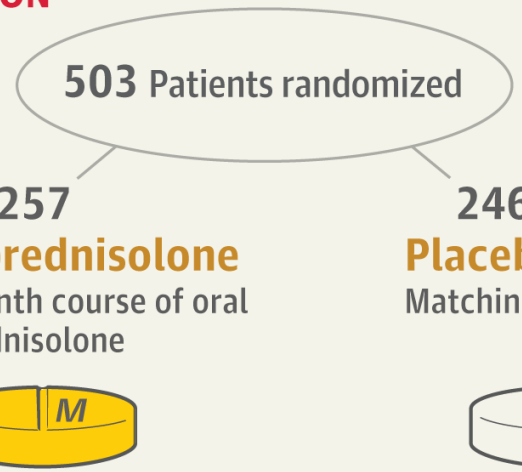
Adults with IgA nephropathy and proteinuria ≥1 g per day

Mean age: **38** years

LOCATIONS

67
Medical centers worldwide

INTERVENTION

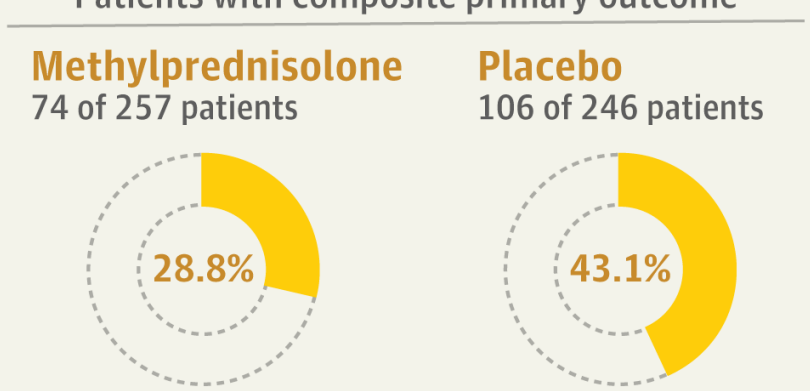


PRIMARY OUTCOME

Composite outcome of of the first occurrence of a sustained 40% decrease in estimated glomerular filtration rate, kidney failure, or death due to kidney disease

FINDINGS

Patients with composite primary outcome



The primary outcome occurred significantly less frequently in the methylprednisolone group:

Hazard ratio, 0.53
(95% CI, 0.39 to 0.72); $P < .001$

Absolute annual event rate difference, −4.8%
(95% CI, −8.0% to −1.6%)

Lv J, Wong MG, Hladunewich MA, et al; for the TESTING Study Group. Effect of oral methylprednisolone on decline in kidney function or kidney failure in patients with IgA nephropathy: the TESTING randomized clinical trial. *JAMA*. Published May 17, 2022. doi:10.1001/jama.2022.5368

Targeted-release formulation budesonide

- 201 patients with IgAN
- Targeted release budesonide (n = 97).
- Placebo (n = 102).
- Treatment for 9 months.
- All had optimized RAS blockade.
- Median proteinuria 1.26 g/g.
- 58% of patients had proteinuria ≥ 2g/d.
- Median eGFR: 55 mL/min/1.73 m^2

Kidney Int. 2023 Feb;103(2):391-402 [14]

Targeted-release formulation budesonide

- 201 patients with IgAN
- Targeted release budesonide (n = 97).
- Placebo (n = 102).
- Treatment for 9 months.
- All had optimized RAS blockade.
- Median proteinuria 1.26 g/g.
- 58% of patients had proteinuria ≥ 2g/d.
- Median eGFR: 55 mL/min/1.73 m^2

- At 9 months, UPCR was 27% lower compared with placebo, along with an eGFR preservation of 3.87 ml/min/1.73 m^2 difference versus placebo.
- TEAEs: 86.6% in Targeted release budesonide vs 73% in placebo. Mostly mild to moderate.
- No severe infections requiring hospitalization.

Kidney Int. 2023 Feb;103(2):391-402 [15]

JAMA Network Open

RCT: Effectiveness of Mycophenolate Mofetil Among Patients With Progressive IgA Nephropathy

POPULATION

94 Males, 76 Females

Patients with immunoglobin A (IgA) nephropathy with proteinuria ≥0.75 g/d

Mean age, 36.6 y

INTERVENTION

170 Patients randomized

85 Supportive care (SC) group
Blockade of renin-angiotensin system with losartan, blood pressure control, lifestyle change, and statin as needed

85 Mycophenolate mofetil (MMF) and SC
SC with oral MMF at 1.5 g/d for 12 mo, then 0.75 to 1.0 g/d for >6 mo

SETTINGS / LOCATIONS

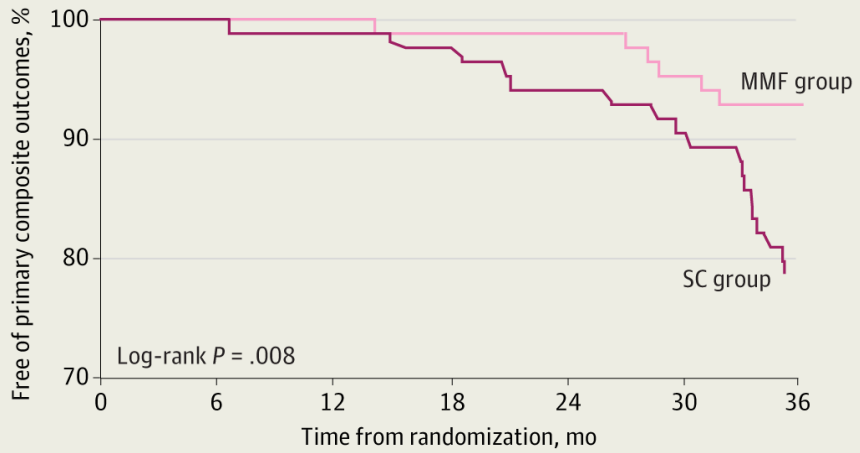
1 Kidney center in China

PRIMARY OUTCOME

A composite of doubling of serum creatinine, end-stage kidney disease (dialysis, transplant, kidney failure without kidney replacement therapy), or death due to kidney or cardiovascular cause, and progression of chronic kidney disease (CKD)

FINDINGS

Addition of MMF to SC, compared with SC alone, significantly reduced the risk of the primary composite outcome and delayed the progression of CKD



Composite kidney outcome, MMF group vs SC group:
7.1% vs 21.2%
aHR, 0.23; 95% CI, 0.09-0.63; *P* < .001

Progression of CKD, MMF group vs SC group:
8.2% vs 27.1%
aHR, 0.23; 95% CI, 0.10-0.57; *P* < .001

Hou FF, Xie D, Wang J, et al; MAIN Trial Investigators. Effectiveness of mycophenolate mofetil among patients with progressive IgA nephropathy: a randomized clinical trial. *JAMA Netw Open*. 2023;6(2):e2254054. doi:10.1001/jamanetworkopen.2022.54054

IgA Nephropathy Patient Baseline Characteristics in the Sparsentan PROTECT Study

ISN INTERNATIONAL SOCIETY OF NEPHROLOGY

Methods & cohort

Patient characteristics PROTECT study (NCT03762850)

