Precision Medicine in IgAN Management: Strategies for Personalizing Treatments

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Taipei, Republic of China

DISCLOSURES

Served as medical advisor for Travere, Otsuka, Kira Pharma, Eledon, CSL-Behring, Dimerix, Alpine, Arrowhead, Novartis (Chinook), Candid Therapuetic, Boehringer Ingelheim.

Received honorarium from AstraZeneca, Amgen, Elli Lily and Baxter, Novartis.

Served as a DMSC members in HEFEF trial (investigator initiated), ARGX-113-2203/AL-1103-014 Trial

Honorary Treasurer of Australia New Zealand Society of Nephrology (ANZSN) that received industry sponsorship for ANZSN activities

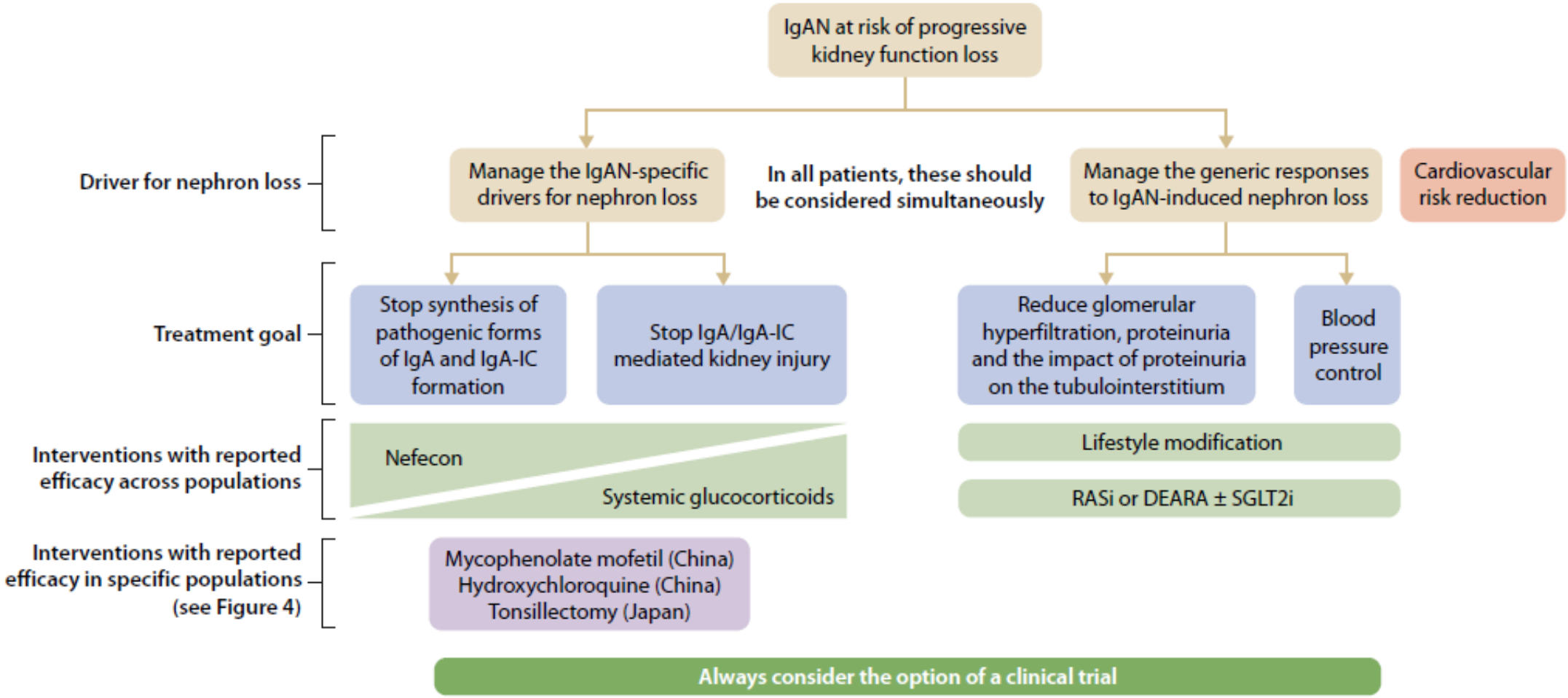
Overview

- Potential of precision approaches in IgA nephropathy
- Markers for diagnosis, predicting treatment response and monitoring disease progression in IgAN
  - Risk prediction tools
  - Genetic
  - Biomarkers
  - Histology and Pathonomics
  - Mechanistic study

What is precision medicine?

- Also known as personalized medicine
- is an innovative approach to tailoring disease prevention and treatment that takes into account differences in people's genes, environments, and lifestyles.

Treatment Targets positioning of drugs in KDIGO guideline 2025



Treatment goals:

- **Proteinuria≥0.5g/d (or equivalent)**
- Reduce the rate of loss of kidney function to **<1 ml/min/year**

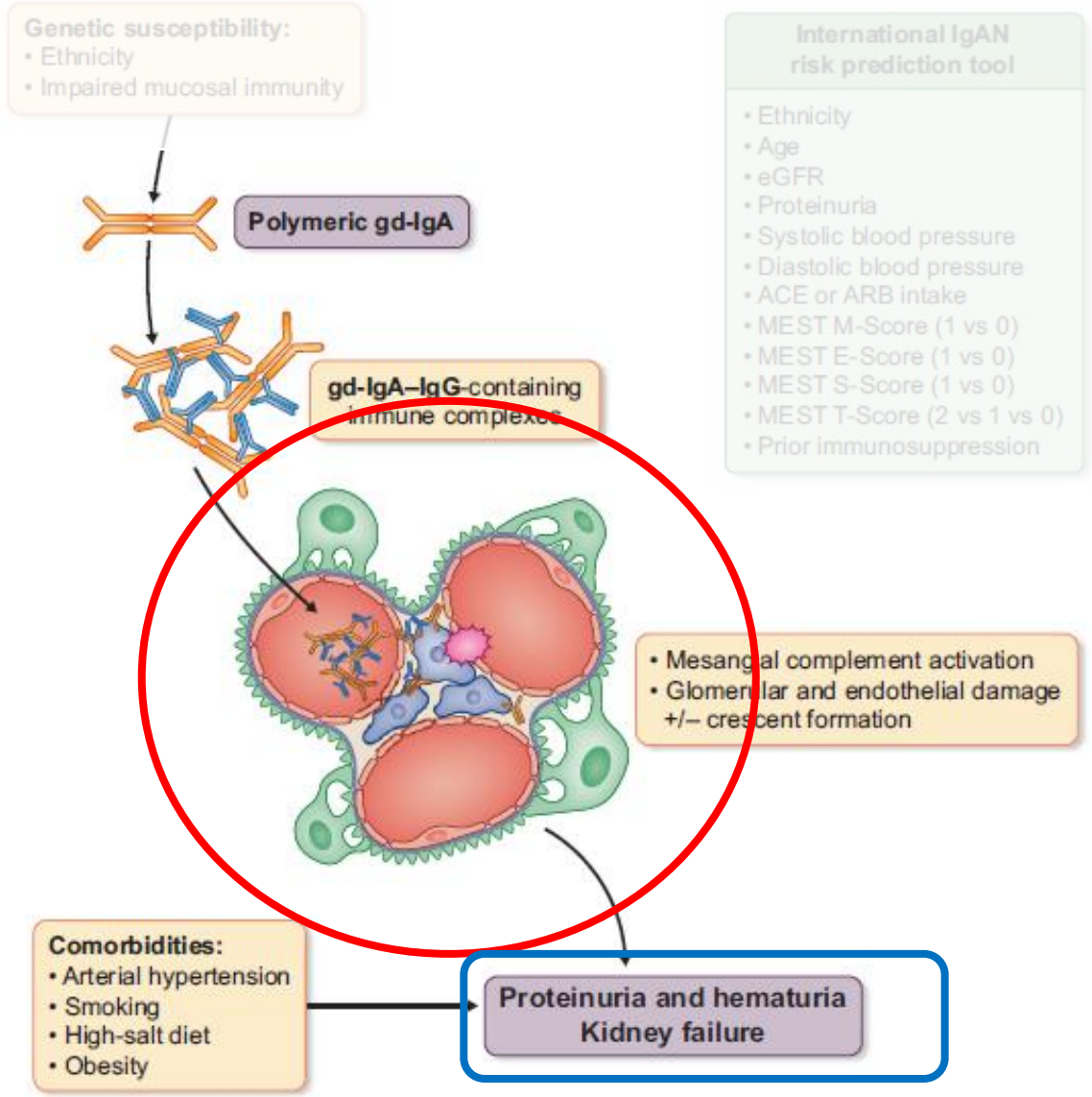
Four Hit Hypothesis and Beyond

Potential Markers

Gd-IgA1 levels
Autoantibodies
Immune Complexes

Tissue injury markers in blood & urine

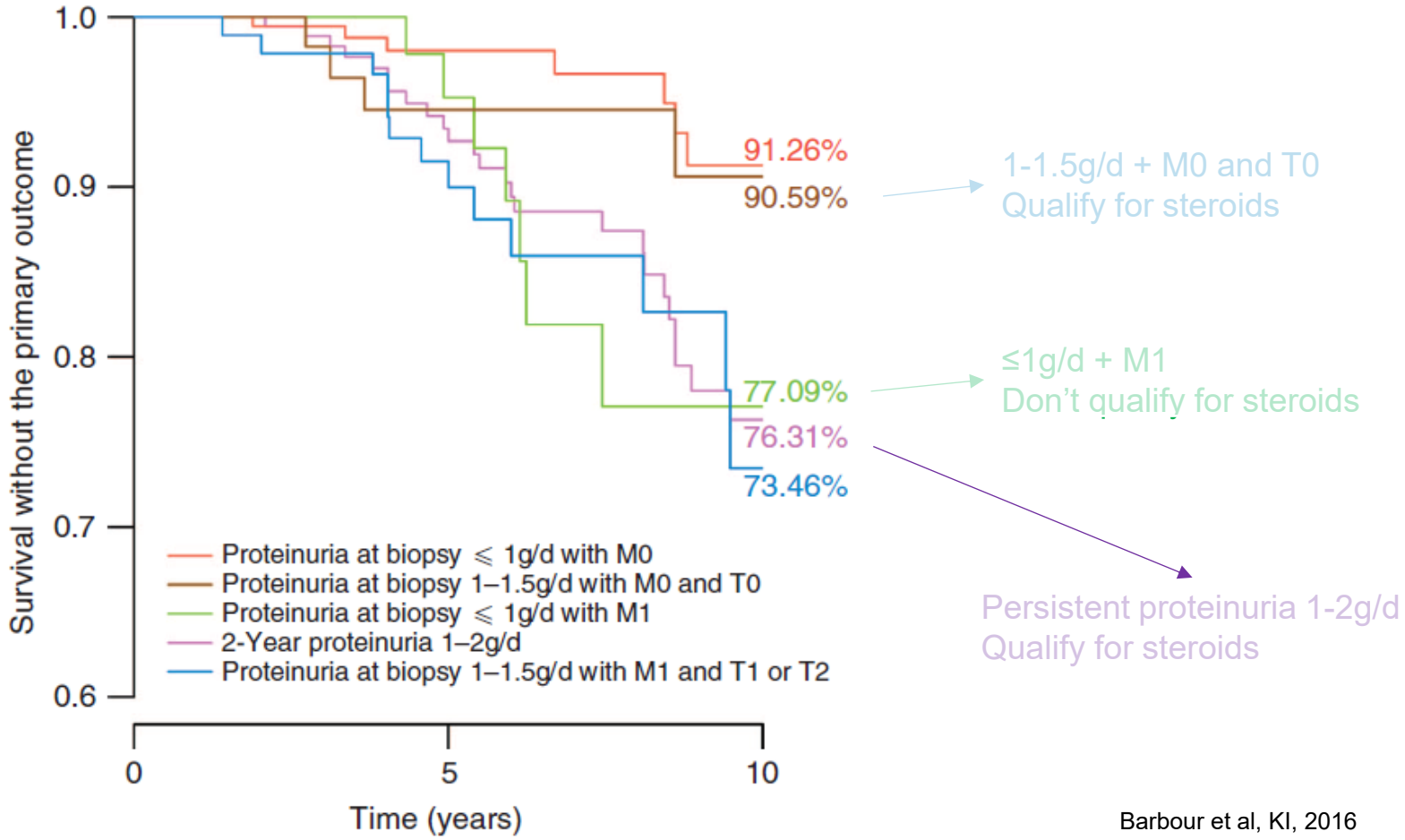
Histopathology & single-cell-resolution omics