Compared to contemporary IgAN phase 3 trials

IgA nephropathy

Proteinuria ≥ 1g/d Max tolerated ACEi &/or ARB

PROTECT study

RCT | Double blind

International | Multicenter

Active controlled

Sparsentan

Single-molecule endothelin angiotensin receptor antagonist

vs

Irbesartan

Active control

Patient characteristics

Patient number N = 404	Median age 46
Stage 3A CKD 35%	Baseline median 24-h urine protein 1.8g/d
Baseline mean BP 129/82	% on max ACEi or ARB 63.4%

Europe	Asia pacific	North America
53%	27%	20%

In Asian vs non-Asian regions; higher % of females, lower BPs & lower % baseline hypertension & related treatment

IgAN, IgA Nephropathy; ACEi angiotensin converting enzyme inhibitor; ARB, angiotensin receptor blocker

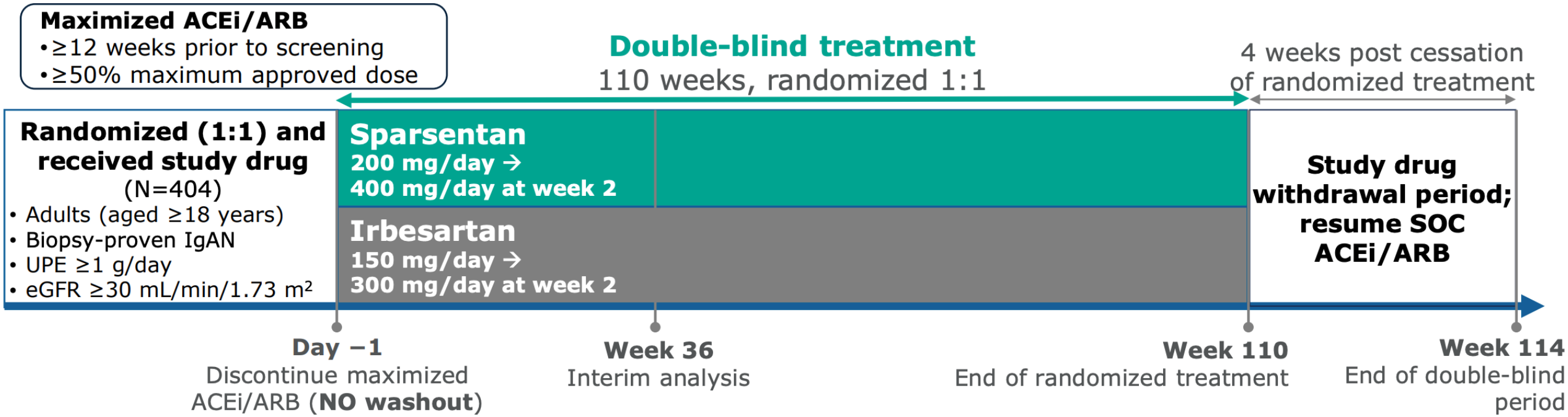
KI REPORTS
Kidney International Reports

Barratt J et al. 2023

Visual abstract by:
Sophia Ambruso, DO
@Sophia_kidney

Conclusion Patient enrollment in PROTECT, with differing racial backgrounds and across CKD stages, will allow for important characterization of the treatment effect of sparsentan in IgAN patients with proteinuria at high risk of kidney failure.