Adapted from Seikrit et al. Nephrol Dial Transplant (2021) 36: ii24–ii30

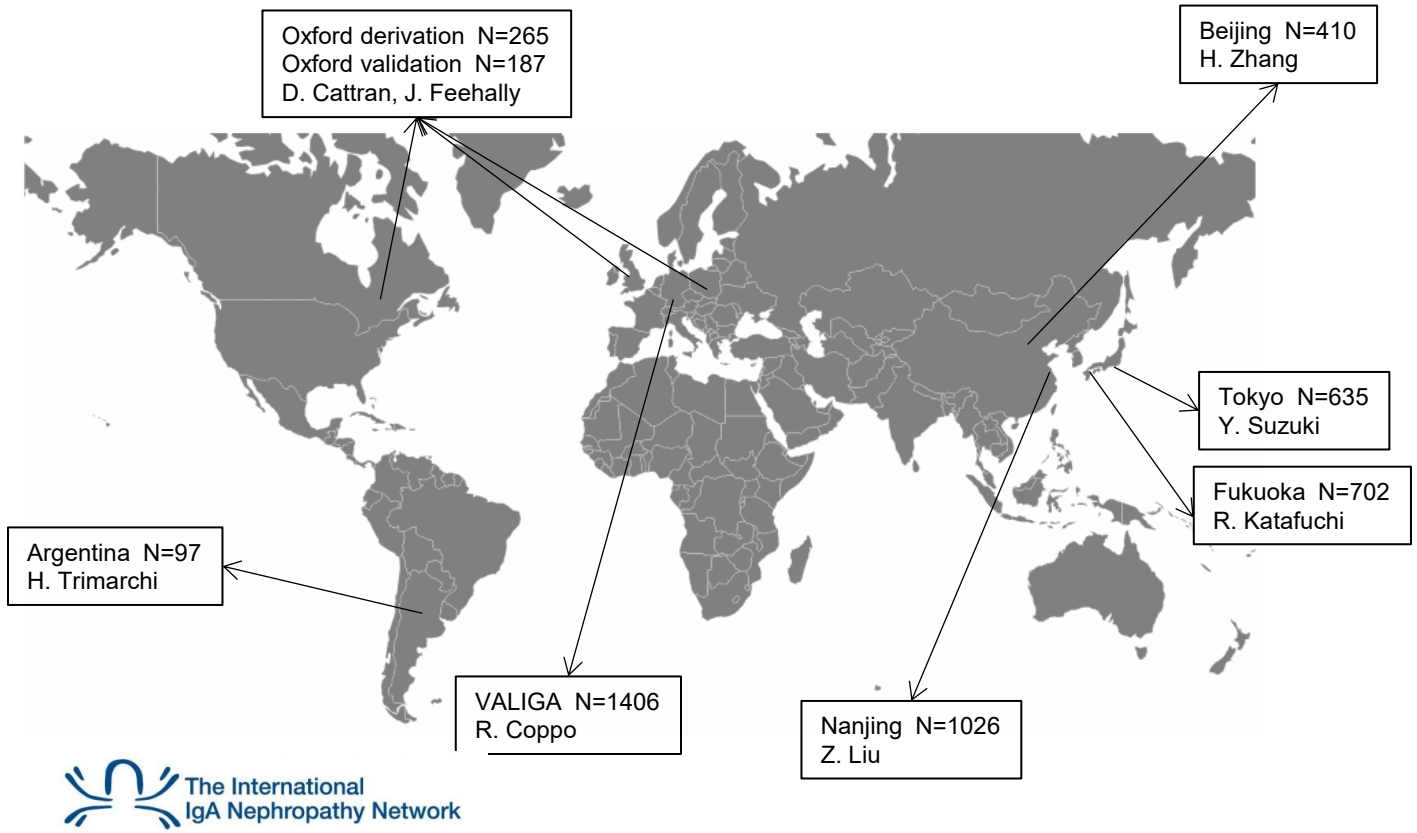
Proteinuria alone is not sufficient for risk stratification

Subgroup eGFR>50: risk of 50% decline eGFR or ESRD

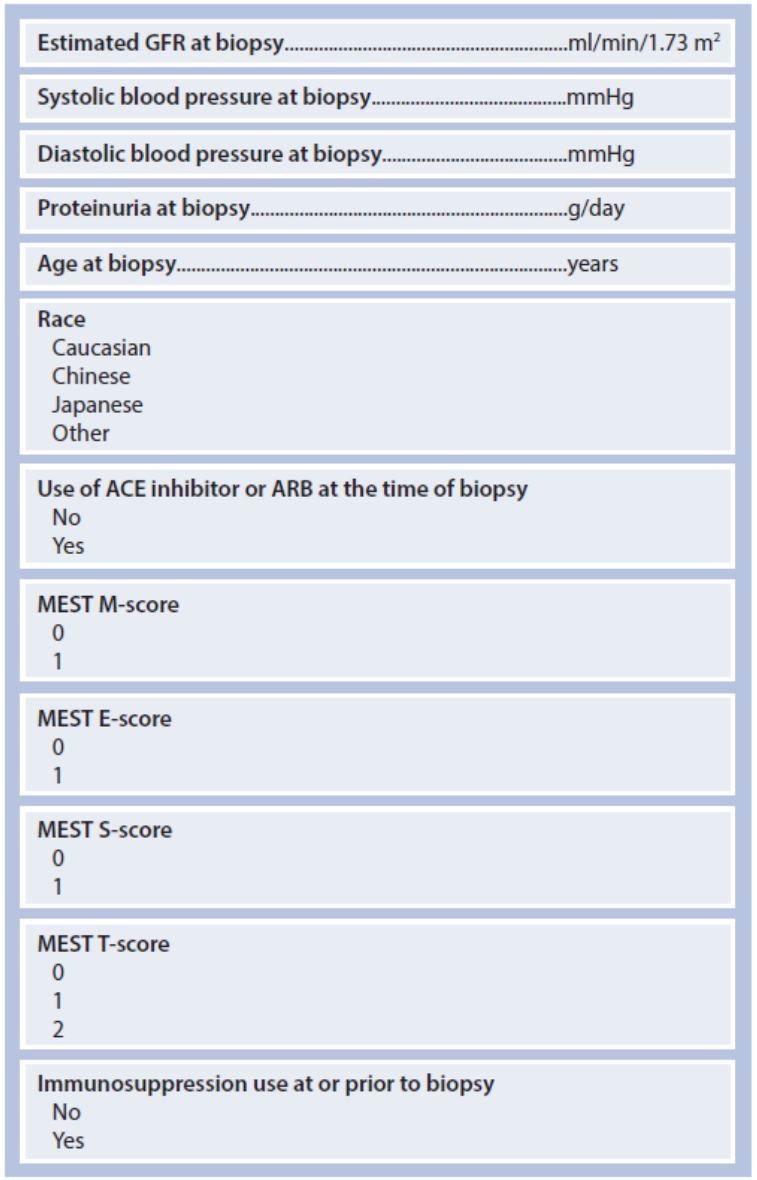


Barbour et al, KI, 2016

International IgAN Network research collaboration: 2014 to present



International IgAN Prediction tool for adults



Estimated GFR at biopsy.....ml/min/1.73 m²

Systolic blood pressure at biopsy.....mmHg

Diastolic blood pressure at biopsy.....mmHg

Proteinuria at biopsy.....g/day

Age at biopsy.....years

Race
- Caucasian
- Chinese
- Japanese
- Other

Use of ACE inhibitor or ARB at the time of biopsy
- No
- Yes

MEST M-score
- 0
- 1

MEST E-score
- 0
- 1

MEST S-score
- 0
- 1

MEST T-score
- 0
- 1
- 2

Immunosuppression use at or prior to biopsy
- No
- Yes

The risk of a 50% decline in eGFR or progression to ESKD 5 years after renal biopsy is 9.77%

Pros

- Risk prediction can go up to 80 months
- Include race
- Formula for paediatric and adults

Caveats

- Requires recent kidney biopsy validated up to 2 years from kidney biopsy
- Does not include C lesions
- Yet to predict treatment response

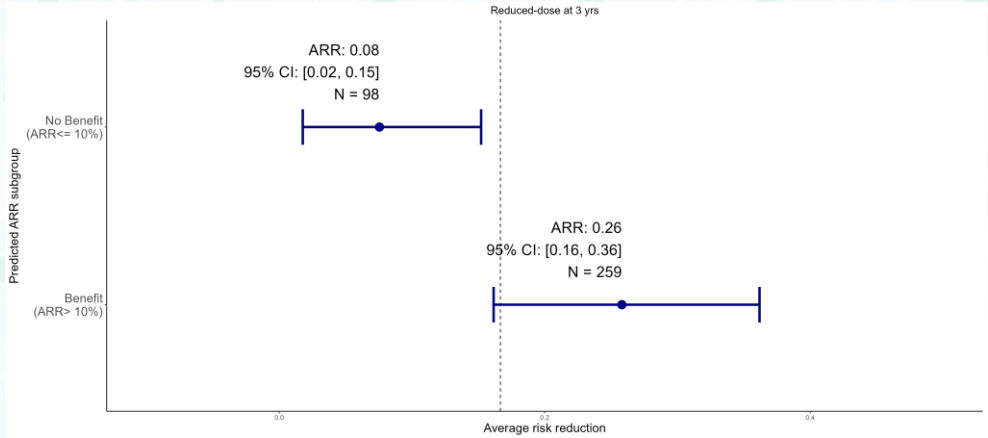
Predicting who is more likely to benefit from corticosteroids

Main effects selected & interactions forced

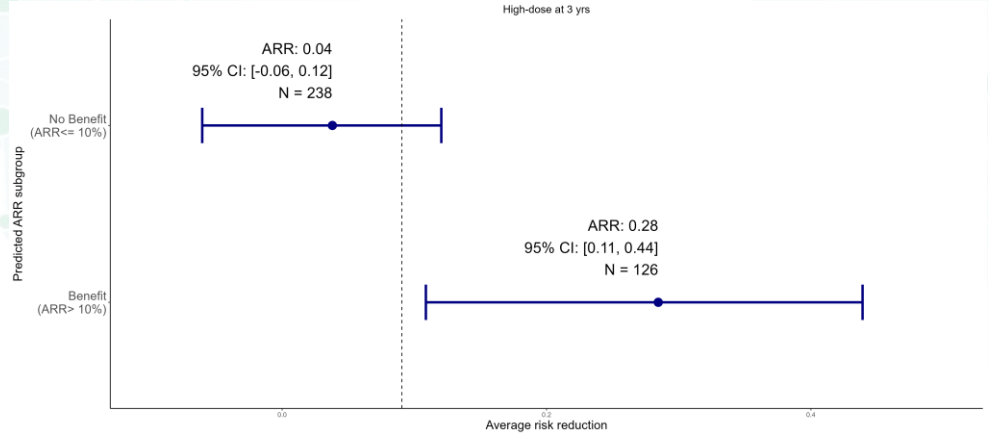
Variables	log-hazard (95% C.I)*
Treatment (Methylprednisolone vs. Placebo)	-0.14 [-0.59 ; -0.03]
eGFR (per 10 increase)	-0.22 [-0.40 ; -0.15]
Age (per 1 increase)	-0.03 [-0.05 ; -0.02]
Proteinuria (per 1 increase)	0.13 [0.07 ; 0.30]
Tubular atrophy	-0.10 [-0.52 ; 0.17]
T1 vs. T0	
T2 vs. T0	0.42 [0.04 ; 0.97]
RASB	0.09 [-0.16 ; 0.60]
50% - <80% vs. 0-50%	
>= 80% vs. 0-50%	0.22 [-0.00 ; 0.87]
C score	0.15 [-0.11 ; 0.55]
C1 vs. C0	
C2 vs. C0	-0.21 [-0.73 ; 0.38]
Race (Chinese vs. non-chinese)	-0.27 [-0.93 ; 0.02]
Time since biopsy (per 1 increase)	-0.00 [-0.00 ; 0.00]
SBP (per 1 increase)	0.01 [-0.00 ; 0.02]
Sex (Male vs. Female)	0.23 [-0.02 ; 0.63]
BMI (per 1 increase)	-0.02 [-0.06 ; 0.02]
Treatment * eGFR (per 10 increase)	-0.05 [-0.16 ; -0.02]
Treatment * Age (per 1 increase)	-0.00 [-0.01 ; 0.01]
Treatment * Proteinuria (per 1 increase)	0.07 [-0.03 ; 0.28]
Treatment * Tubular atrophy	-0.08 [-0.61 ; 0.33]
T1	
T2	-0.32 [-1.30 ; 0.14]
Treatment * RASB	0.05 [-0.32 ; 0.58]
>= 50% - <80%	
>=80	0.03 [-0.42 ; 0.46]
Treatment * C score	-0.14 [-0.67 ; 0.24]
C1	
C2	-0.42 [-1.74 ; 0.28]
Treatment * Race	0.09 [-0.13 ; 0.87]
Treatment * time since biopsy (per 1 increase)	0.00 [-0.01 ; 0.01]
Treatment * SBP (per 1 increase)	-0.00 [-0.01 ; -0.00]
Treatment * Sex	-0.09 [-0.57 ; 0.28]
Treatment * BMI (per 1 increase)	-0.00 [-0.01 ; 0.02]

** log-hazard estimates obtained from Ridge regression model with bootstrapped Conf. intervals*

Reduced dose

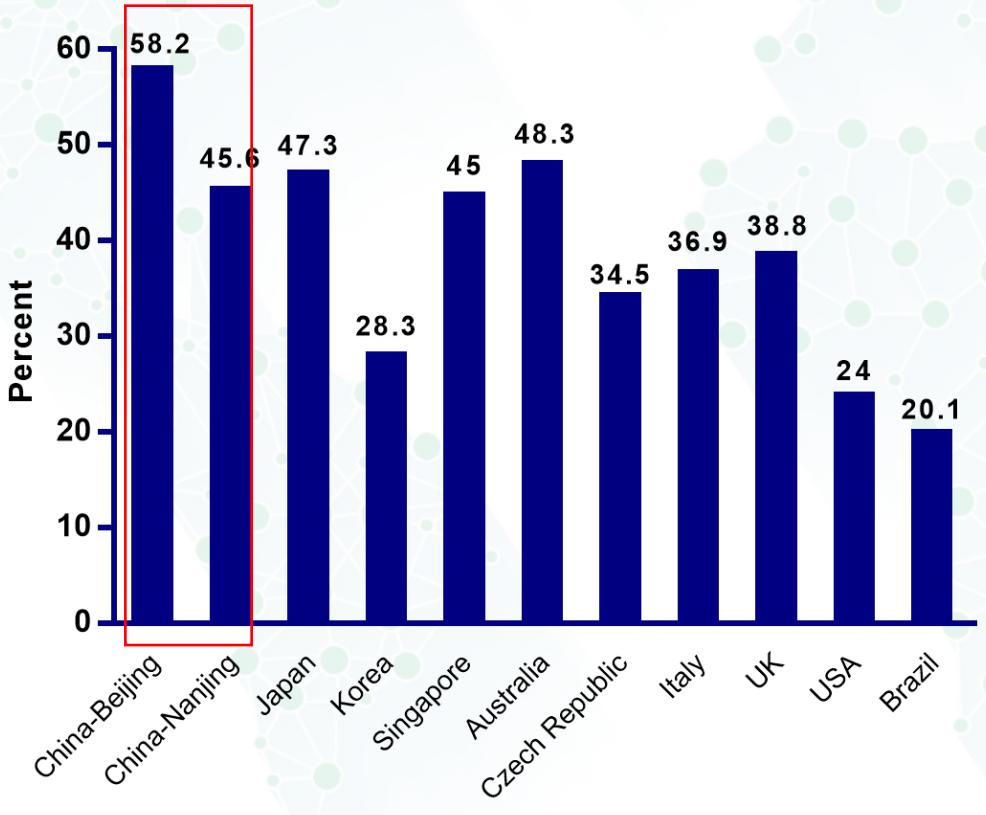


Full dose

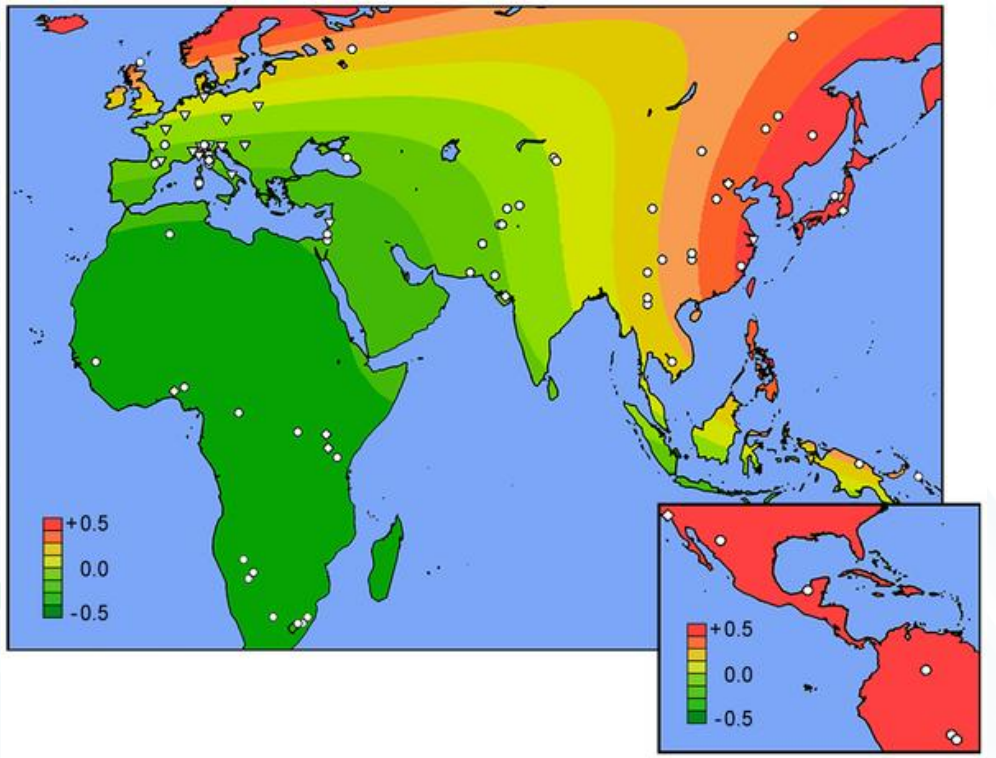


Manuscript under review

Worldwide prevalence and geospatial risk analysis of IgA nephropathy



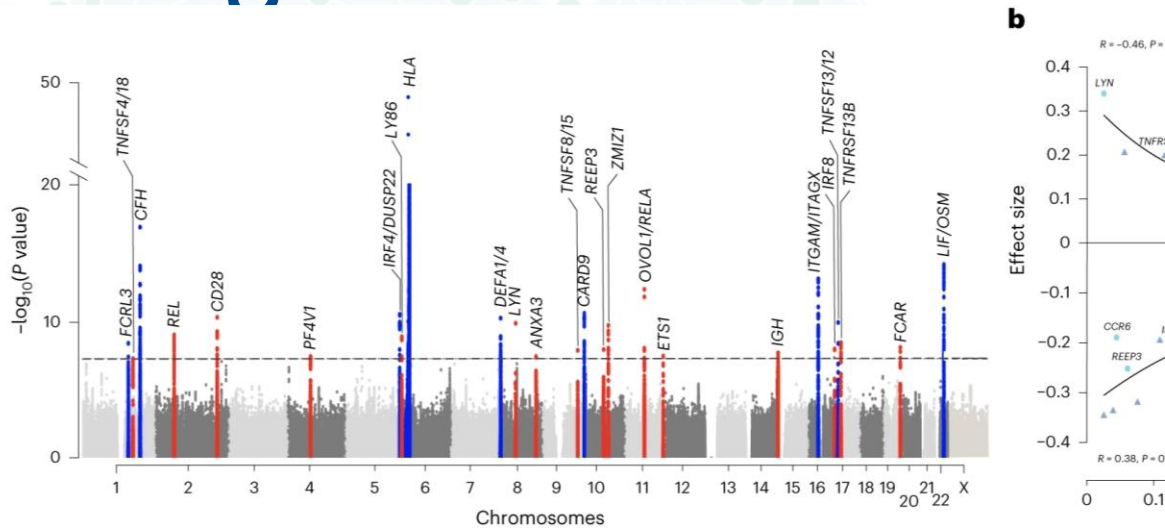
Chinese

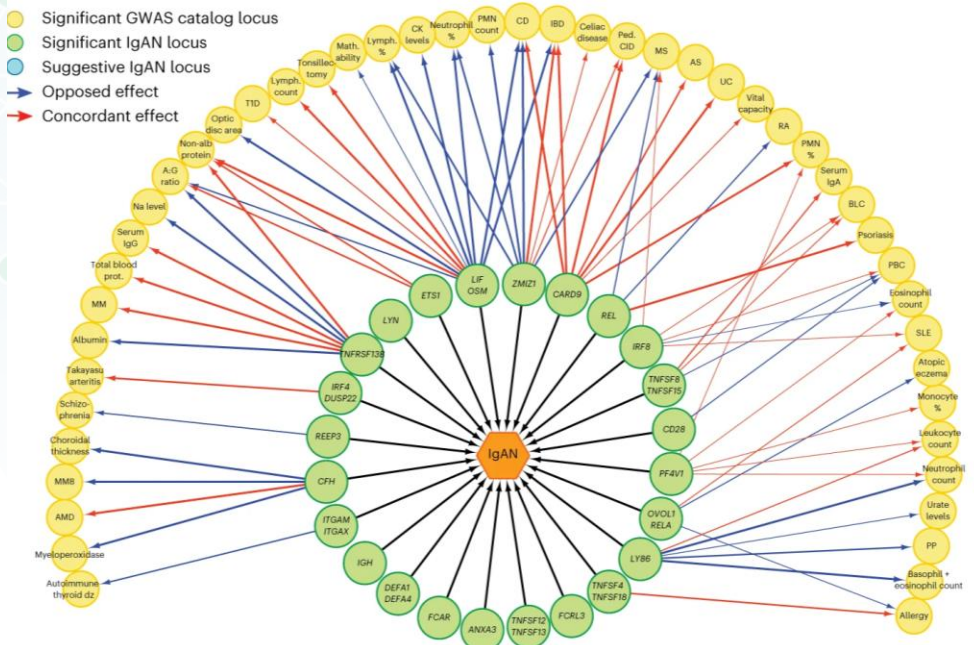


Kiryluk K, et al. (2012) PLoS Genet 8(6): e1002765

Genome wide analyses for IgAN

- 16 loci were new, including *TNFSF4/TNFSF18*, *REL*, *CD28*, *PF4V1*, *LY86*, *LYN*, *ANXA3*, *TNFSF8/TNFSF15*, *REEP3*, *ZMIZ1*, *OVOL1/RELA*, *ETS1*, *IGH*, *IRF8*, *TNFRSF13B* and *FCAR*.
- The risk loci were enriched in gene
  - Causing abnormal IgA levels
  - Integrity of the intestinal mucosal barrier
  - Involvement of adaptive (T & B cells) and innate immunity.

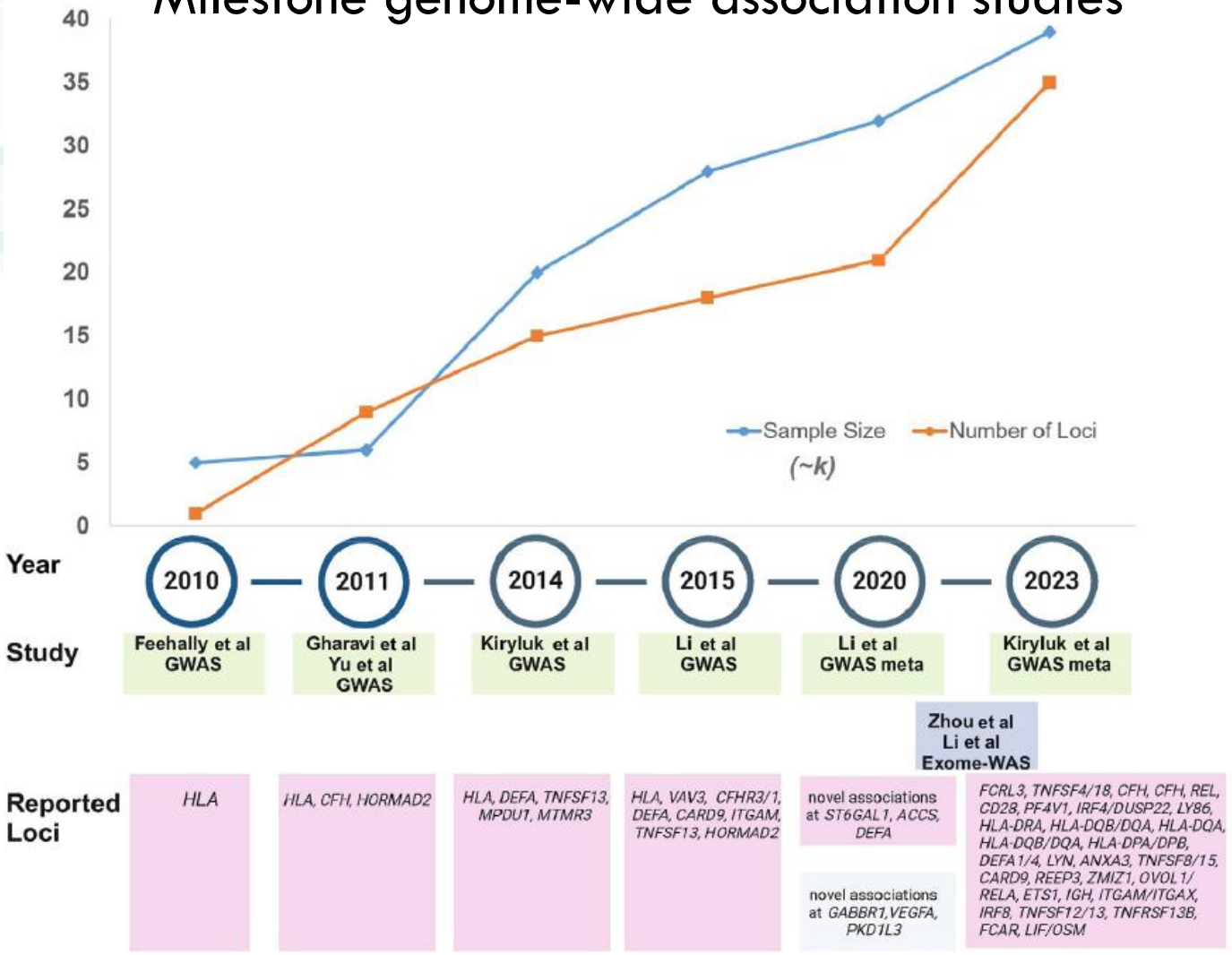




Kiryluk at al Nature Genetics volume 55,1091–1105 (2023)

Discovery in genetic has led to translation

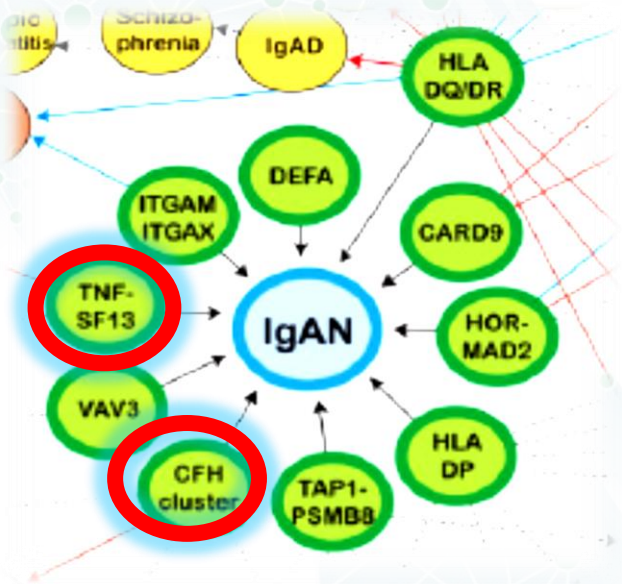
Milestone genome-wide association studies



GWAS in IgAN

Gharavi A et al: 2011, Yu XQ et al: 2012, Kiryluk K et al.: 2014

nature genetics



- ✓ **TNFSF13: APRIL/BAFF**
- ✓ **TNFSF13A; APRIL**
- ✓ **CFH cluster**

X Zhou, H Zhang *SeminNephrol* 44:151567 2025

Kiryluk at al Nature Genetics volume 55,1091–1105 (2023)