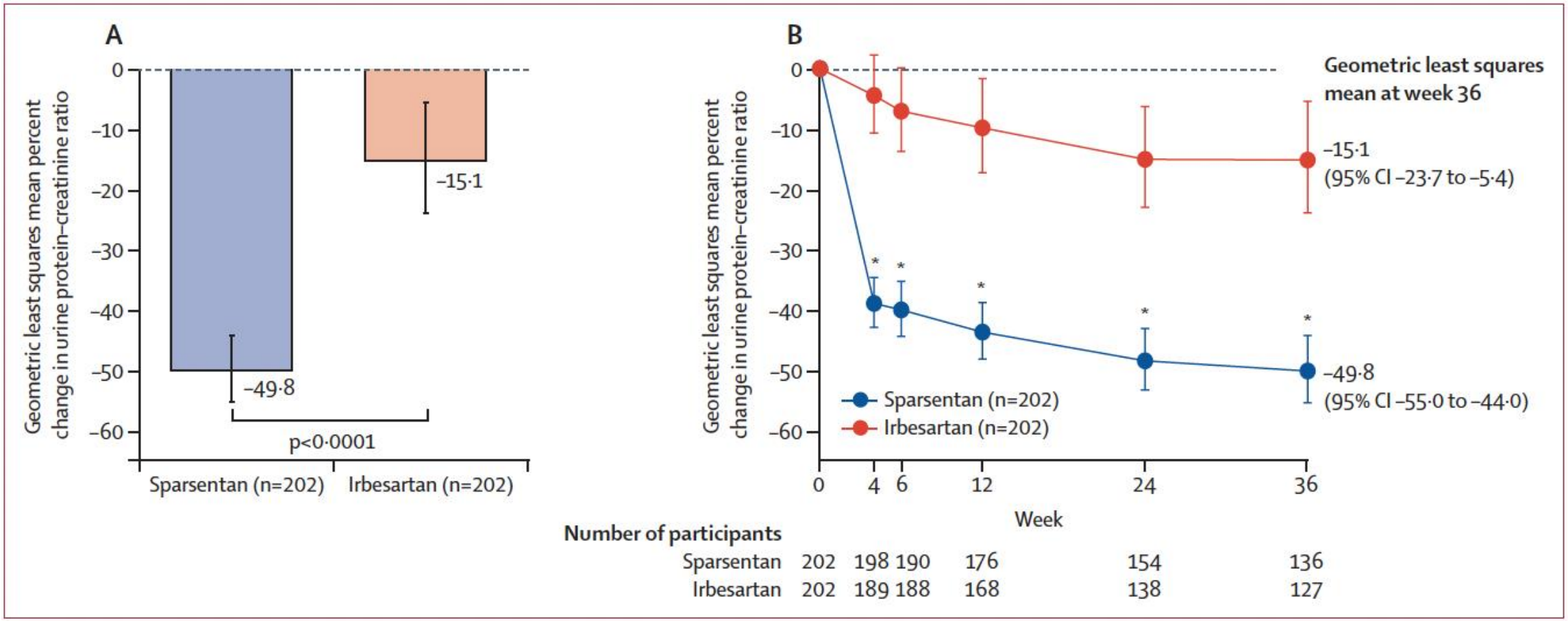
Trial* Design



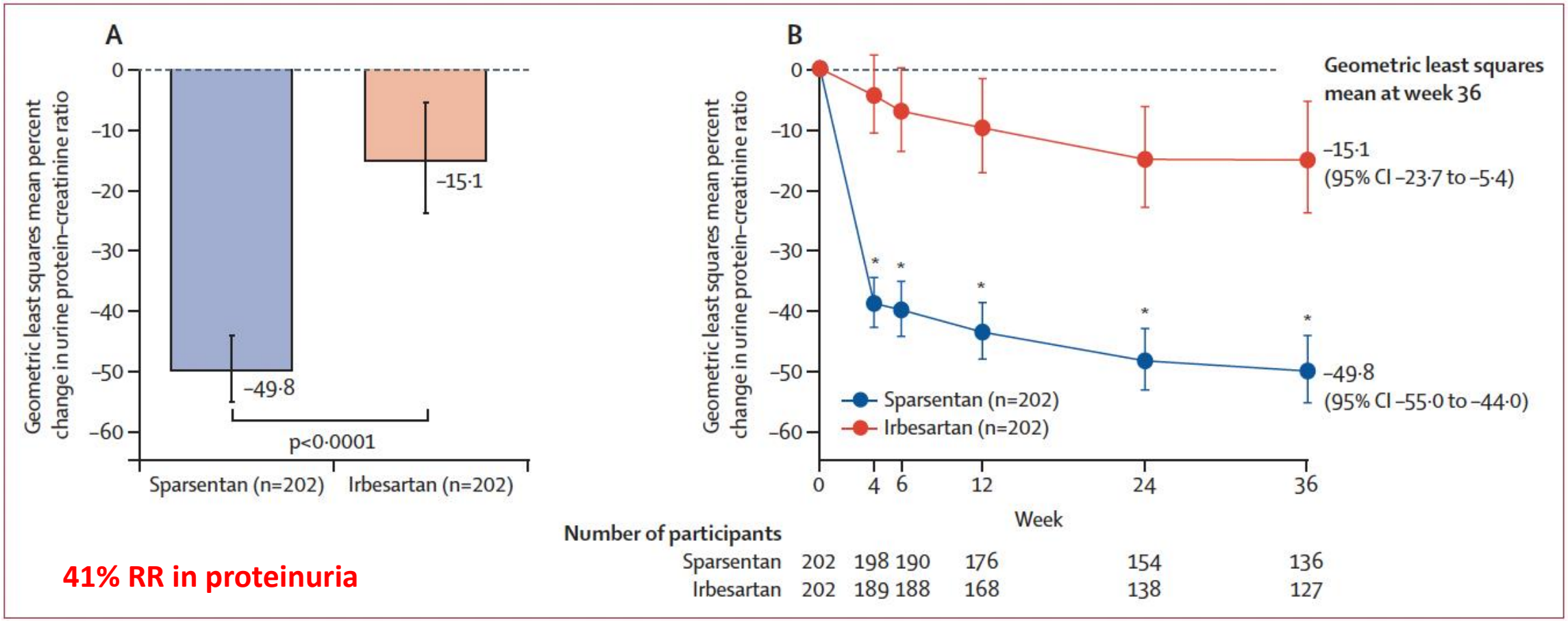
Primary Efficacy Endpoint
Change in UPCR from baseline to week 36

Key Secondary Efficacy Endpoint
eGFR slope: **chronic** (weeks 6-110) and **total** (day 1-week 110)