Promising B-cell modulating agents in IgAN

Anti-April % proteinuria reduction at month 12

The NEW ENGLAND JOURNAL of MEDICINE

ORIGINAL ARTICLE

Sibeprenlim… …pathy — Interim A… …3 Trial

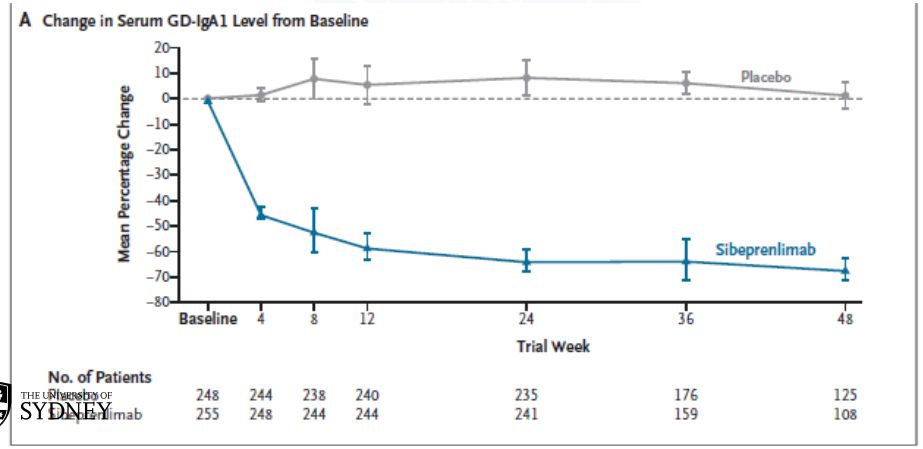
V. Perkovic,[1] H. Trimarc… …Wong,[5] J. Barratt,[6] Y. Suzuki,[7] A. Liew,[8] H. Zh… …evedo,[14] S.H. Han,[15] M. Praga,[16] B. Chacko,[17] M… …nga,[19] M. Walsh,[20,21] J. Xia,[22] C. Fajar… …V. Rizk,[23] for the VISIONARY Trial Investigators Group*

Placebo-adjusted treatment effect

54.3%

95% CI, 46.4%-60.9%

GdIgA1 levels



Anti-April and Anti-BAFF % proteinuria reduction at month 9

The NEW ENGLAND JOURNAL of MEDICINE

ORIGINAL ARTICLE

A Phase 3 Trial of Atacicept in Patients w… …ny

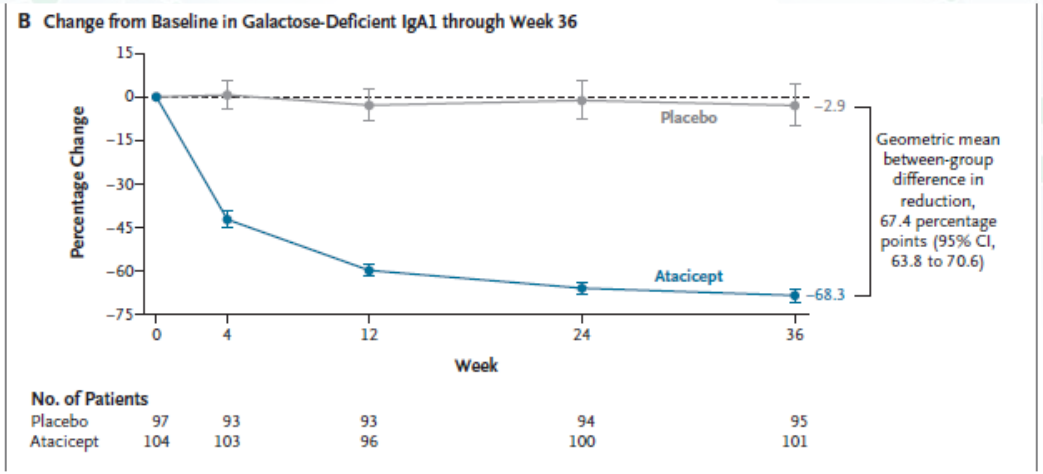
Richard Lafayette, M… …t M. Brenner, M.D.,[3] Kirk N. Campbell, M.D… …Jürgen Floege, M.D.,[6] Vivekanand Jha, M… …Kim, M.D., Ph.D.,[8] Adrian Liew, M.D… …Pal, M.D., D.M.,[11] Roberto Pecoits-… …Phoon, M.D.,[14,15] Dana V. Rizk, M.D.,[16] H… …ir Tesař, M.D., Ph.D.,[18] Hernán Trimarchi, M.D… …g Zhang, M.D., Ph.D.,[20] and Jonathan Barratt, Ph.D.,[21] for the ORIGIN Phase 3 Trial Investigators*

Placebo-adjusted treatment effect

41.8%

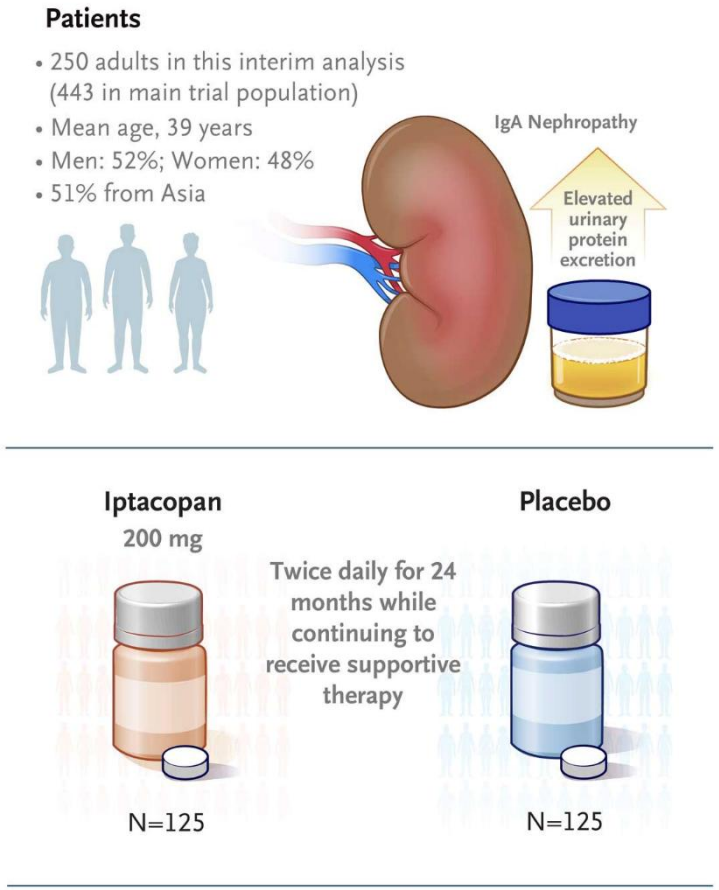
95% CI, 28.9%-52.3%

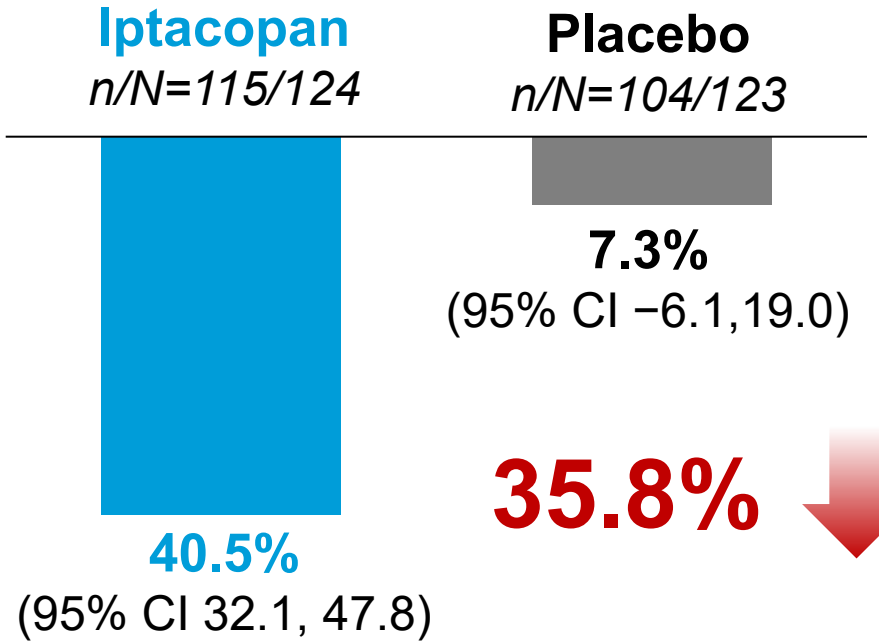
GdIgA1 levels

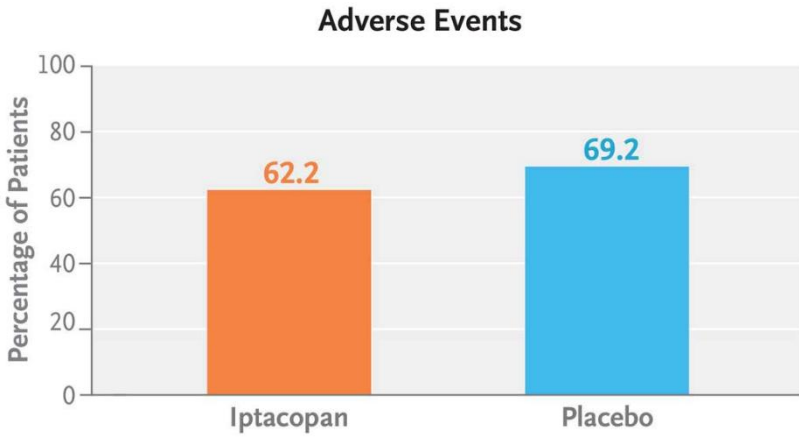


APPLAUSE trial (interim analysis): Targeting alternate complement pathway in IgA nephropathy

Reduction in UPCR-FMV at Month 9



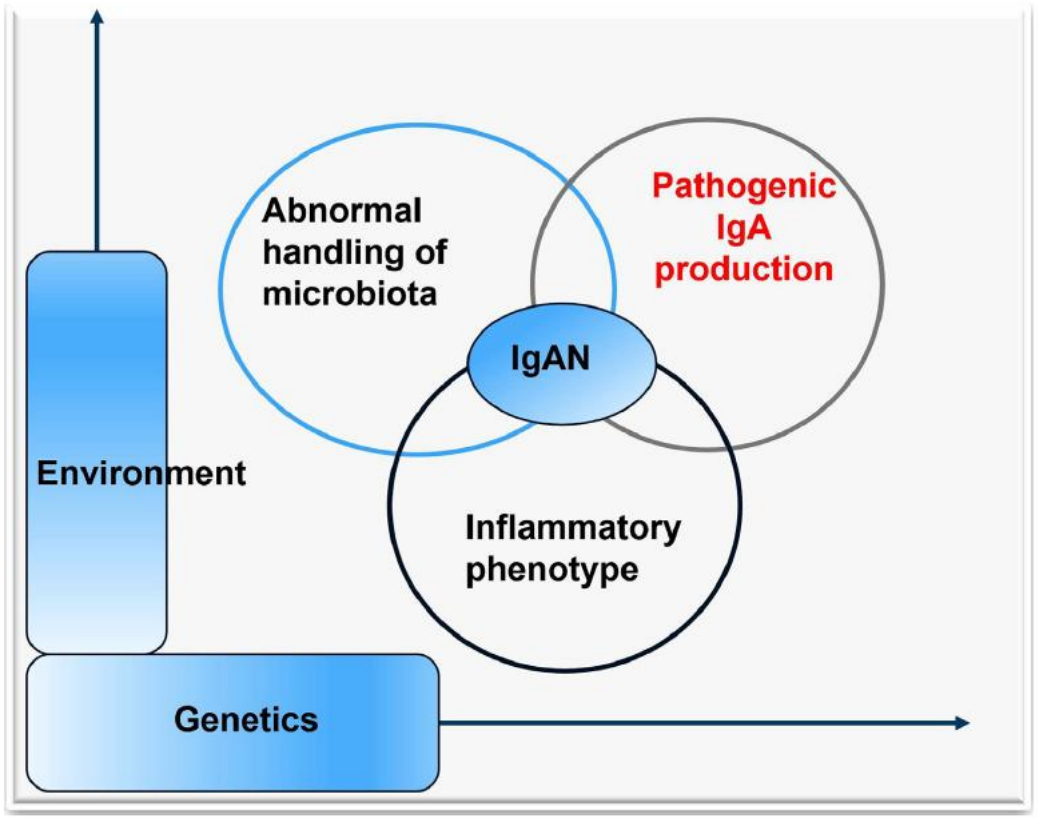




Relative percent reduction between arms
35.8% (95% CI 22.6, 46.7); *P*<0.0001†

Perkovic et al N Engl J Med 2025;392:531-543

Complex Interactions of Genetic Risk Loci with Environmental Exposures in IgAN

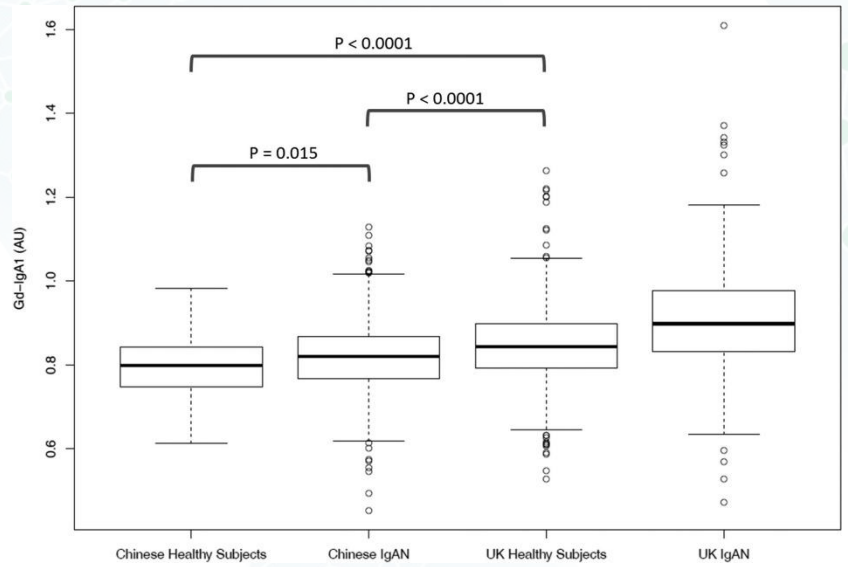


IgAN is thought to occur when individuals with genetic susceptibility traits come into contact with disease-triggering environmental risk factors such as infections, resulting in the activation of both innate and adaptive immunity