*NCT03762850

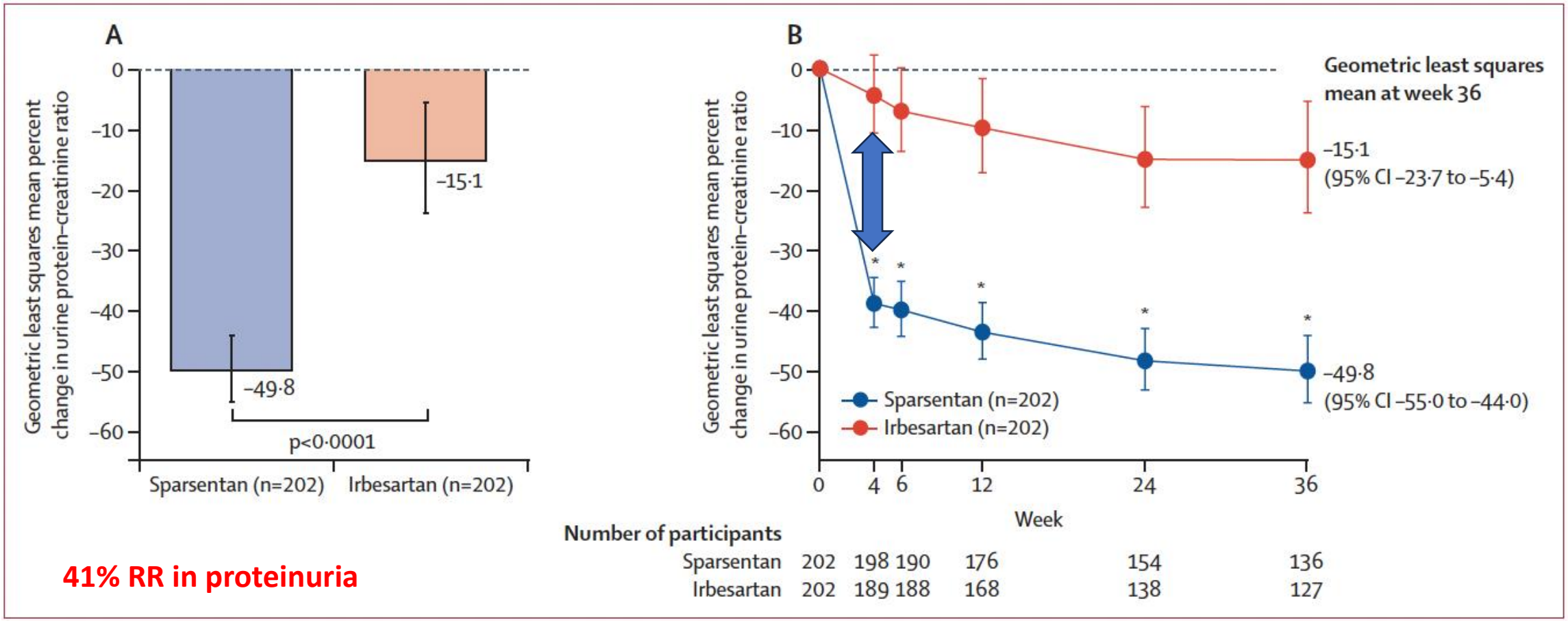


Heerspink et al, Lancet, 2023



41% RR in proteinuria

Heerspink et al, Lancet, 2023



41% RR in proteinuria

Heerspink et al, Lancet, 2023

The NEW ENGLAND JOURNAL of MEDICINE

ORIGINAL ARTICLE

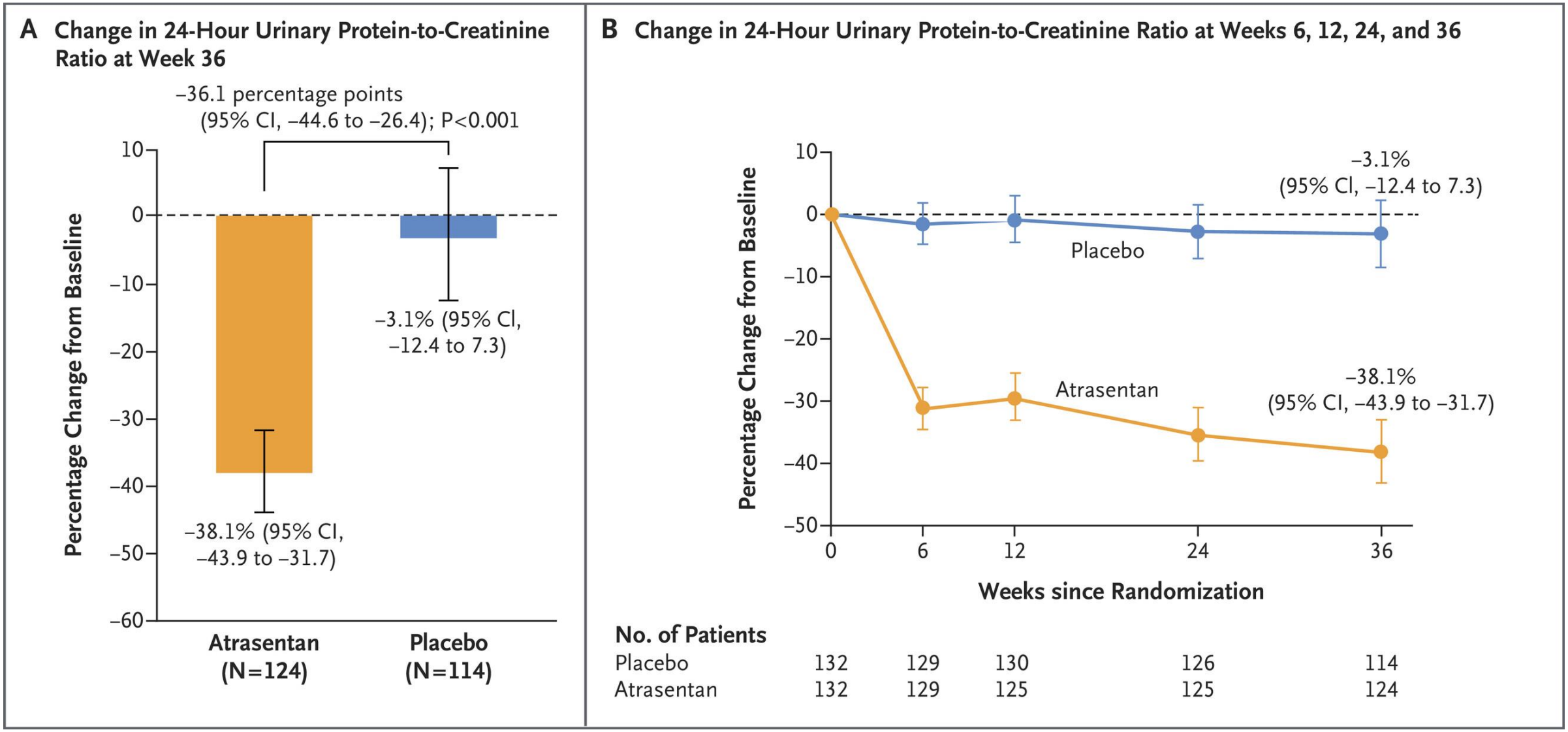
Atrasentan in Patients with IgA Nephropathy

Hiddo J.L. Heerspink, Ph.D.,[1] Meg Jardine, M.B., B.S., Ph.D.,[2] Donald E. Kohan, M.D., Ph.D.,[3] Richard A. Lafayette, M.D.,[4] Adeera Levin, M.D.,[5] Adrian Liew, M.D.,[6] Hong Zhang, Ph.D.,[7] Amit Lodha, M.B., B.S.,[8] Todd Gray, M.S.P.H.,[9] Yi Wang, Ph.D.,[8] Ronny Renfurm, M.D.,[10] and Jonathan Barratt, M.D.,[11] for the ALIGN Study Investigators*

N Engl J Med. 2025 Feb 6;392(6):531-543

ALIGN – Atresentan in IgAN

- Phase 3, multinational, double-blind, randomized, controlled trial involving adults with biopsy-proven IgA nephropathy, a total urinary protein excretion of at least 1 g per day, and an estimated glomerular filtration rate of at least 30 ml per minute per 1.73 m2 of body-surface area.
- The primary outcome, assessed at a prespecified interim analysis of data from the first 270 patients in the main stratum, was the change in the 24-hour urinary protein-to-creatinine ratio from baseline to week 36; the change was estimated with the use of a repeated-measures model.
- Fluid retention was reported by 19 of 169 patients (11.2%) in the atrasenta group and in 14 of 170 (8.2%) in the placebo group but did not lead to discontinuation of the trial regimen.
- Lesser LFT issues than with sparsentan.



N Engl J Med. 2025 Feb 6;392(6):531-543

B Cell Therapeutics.

- Telitacicept – BAFF / APRIL
- Atacicept – BAFF / ARPIL
- Povetacicept – BAFF / ARPIL
- Sibeprenlimab – APRIL
- Zigakibart – APRIL

B Cell Therapeutics.

Telitacicept – BAFF / APRIL

- Phase II trial in China.
- 44 IgAN patients.
- eGFR >35 & proteinuria ≥ 0.75 g/d.
- Telitacicept, 160 mg (n=16) or 240 mg (n=14) subcut every week.
- Vs Placebo (n=14).
- Over 24 weeks.

Kidney Int Rep. 2022 Dec 29;8(3):499-506.

B Cell Therapeutics.

Telitacicept – BAFF / APRIL

- Phase II trial in China.
- 44 IgAN patients.
- eGFR >35 & proteinuria ≥ 0.75 g/d.
- Telitacicept, 160 mg (n=16) or 240 mg (n=14) subcut every week.
- Vs Placebo (n=14).
- Over 24 weeks.

- Proteinuria (mean change +/- SD).
  - Placebo, 0.03 +/- 1.09 (-0.66 - 0.60).
  - Telitacicept 160, -0.32 +/- 0.82 (-0.75 - 0.12).
  - Telitacicept 240, -0.89 +/- 0.85.

- EGFR (mean change +/- SD).
  - Placebo, -5.70 +/- 8.99 (-11.41 - 0.01).
  - Telitacicept 160, 4.32 +/- 9.14 (-0.55 - 9.19).
  - Telitacicept 240, 2.34 +/- 5.21 (-0.67 - 5.35).

- SAEs (percentage).
  - Placebo, 7.10.
  - Telitacicept 160, 6.30.
  - Telitacicept 240, 14.40.

Kidney Int Rep. 2022 Dec 29;8(3):499-506. [27]

B Cell Therapeutics.

Telitacicept – BAFF / APRIL

- Phase II RCT.
- 24 IgAN patients.
- eGFR >35 & proteinuria ≥ 0.75 g/d.
- Telitacicept, 160 mg (n=9) or 240 mg (n=9).
- Vs Placebo (n=8).
- Over 24 weeks.

Kidney Int Rep. 2024 Apr; 9(4): 1067–1071

B Cell Therapeutics.

Telitacicept – BAFF / APRIL

- Phase II RCT.
- 24 IgAN patients.
- eGFR >35 & proteinuria ≥ 0.75 g/d.
- Telitacicept, 160 mg (n=9) or 240 mg (n=9).
- Vs Placebo (n=8).
- Over 24 weeks.

- Gd-IgA1, mean decrease % (95% CI).
  - Placebo, Ref.
  - Telitacicept 160, 43.9% (29.8%, 55.1%).
  - Telitacicept 240, 50.4% (38.6%, 59.9%).

- IgG-IgA IC, mean decrease % (95% CI).
  - Placebo, Ref.
  - Telitacicept 160, 31.7% (14.4%, 45.5%).
  - Telitacicept 240, 42.7% (29.5%, 53.4%).