X Zhou, H Zhang *SeminNephrol* 44:151567 2025

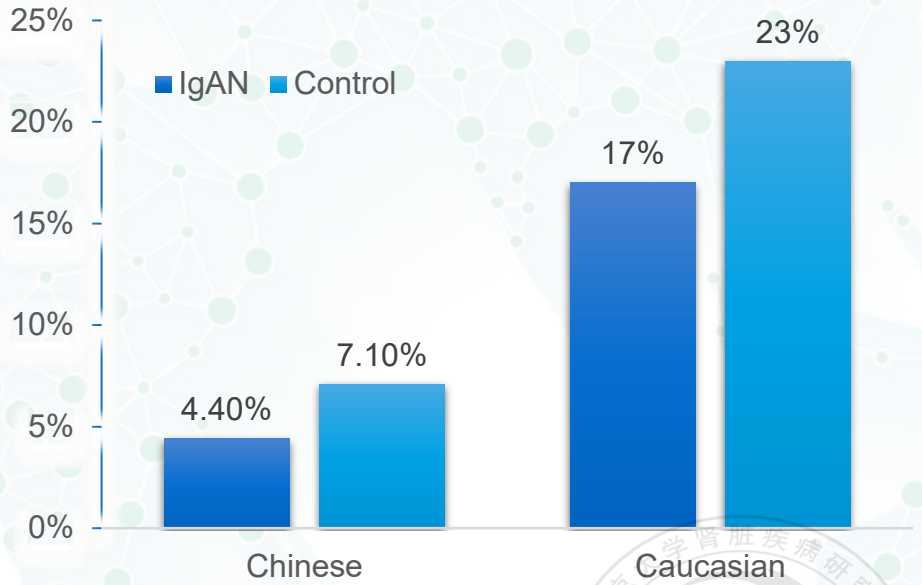
Genetic influence of risk for IgA nephropathy by race

Galactose deficient-IgA1 levels



Chinese **Caucasian**

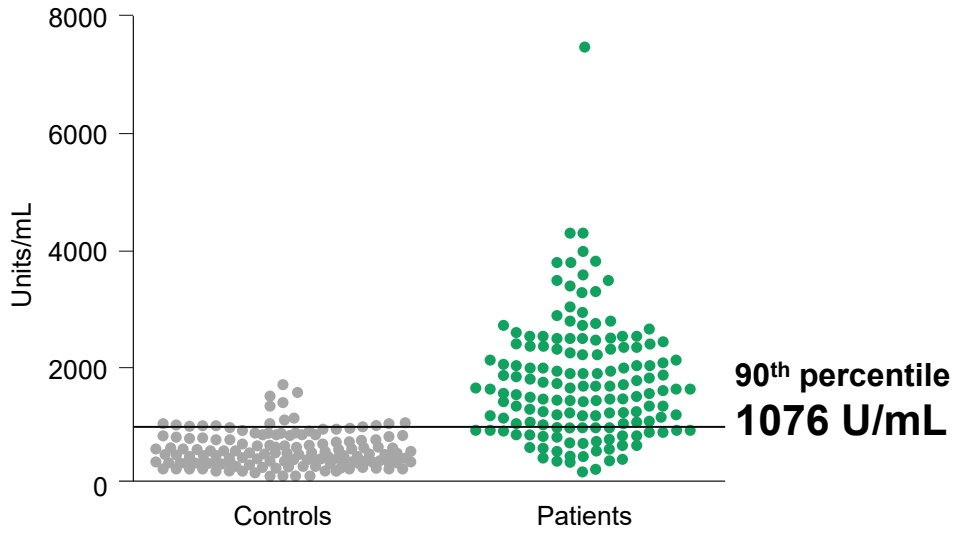
rs6677604 A allele (CFHR1,3Δ) frequency



Daniel P. Gale et al. JASN 2017;28:2158-2166
Gharavi AG, et al. Nat Genet. 2011;43(4):321-7.

Relationship of Gd-IgA1 and Therapeutic Response

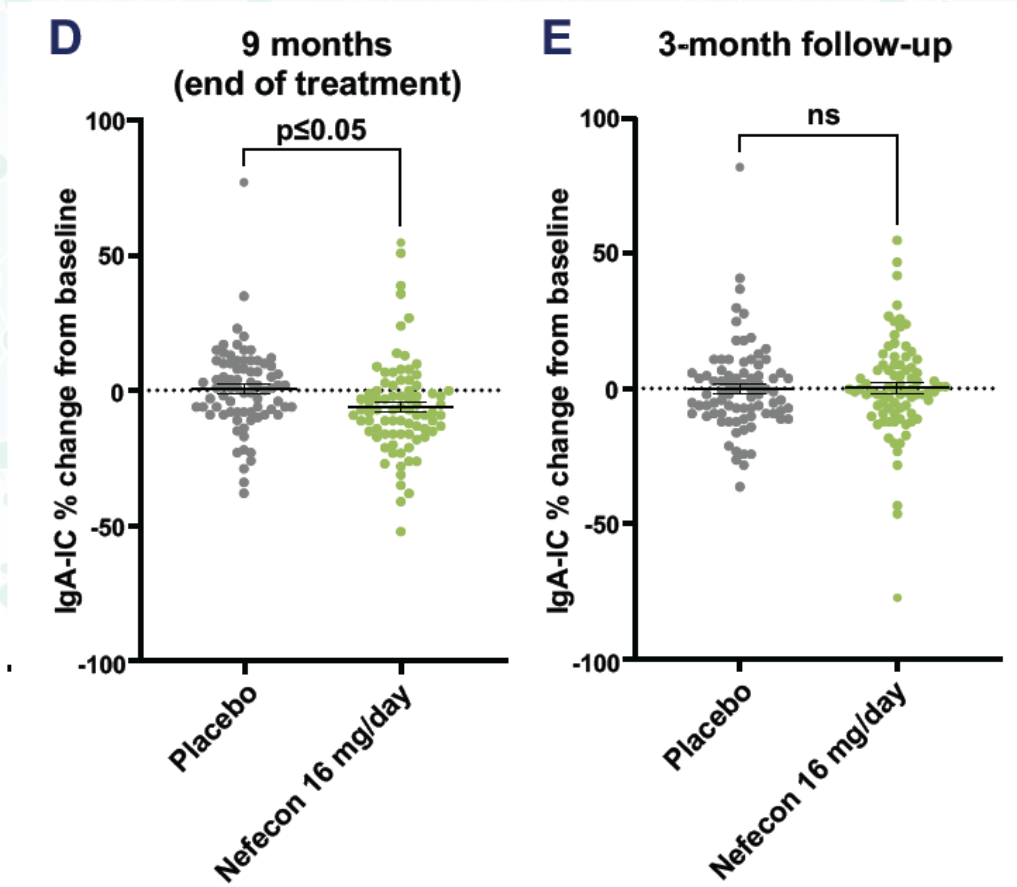
Serum Levels of Gd-IgA1 in Patients With IgAN and Healthy Controls[1]



HAA-IgA1 >1076 U/mL has PPV 88.6% and NPV 78.9%

Figure adapted from Moldoveanu et al. (2007)[1]

Nefecon and GdIgA1

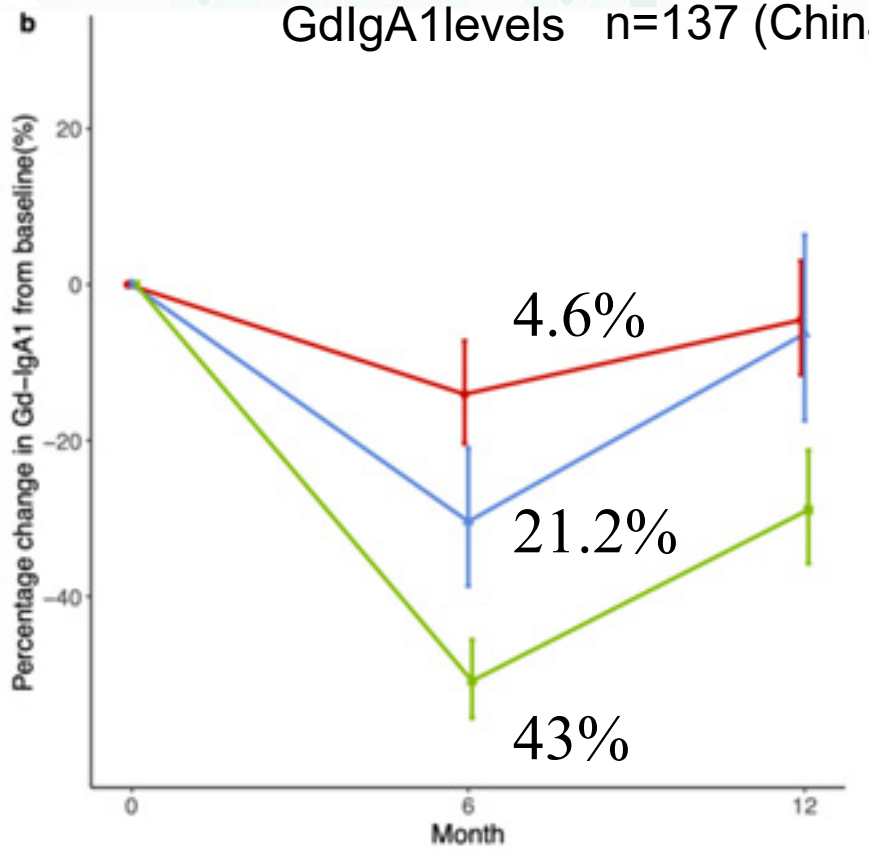


ERA Poster (2023)

1. Moldoveanu Z, et al. *Kidney Int*. 2007;71(11):1148-1154. 2. Suzuki Y, et al. *Clin Exp Nephrol*. 2014;18:770-777.

TESTING trial: Corticosteroids and circulating GdIgA1 levels

GdIgA1levels n=137 (China)

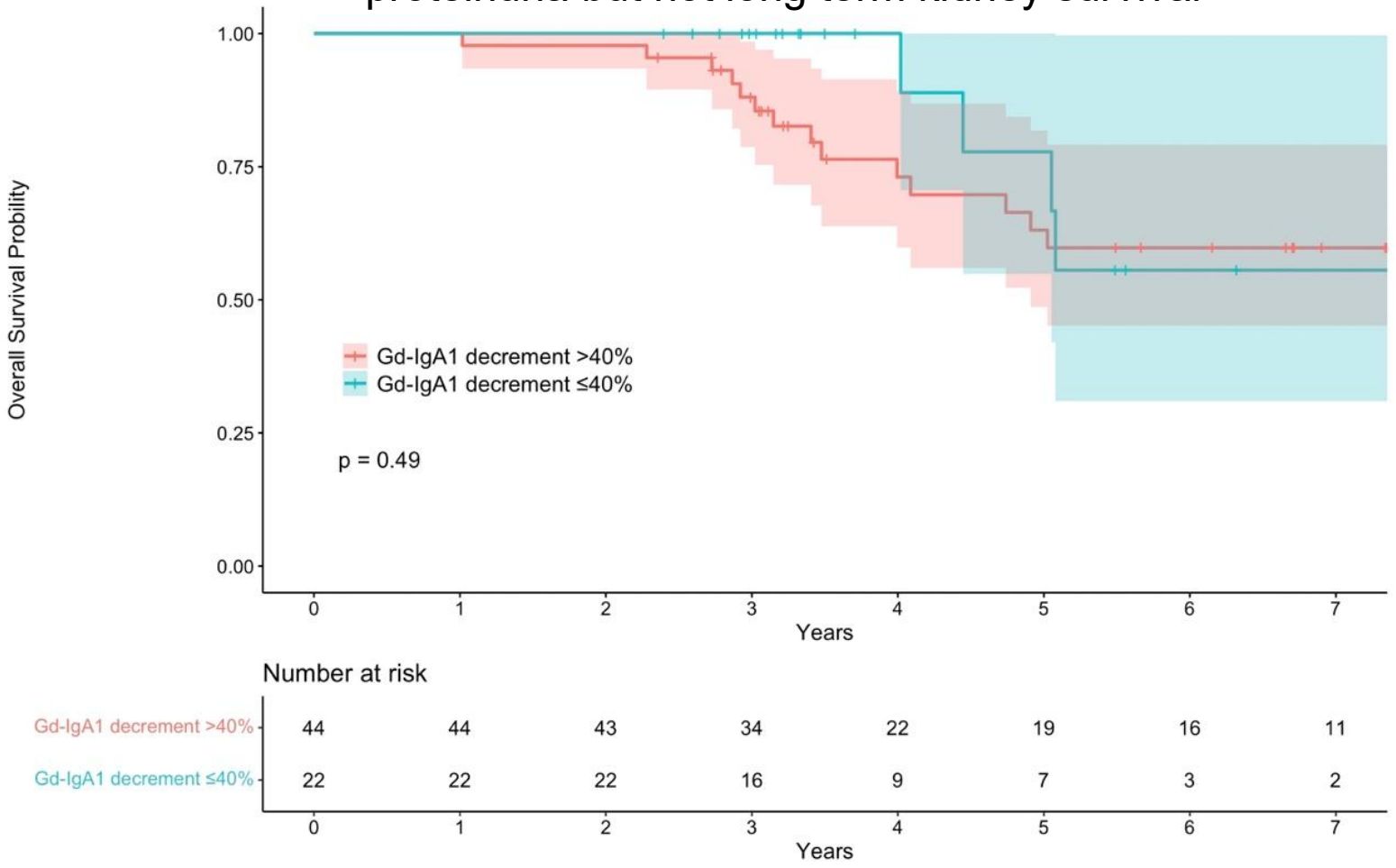


Placebo

Full dose

Reduced dose

Greater reduction in GdIgA1 at 6 months correlates to proteinuria but not long term kidney survival