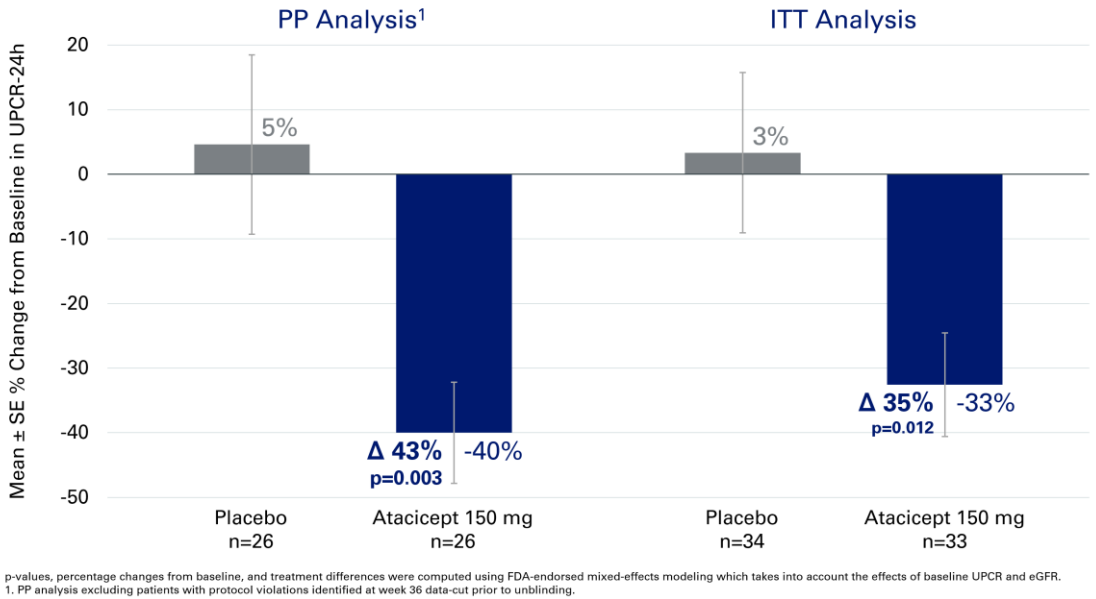
- Poly-IgA IC, mean decrease % (95% CI).
  - Placebo, Ref.
  - Telitacicept 160, 41.3% (6.5%, 63.1%).
  - Telitacicept 240, 67.2% (48.5%, 79.1%).

Kidney Int Rep. 2024 Apr; 9(4): 1067–1071

B Cell Therapeutics.

Atacicept – BAFF / APRIL.

At week 36, patients receiving atacicept 150 mg had a statistically significant reduction in proteinuria compared to placebo.



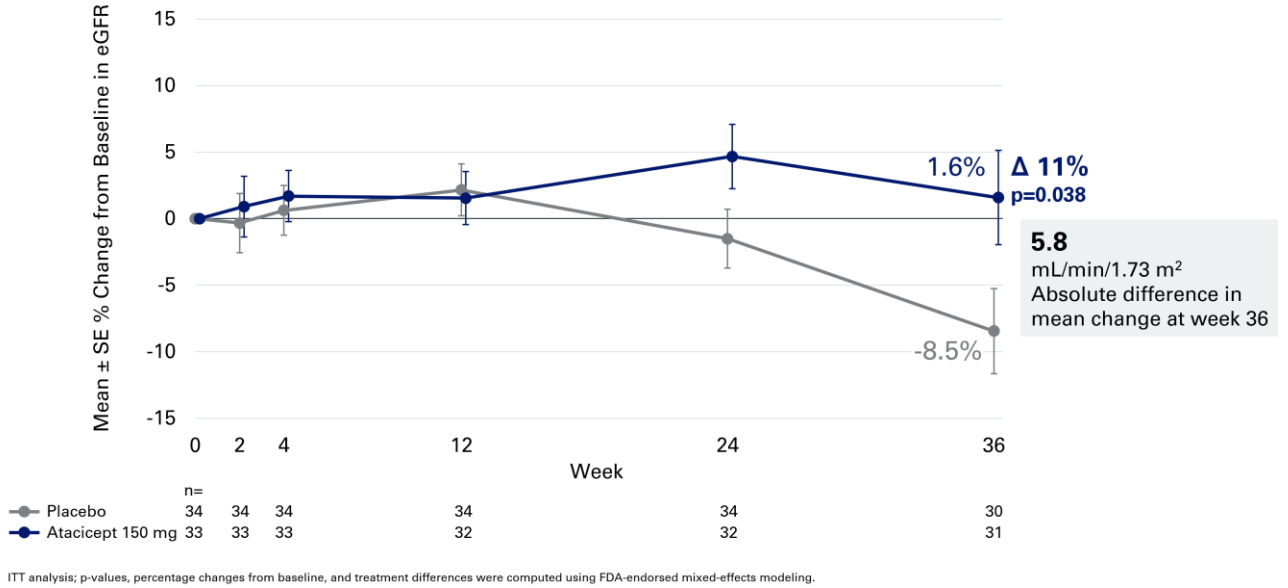
p-values, percentage changes from baseline, and treatment differences were computed using FDA-endorsed mixed-effects modeling which takes into account the effects of baseline UPCR and eGFR.
1. PP analysis excluding patients with protocol violations identified at week 36 data-cut prior to unblinding.

ORIGIN 2B by VERA therapeutics

B Cell Therapeutics.

Atacicept – BAFF / APRIL.

Patients receiving atacicept had stable eGFR through week 36, demonstrating a statistically significant and clinically meaningful difference of 5.8 mL/min/1.73 m^2 compared to placebo at week 36.



Plasma Cell Therapeutics.

- Bortezomib– plasma cell depletion.
- Felzartamab – plasma cell depletion
- Mezagitamab – plasma cell depletion

Plasma Cell Therapeutics.

Bortezomib– plasma cell depletion

- Pilot open label trial

- Three out of eight patients with full remission.

Plasma Cell Therapeutics.

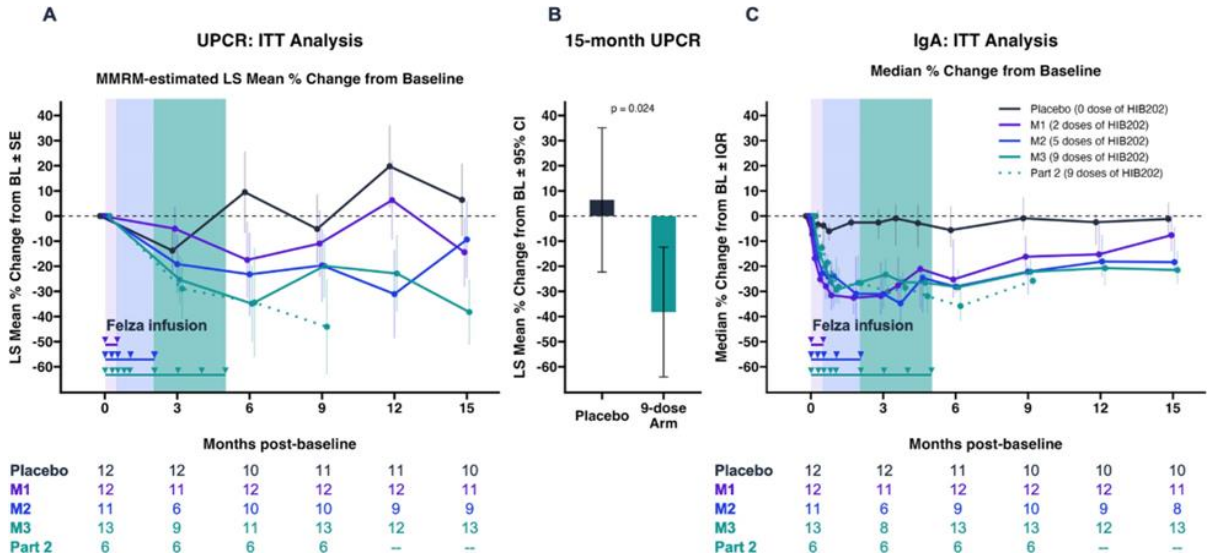
Felzartamab– plasma cell depletion

- Phase II randomized trial.
- Multi-center, double blind, placebo controlled.
- 1:1:1:1 allocation with 3 active arms.
- Enrolled 48 subjects, 46 completed.

Plasma Cell Therapeutics.

Felzartamab– plasma cell depletion

- Phase II randomized trial.
- Multi-center, double blind, placebo controlled.
- 1:1:1:1 allocation with 3 active arms.
- Enrolled 48 subjects, 46 completed.



35% reduction at 6mo
38% reduction at 15 mo

Plasma Cell Therapeutics.

Mezagitamab– plasma cell depletion

- Phase Ib open label trial ongoing.

Complement Blockade.

Reduce the burden of the downstream complement activation.

- Avacopan – C5a receptor antagonist
- Cemdisiran – C5 in liver
- Iptacopan – Factor B inhibitor
- Narsoplimab – MASP2 inhibitor
- Ravulizumab – C5 inhibitor
- Pegcetacoplan – C3 inhibitor

Complement Blockade.

- **Avacopan – C5a receptor antagonist**
- Cemdisiran – C5 in liver
- Iptacopan – Factor B inhibitor
- Narsoplimab – MASP2 inhibitor
- Ravulizumab – C5 inhibitor
- Pegcetacoplan – C3 inhibitor
- IONIS-FB-LRx – Factor B

- Pilot Phase II open label trial.
- 6/7 patient with uPCR reduction.

Complement Blockade.

- Avacopan – C5a receptor antagonist
- **Cemdisiran – C5 in liver**
- Iptacopan – Factor B inhibitor
- Narsoplimab – MASP2 inhibitor
- Ravulizumab – C5 inhibitor
- Pegcetacoplan – C3 inhibitor
- IONIS-FB-LRx – Factor B

- Phase II trial results Jun 2022
- Descriptive
- 31 subjects, 22 on Cemdisiran and 9 on placebo.
- UPCR at 32 weeks:
  - 37% reduction with Cemdisiran
  - 90% CI 0-61

Complement Blockade.

- Avacopan – C5a receptor antagonist
- Cemdisiran – C5 in liver
- Iptacopan – Factor B inhibitor
- **Narsoplimab – MASP2 inhibitor**
- Ravulizumab – C5 inhibitor
- Pegcetacoplan – C3 inhibitor
- IONIS-FB-LRx – Factor B

- Phase II trial
- Second part was randomized, placebo controlled, involved 9 patients.
- 8 patients entered open extension.
- Median proteinuria reduction: 61.4% at 31-54 weeks.

- Phase III trial
- Terminated early due to lack of efficacy.

Complement Blockade.

- Avacopan – C5a receptor antagonist
- Cemdisiran – C5 in liver
- Iptacopan – Factor B inhibitor
- Narsoplimab – MASP2 inhibitor
- **Ravulizumab – C5 inhibitor**
- Pegcetacoplan – C3 inhibitor
- IONIS-FB-LRx – Factor B

- Phase II randomized double blind trial.
- 33.1% reduction in proteinuria at 26w.

Complement Blockade.

- Avacopan – C5a receptor antagonist
- Cemdisiran – C5 in liver
- Iptacopan – Factor B inhibitor
- Narsoplimab – MASP2 inhibitor
- Ravulizumab – C5 inhibitor
- **Pegcetacoplan – C3 inhibitor**
- IONIS-FB-LRx – Factor B

- Phase II single arm open label.
- Proteinuria response at 48 weeks.
- Results awaited.

Complement Blockade.

- Avacopan – C5a receptor antagonist
- Cemdisiran – C5 in liver
- Iptacopan – Factor B inhibitor
- Narsoplimab – MASP2 inhibitor
- Ravulizumab – C5 inhibitor
- Pegcetacoplan – C3 inhibitor
- **IONIS-FB-LRx – Factor B**

- Phase II open label study.
- 8/10 pts with uPCR reduction at 29w.

- Phase III randomized, double blind, placebo controlled trial is ongoing.

Results of a randomized double-blind placebo-controlled Phase 2 study propose iptacopan as an alternative complement pathway inhibitor for IgA nephropathy.

kidney INTERNATIONAL

ISN INTERNATIONAL SOCIETY OF NEPHROLOGY

Methods

PHASE 2

- Proof of concept, dose-ranging study
- Double-blind
- Randomized
- Biopsy-confirmed primary IgA nephropathy

Intervention

Iptacopan versus placebo (administered orally, twice daily)

Part 1 (3-month treatment)

(N=46)
- Iptacopan 10 mg
- Iptacopan 50 mg
- Iptacopan 200 mg
- Placebo

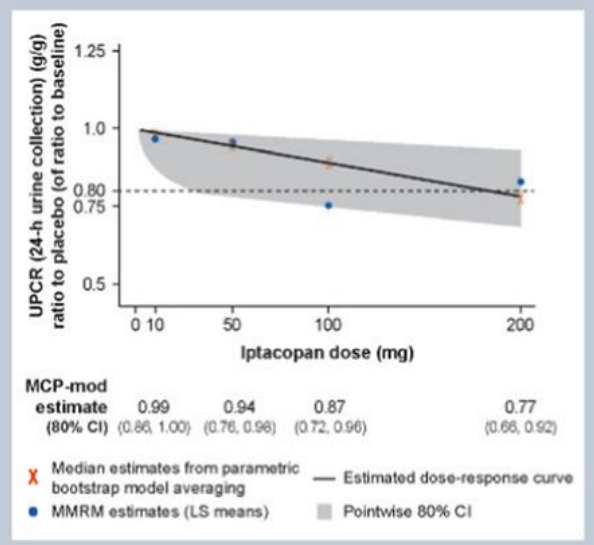
Part 2 (6-month treatment)

(N=66)
- Iptacopan 10 mg
- Iptacopan 50 mg
- Iptacopan 100 mg
- Iptacopan 200 mg
- Placebo

Findings

Primary analysis:

- Statistically significant dose-response effect observed (P=0.038), with a 23% (80% CI: 8–34) reduction in UPCR from baseline achieved with iptacopan 200 mg vs placebo at 3 months



Secondary analysis:

- Strong inhibition of several biomarkers of alternative complement activity (plasma Bb, Wieslab assay in serum, and urinary sC5b-9) observed; maximal inhibition observed with iptacopan 200 mg that was sustained through month 6

Zhang, 2023

CONCLUSION

This is the first study to show that selective inhibition of alternative pathway with iptacopan 200 mg results in a clinically meaningful reduction in proteinuria in patients with IgA nephropathy