Zan et al (Accepted for publication, CJASN

Targeted-release budesonide modifies key pathogenic biomarkers in immunoglobulin A nephropathy: Insights from the NEFIGAN trial

kidney INTERNATIONAL

ISN INTERNATIONAL SOCIETY OF NEPHROLOGY

Objective: To assess the effect of Nefecon on the various molecular mechanisms involved in the pathogenesis of IgAN

150 patients with **IgAN**

Randomized and dosed

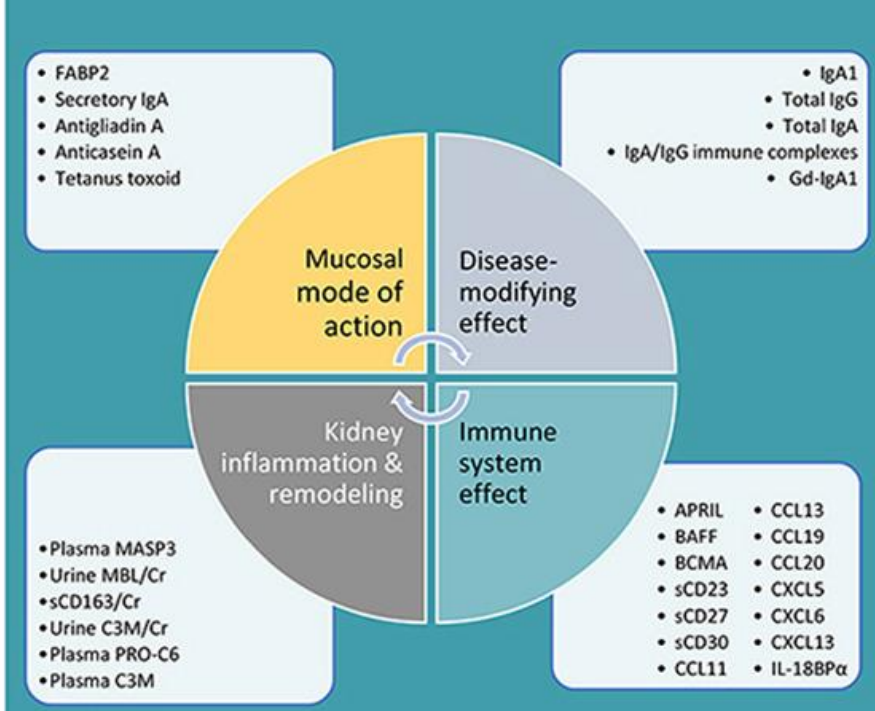
- **Nefecon** 8 mg/d: *n* = 51
- **Nefecon** 16 mg/d: *n* = 49
- **Placebo**: *n* = 50

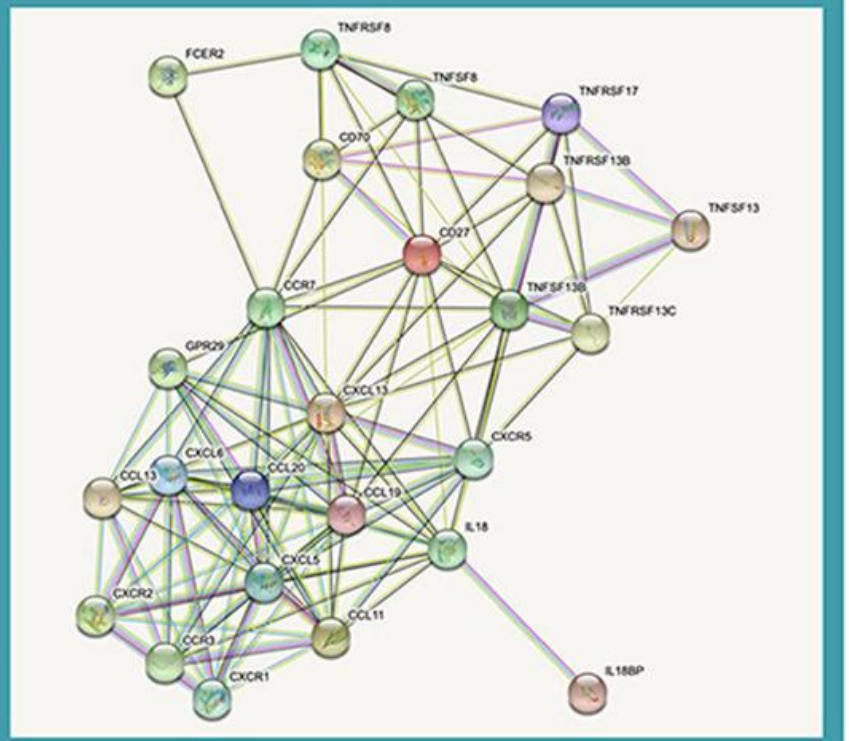
9 months of treatment → 3-month follow-up

Blood and urine samples

Robust regression analysis and protein-protein interactions network

Biomarkers with significant changes from baseline at 9 months with Nefecon 16 mg/d





Wimbury D, Muto M, et al. 2023

CONCLUSION

Nefecon exerts a direct disease-modifying effect in IgAN by targeting the GALT, regulating mucosal immune dysfunction and potentially modulating the systemic immune response

Urinary biomarkers for IgAN

Prospective clinical study

Urine proteomics for prediction of disease progression in patients with IgA nephropathy

Background

In IgA nephropathy (IgAN), predicting risk of rapid kidney disease progression is challenging

Current risk stratification tools require kidney biopsy; non-invasive markers are lacking

Methods

Multicenter
Europe (n=6)/Canada (n=1)

Biopsy-proven IgAN
(eGFR >20ml/min/1.73 m²)

IgAN237:
- Urine proteome profiling
- 237 discriminatory peptides

Outcome: eGFR slope
- Annual change in eGFR after baseline
- Divided into tertiles

Results

Group	Baseline eGFR (ml/min/1.73 m²)	eGFR slope (ml/min/1.73 m²)	Progression to ESKD
Fastest progression n=70	64.9 (30.8)	−6.4 (95% CI −8.8 to −5.2)	20%
Intermediate progression n=69	62.8 (29.5)	−1.4 (95% CI −1.8 to −1.1)	6%
Slowest progression n=70	60.7 (26.6)	+3.4 (95% CI 2.3 to 4.7)	0%
	p=0.842	P<0.001	P<0.001

AUC for fast progression

Clinical parameters alone
0.72
(95% CI 0.64–0.81)

IgAN237 + clinical parameters
0.89
(95% CI 0.83–0.95)

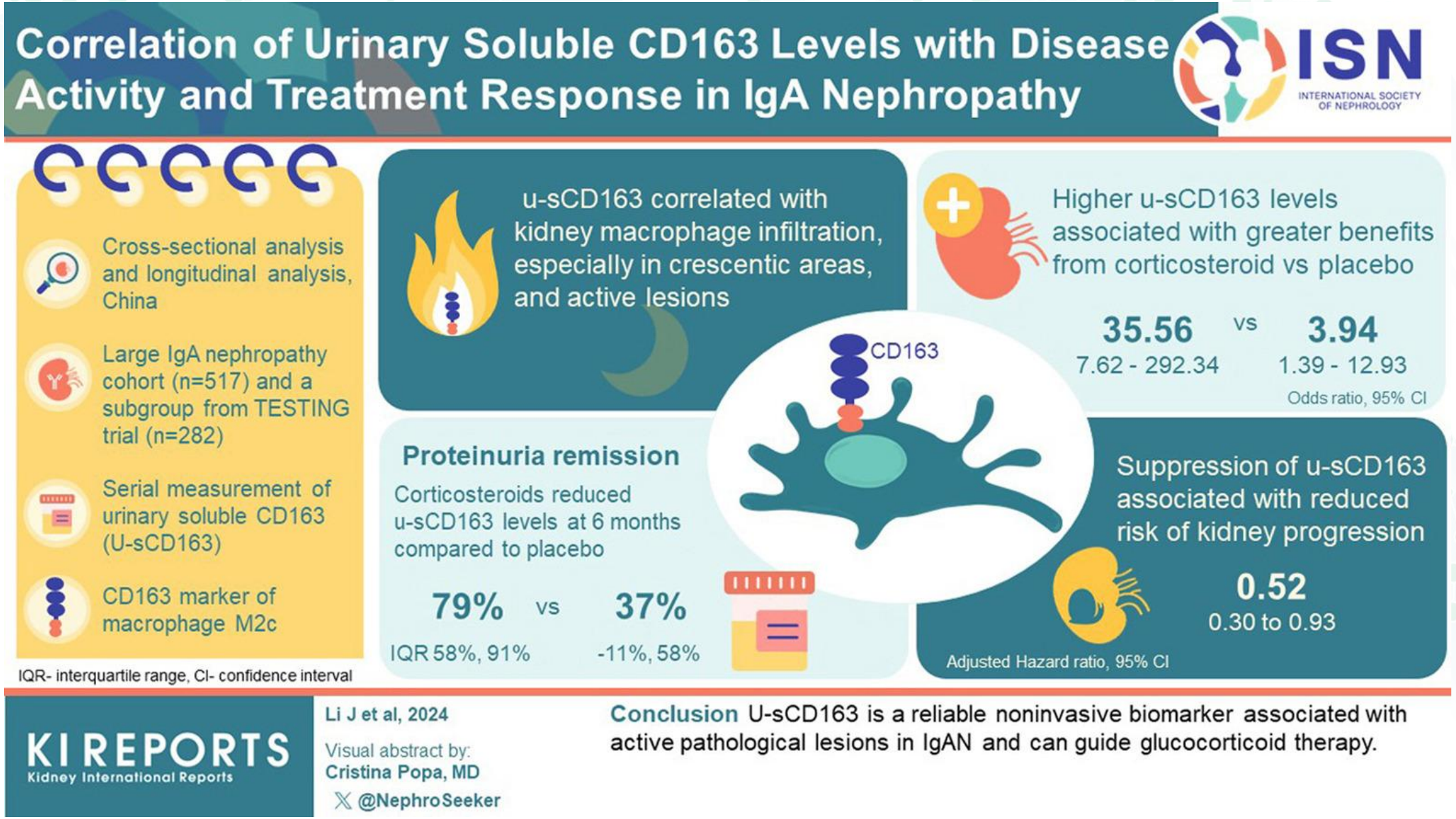
Conclusion

In IgAN, a urinary peptide classifier (IgAN237) along with clinical parameters predicts progressive loss of kidney function better than clinical parameters alone.

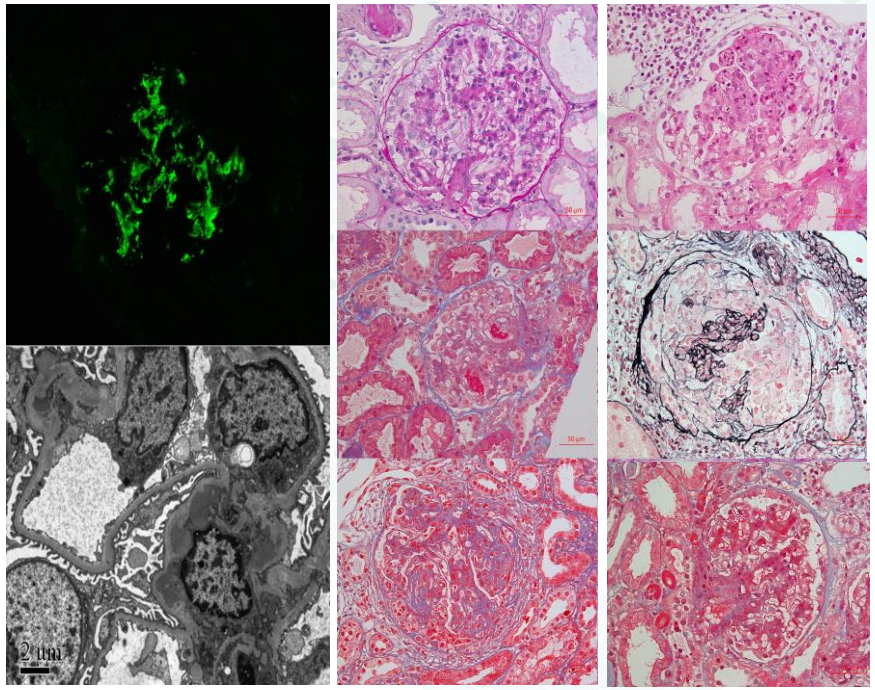
ndt NEPHROLOGY DIALYSIS TRANSPLANTATION
era EDTA

Rudnicki, M., et al. NDT (2020)
@NDTSocial

Urinary CD163 in predicting disease activity and treatment response in IgAN



Clinical-pathological presentation in IgAN is highly variable



- Histopathology and clinical presentation: highly variable

KDIGO

Practice points: ..there are no data from clinical studies that show patients prospectively randomized to particular treatment regimens based on their Oxford Classification MEST-C scores have better clinical outcomes. In particular:

Practice points: ..

.....insufficient evidence to base treatment decisions based on histology…….Histopathologic features must be interpreted in the context of clinical features, in particular the rate of change in eGFR.