The NEW ENGLAND JOURNAL of MEDICINE

ESTABLISHED IN 1812 FEBRUARY 6, 2025 VOL. 392 NO. 6

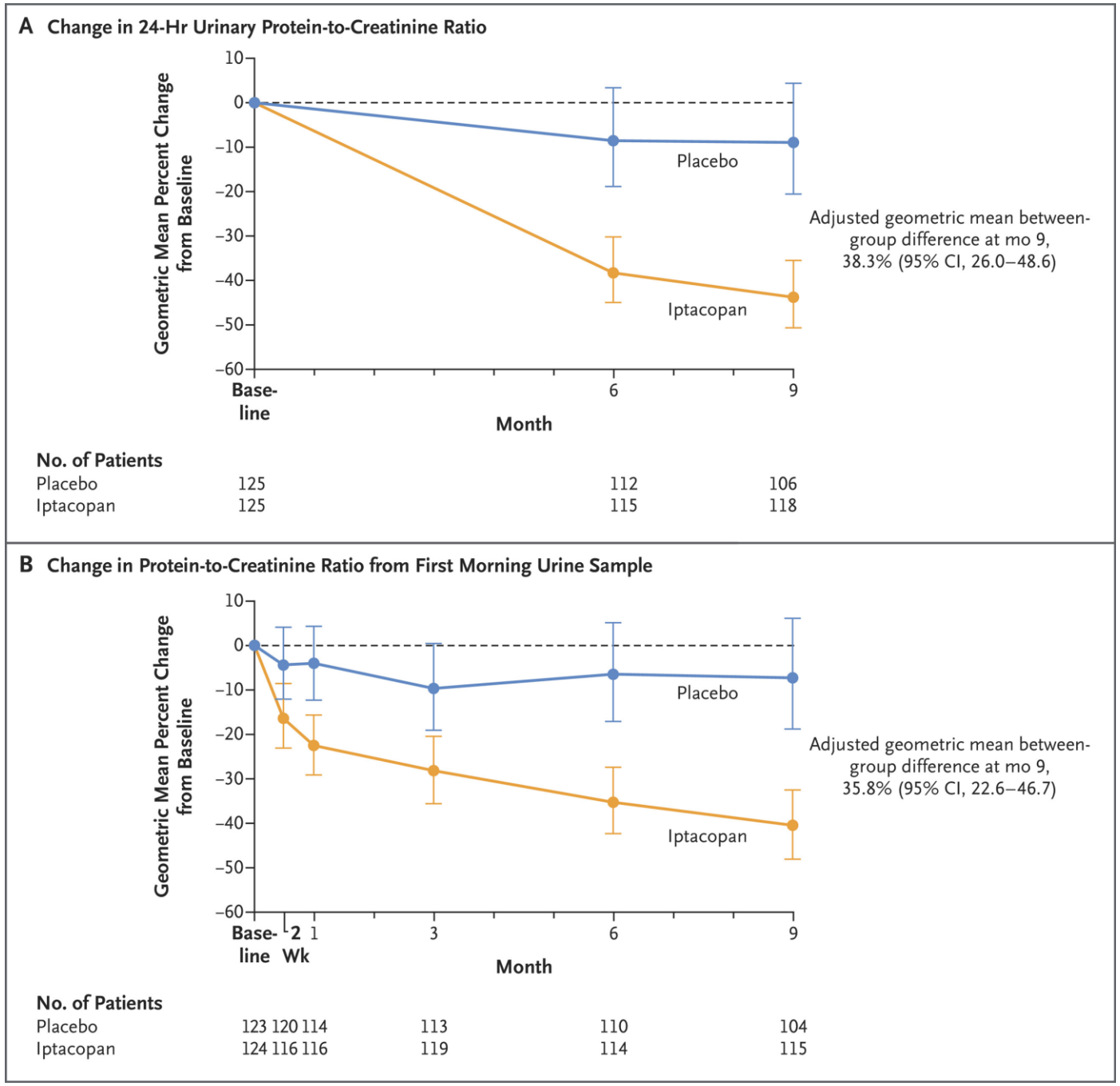
Alternative Complement Pathway Inhibition with Iptacopan in IgA Nephropathy

V. Perkovic,[1] J. Barratt,[2] B. Rovin,[3] N. Kashihara,[4] B. Maes,[5] H. Zhang,[6] H. Trimarchi,[7] D. Kollins,[8] O. Papachristofi,[8] S. Jacinto-Sanders,[8] T. Merkel,[8] N. Guerard,[8] R. Renfurm,[8] T. Hach,[8] and D.V. Rizk,[9] for the APPLAUSE-IgAN Investigators*

N Engl J Med. 2025 Feb 6;392(6):531-543

APPLAUSE-IgAN – Iptacopan in IgAN

- Phase 3, double-blind, randomized, placebo-controlled trial, we enrolled adults with biopsy-confirmed IgA nephropathy and proteinuria (defined as a 24-hour urinary protein-to-creatinine ratio of ≥1 [with protein and creatinine both measured in grams]) despite optimized supportive therapy.
- Patients were randomly assigned, in a 1:1 ratio, 222 patients in the iptacopan group and 221 in the placebo group, to receive oral iptacopan (200 mg) or placebo twice daily for 24 months while continuing to receive supportive therapy.
- The primary objective of this prespecified interim analysis was to assess the efficacy of iptacopan as compared with that of placebo in reducing proteinuria at month 9.


A Change in 24-Hr Urinary Protein-to-Creatinine Ratio
Geometric Mean Percent Change from Baseline
10
0
−10
−20
−30
−40
−50
−60
Placebo
Iptacopan
Adjusted geometric mean between-group difference at mo 9, 38.3% (95% CI, 26.0–48.6)
Base-line
6
9
Month
No. of Patients
Placebo 125 112 106
Iptacopan 125 115 118
B Change in Protein-to-Creatinine Ratio from First Morning Urine Sample
Geometric Mean Percent Change from Baseline
10
0
−10
−20
−30
−40
−50
−60
Placebo
Iptacopan
Adjusted geometric mean between-group difference at mo 9, 35.8% (95% CI, 22.6–46.7)
Base-line
2 Wk
1
3
6
9
Month
No. of Patients
Placebo 123 120 114 113 110 104
Iptacopan 124 116 116 119 114 115