Al-Lawati AI et al. Nephrol Dial Transplant. 2017;32:i30–i36;
Tan M et al. Kidney Blood Press Res. 2015;40:200–206.

Predictive value of the Oxford Classification for the effect of glucocorticoid therapy in IgA nephropathy

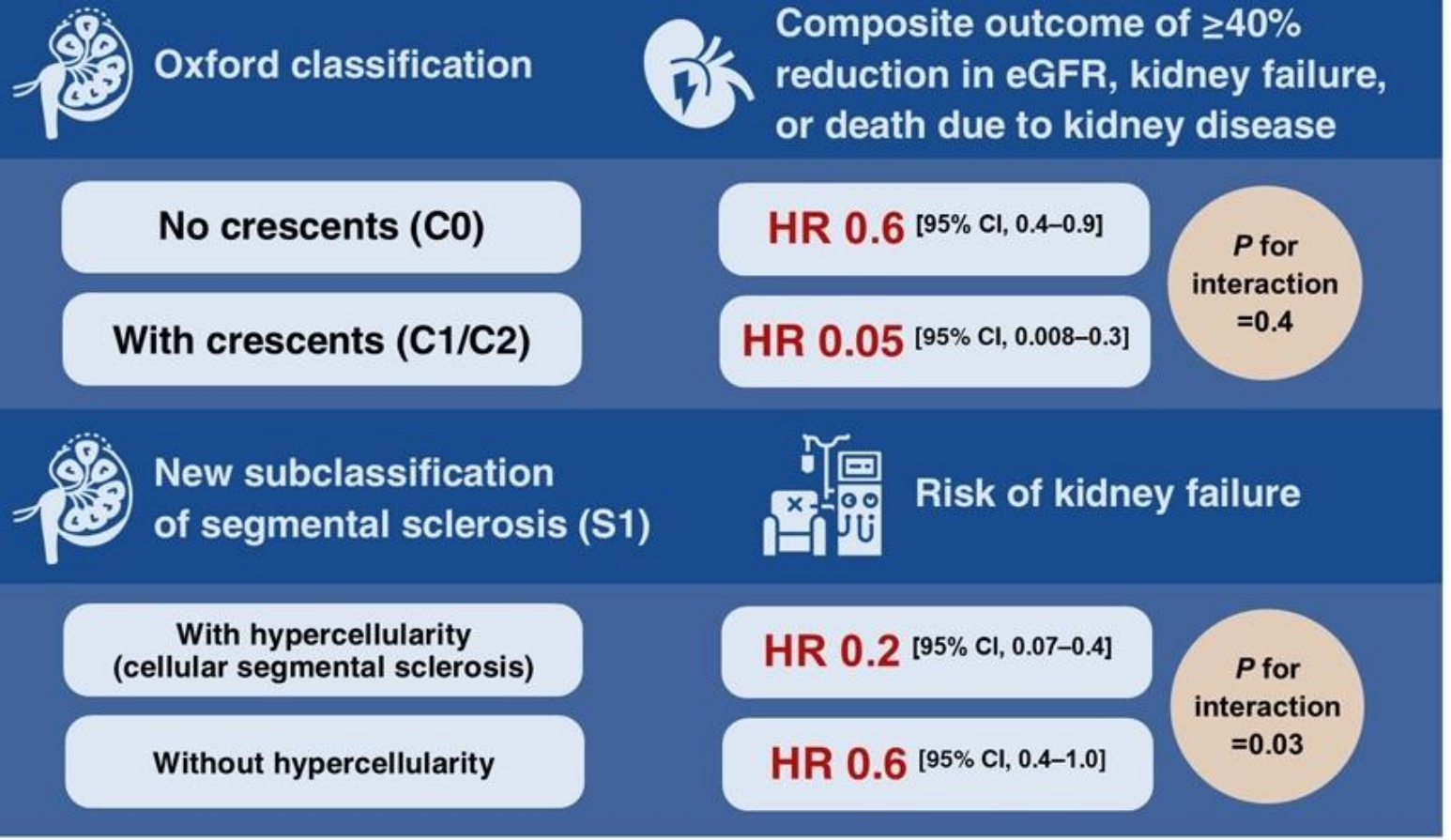
JASN
Journal of the American Society of Nephrology
Clinical Research

Secondary analysis of TESTING trial

N=279
Chinese participants with kidney biopsy slides available for central pathology review

Glucocorticoid therapy

Median Bx → Rx = 4 months



Conclusions: Crescents and cellular segmental sclerosis in IgA nephropathy suggested a favorable response to glucocorticoid therapy.

Sufang Shi, Ian S.D. Roberts, Zixuan Wang, et al. ***Predictive Value of the Oxford Classification for the Effect of Glucocorticoid Therapy in IgA Nephropathy***. JASN DOI: 10.1681/ASN.0000000796. **Visual Abstract by Paolo Nikolai So, MD**

Disagreement between local and central pathology review

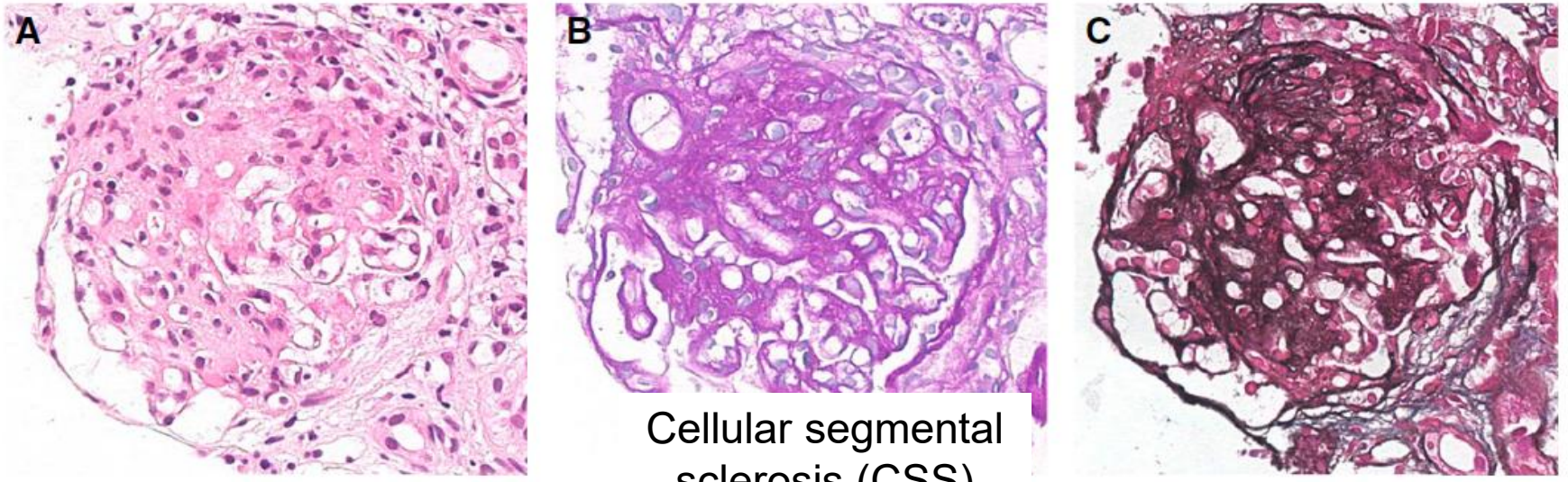
Pathology lesions	Central pathological review	Local pathology report	Kappa value
M1(%)	89/279(32)	160/272(59)	0.1
E1(%)	75/279(27)	82/279(29)	0.2
S1(%)	239/279(86)	200/272(74)	0.1
T0(%)	173/279(62)	114/272(42)	0.3
T1(%)	84/279(30)	110/272(40)	0.3
T2(%)	22/279(8)	48/272(18)	0.3
C0(%)	230/279(82)	102/274(37)	0.1
C1(%)	48/279(17)	127/274(46)	0.1
C2(%)	1/279(0.4)	45/274(16)	0.1

M, mesangial hypercellularity; E, endocapillary hypercellularity;S, segmental sclerosis; T, tubular atrophy/interstitial fibrosis; C, cellular/fibrocellular crescents.

Shu et al JASN 00: 1–10, 2025. doi: https://doi.org/10.1681/ASN.0000000796

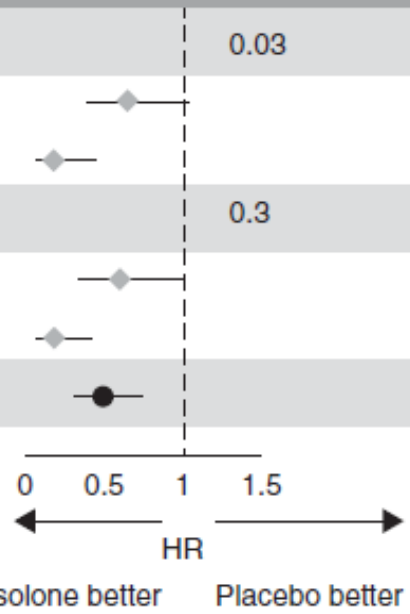
Subtype of S lesions

Cellular segmental sclerosis



Cellular segmental sclerosis (CSS)

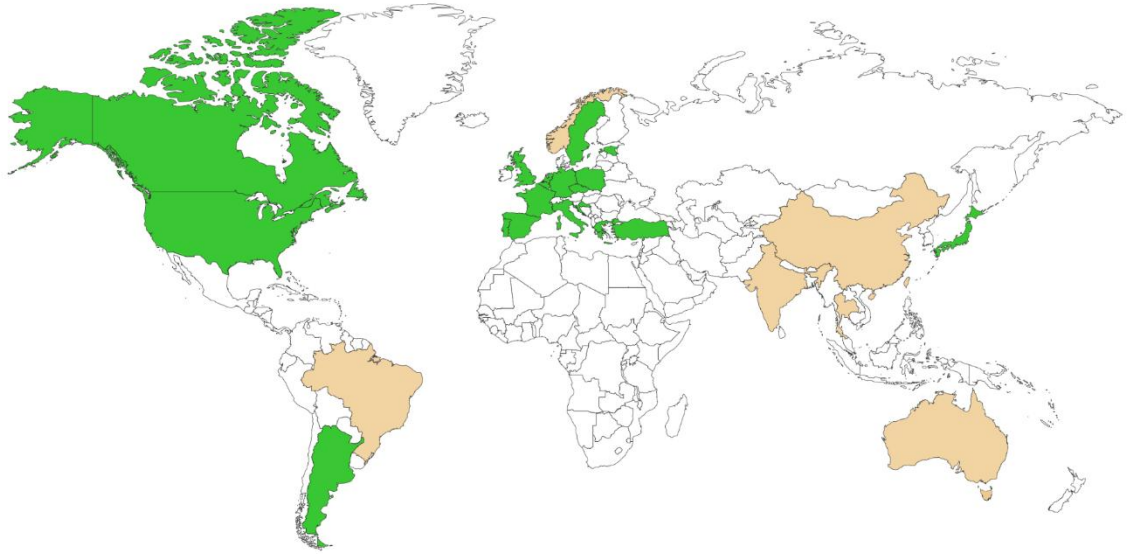
Characteristic	No./total No. (%) Methylprednisolone	Placebo	Hazard ratio (95%CI)	*P* for interaction
Cellular segmental sclerosis				0.03
No	37/104(36)	44/103(43)	0.6(0.4-1.0)	
Yes	11/35(31)	21/37(57)	0.2(0.07-0.4)	
podocytopathic segmental sclerosis				0.3
No	30/109(28)	39/102(38)	0.6(0.4-1.0)	
Yes	18/30(60)	26/38(68)	0.2(0.07-0.4)	
Overall	48/139(35)	65/140(46)	0.5(0.3-0.7)	