Trials in GNs other than IgAN

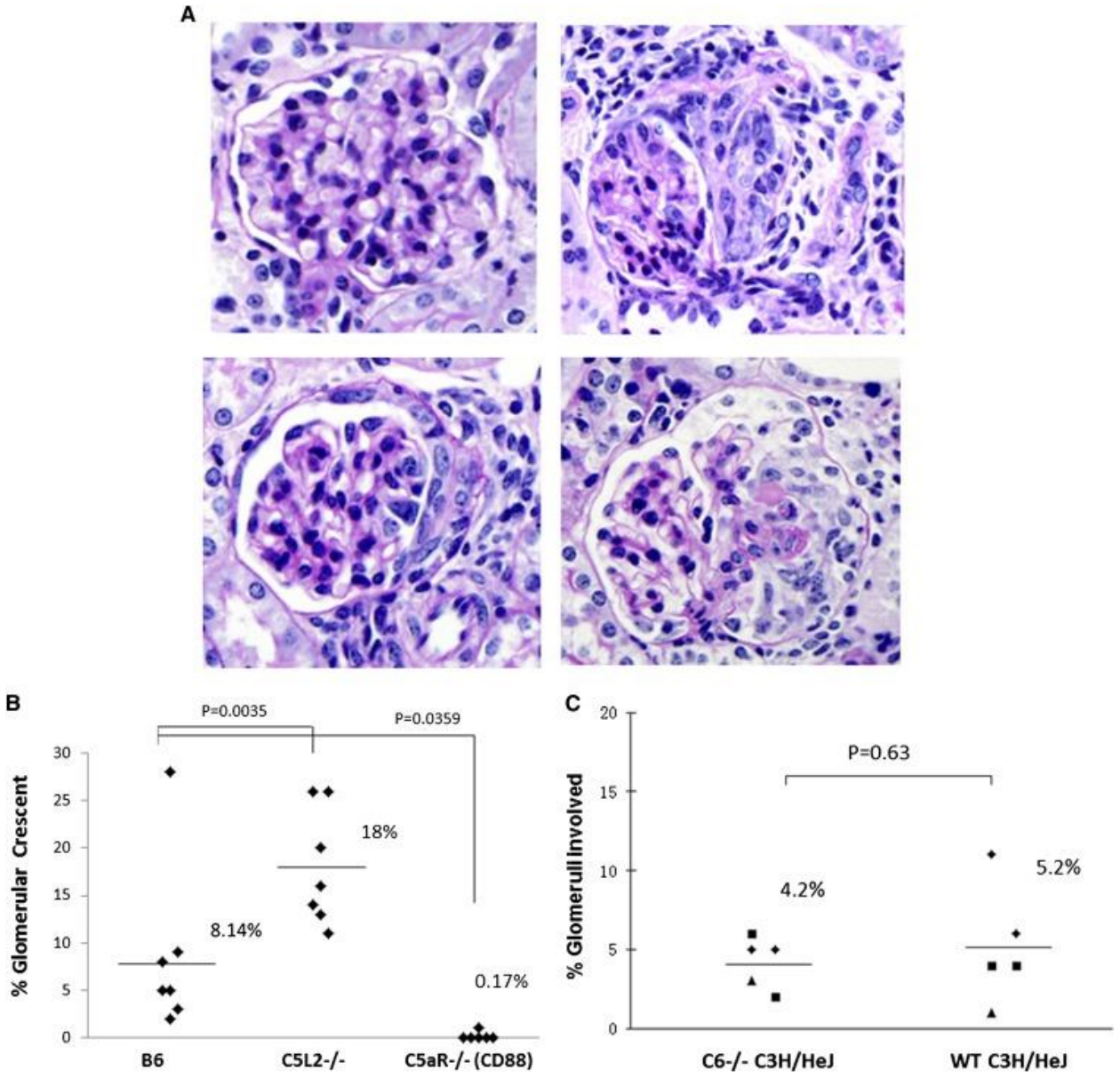
BRIEF COMMUNICATION www.jasn.org

C5a Receptor (CD88) Blockade Protects against MPO-ANCA GN

Hong Xiao,*† Daniel J. Dairaghi,‡ Jay P. Powers,‡ Linda S. Ertl, ‡ Trageen Baumgart,‡ Yu Wang,‡ Lisa C. Seitz,‡ Mark E.T. Penfold,‡ Lin Gan,§ Peiqi Hu,*† Bao Lu,§ Norma P. Gerard,‖ Craig Gerard,‖ Thomas J. Schall,‡ Juan C. Jaen,‡ Ronald J. Falk,*† and J. Charles Jennette*†

*Department of Pathology and Laboratory Medicine and †UNC Kidney Center, University of North Carolina, Chapel Hill, North Carolina; ‡ChemoCentryx, Inc., Mountain View, California; §Department of Ophthalmology, University of Rochester, Rochester, New York; and ‖Department of Pediatrics, Boston Children's Hospital, Harvard Medical School, Boston, Massachusetts

J Am Soc Nephrol. 2014 Feb;25(2):225-31



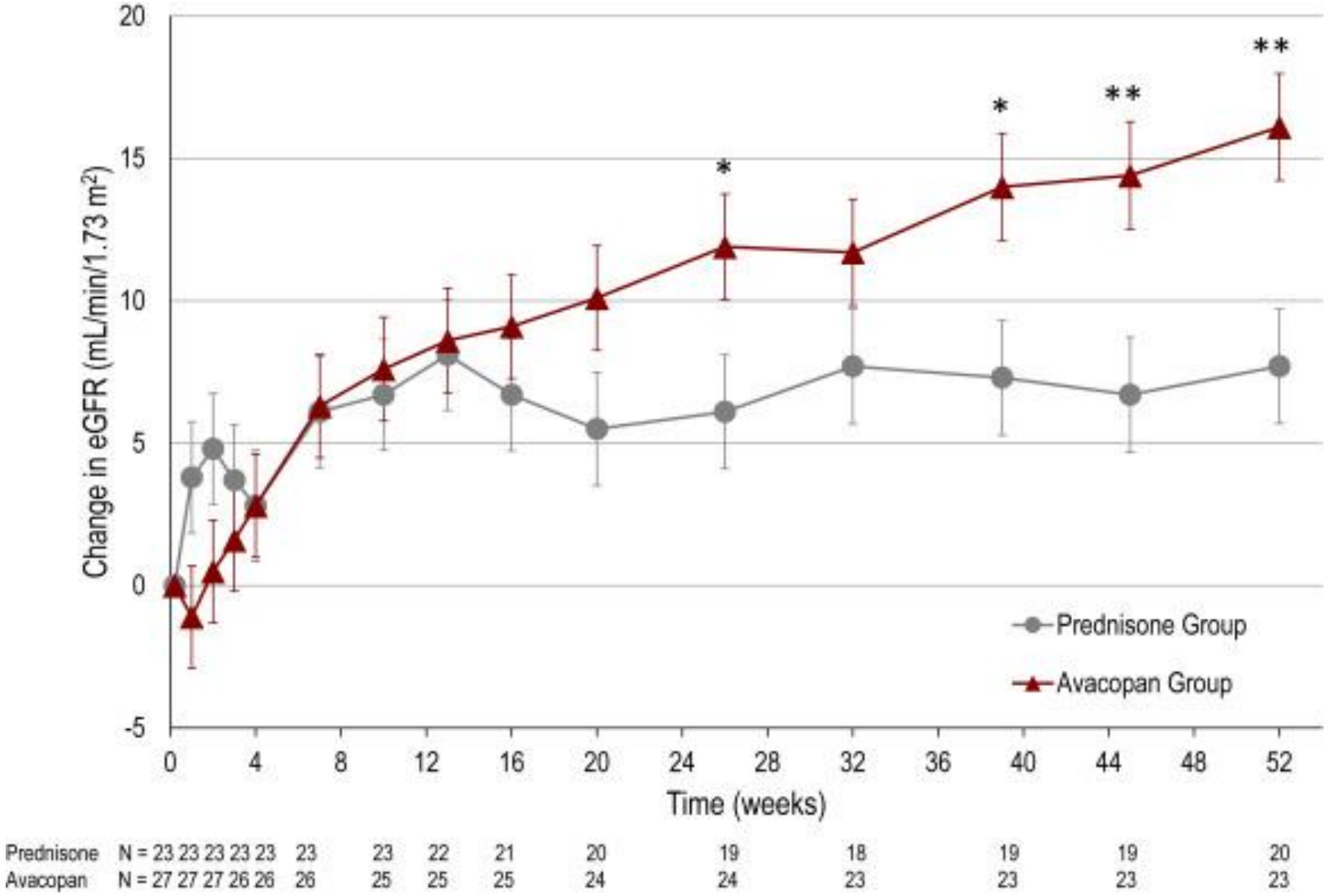
J Am Soc Nephrol. 2014 Feb;25(2):225-31[49]

The NEW ENGLAND JOURNAL of MEDICINE

ESTABLISHED IN 1812 FEBRUARY 18, 2021 VOL. 384 NO. 7

Avacopan for the Treatment of ANCA-Associated Vasculitis

David R.W. Jayne, M.D., Peter A. Merkel, M.D., M.P.H., Thomas J. Schall, Ph.D., and Pirow Bekker, M.D, Ph.D.,
for the ADVOCATE Study Group*



Kidney Int Rep. 2023 Feb 3;8(4):860-870

TAKE HOME MESSAGES

- There is a revolution of therapeutics in the GN arena.
- Long term outcome data is needed to prove efficiency.
- Prudent to discuss clinical trials with patients upfront.
- Individualized approach.