Shu et al JASN 00: 1–10, 2025. doi: https://doi.org/10.1681/ASN.0000000796

AI4gAN study

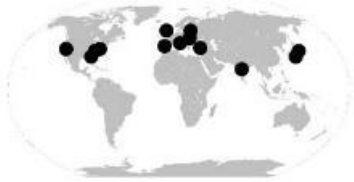
Data from > 150 centers & 26 countries



LaBooratory of Nephropathology

www.labooratory.ukaachen.de

Large dataset



>3000 IgAN biopsies
Clinical follow-up data

Ground truth

Quality checks

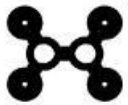
AI model development

- Disease progression
- Treatment response
- IgAN vs. IgAV

AI data integration

IHC/IF

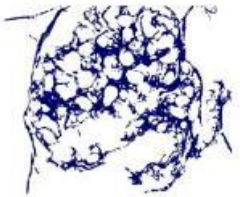
Multiple Stains



Clinical data

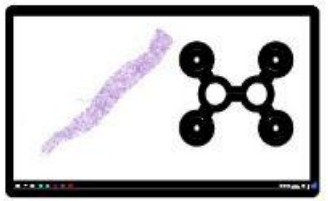
Electron Microscopy

AI model explainability



Visualization of predictions

Implementation



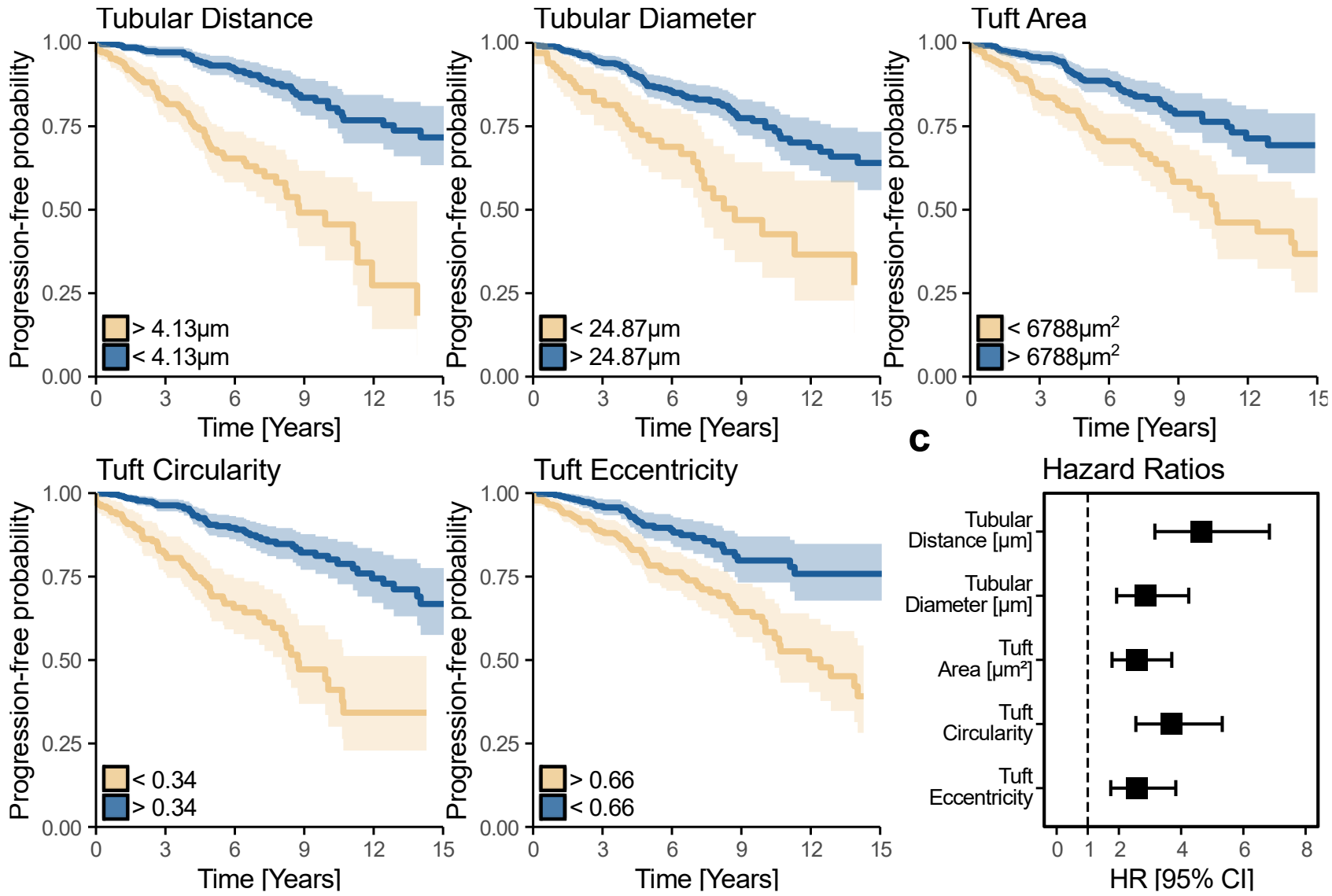
- Validation
- In-house IVD

Pathomics as prognostic biomarker in IgAN (VALIGA)

Multivariate Cox proportional hazards analysis: Adjusted for age, sex, eGFR at time of biopsy & MEST-C score

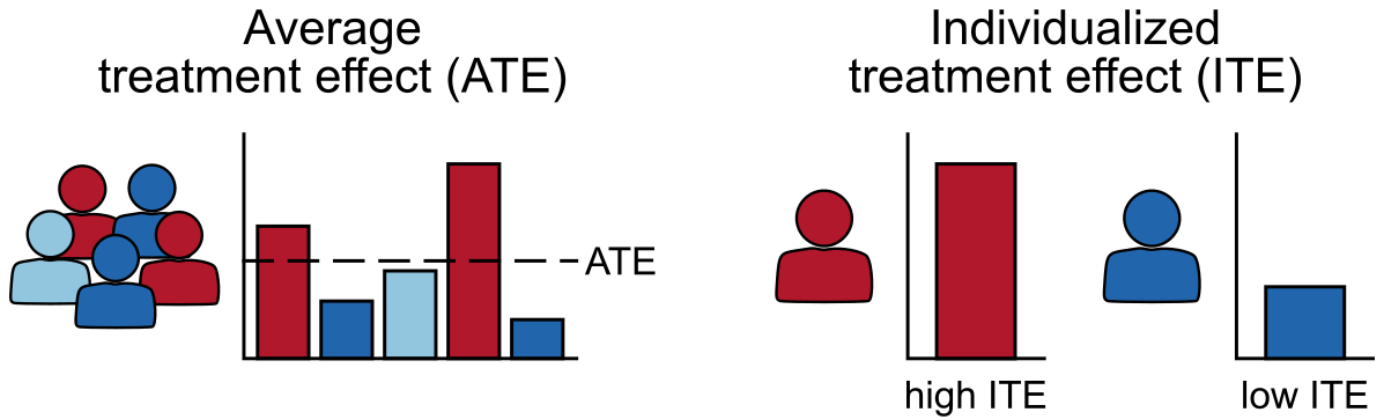
Five morphometric features are independent predictors of IgAN progression

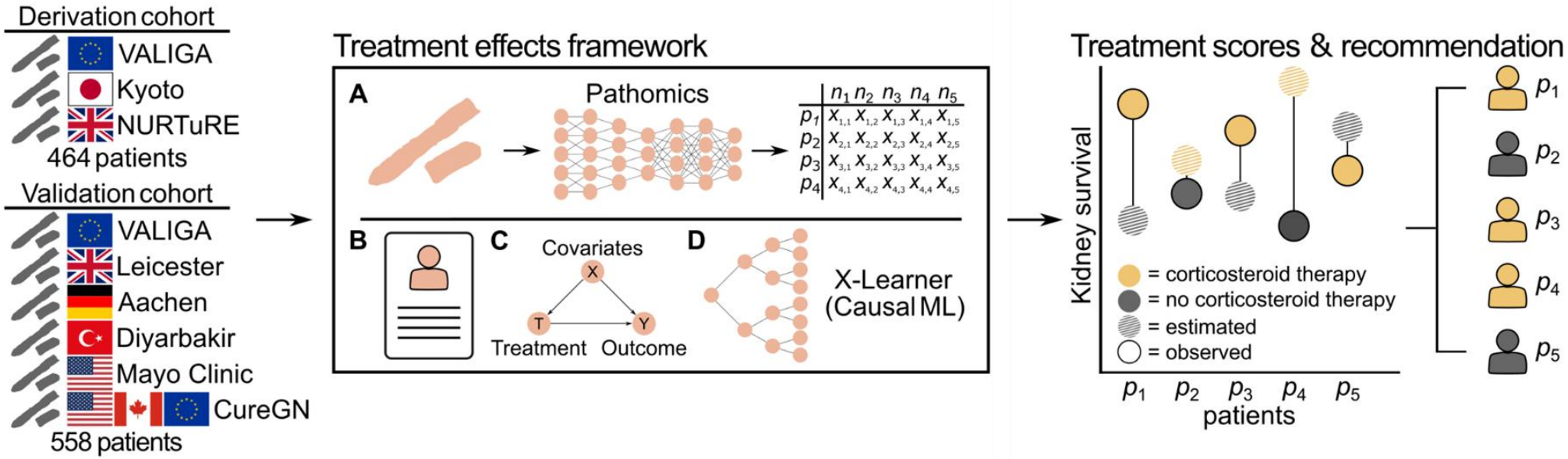
1. **Tubular distance**
2. **Tubular diameter**
3. **Tuff area**
4. **Tuff circularity**
5. **Tuff Eccentricity**



Hölscher et al., Nat Commun 2023

Pathomics for individual treatment response in IgAN

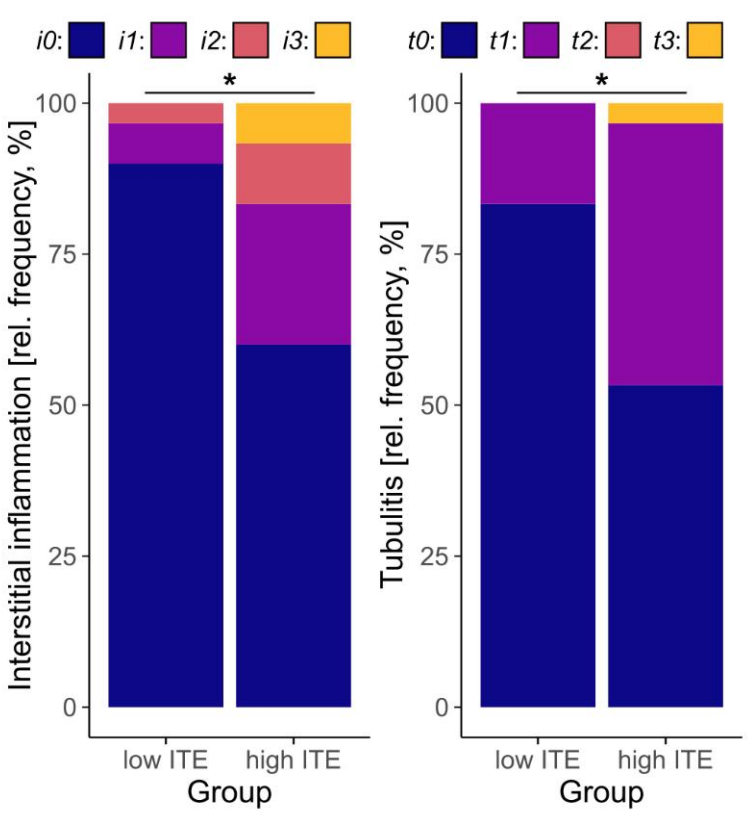


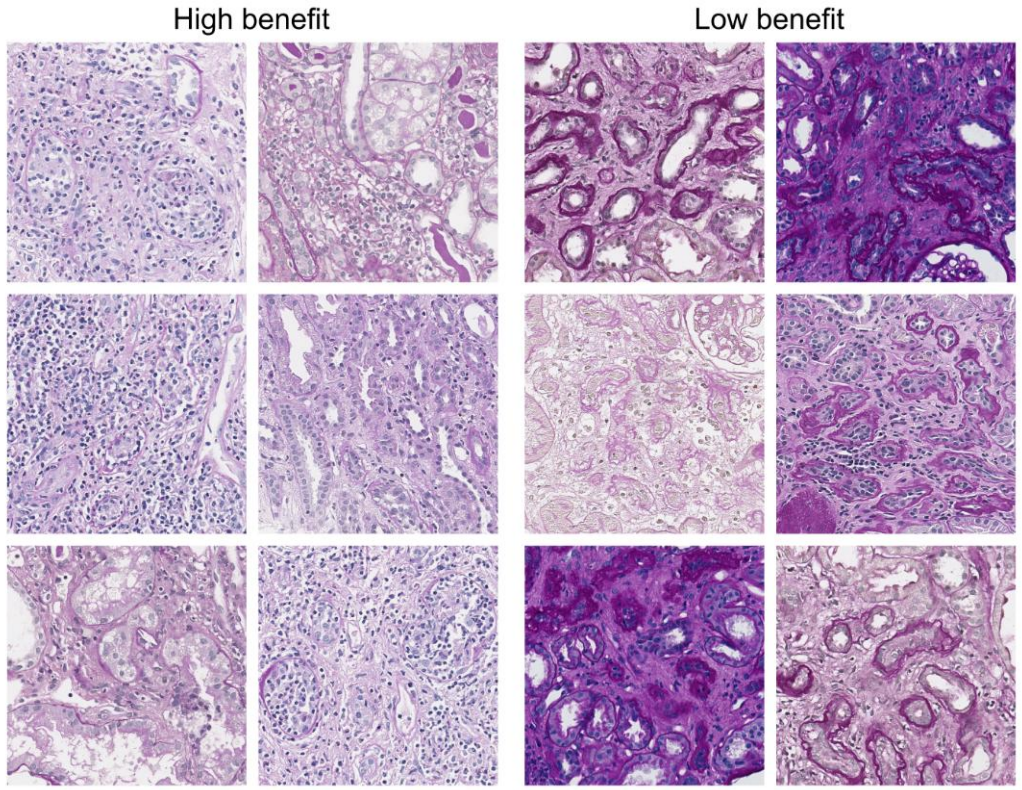


https://www.medrxiv.org/content/10.1101/2025.10.08.25337548v1

Pathomics can identify predictors of treatment

Tubulointerstitial inflammation





Potential reduction of corticosteroid therapy by 67.3%

Precision medicine framework (for any treatment)

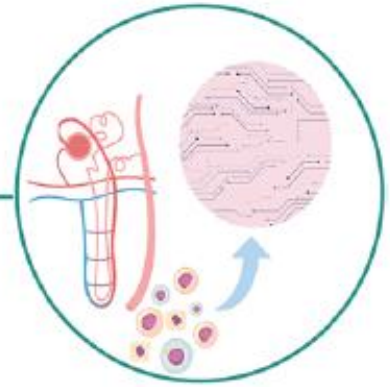
https://www.medrxiv.org/content/10.1101/2025.10.08.25337548v1

REMODEL trial: Mechanistic trial in DKD

Assess change in functional MRI end points

co-primary outcomes (MRI) based, including change in kidney oxygenation, perfusion and inflammation

Integrate single-nucleus and spatial transcriptomics from kidney tissue



Semaglutide

REMODEL

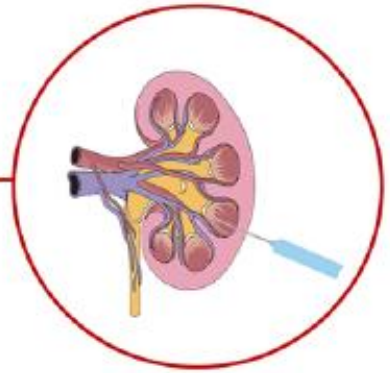
vs

placebo

Bridge to clinical parameters of kidney function

N=33, 2 kidney biopsies

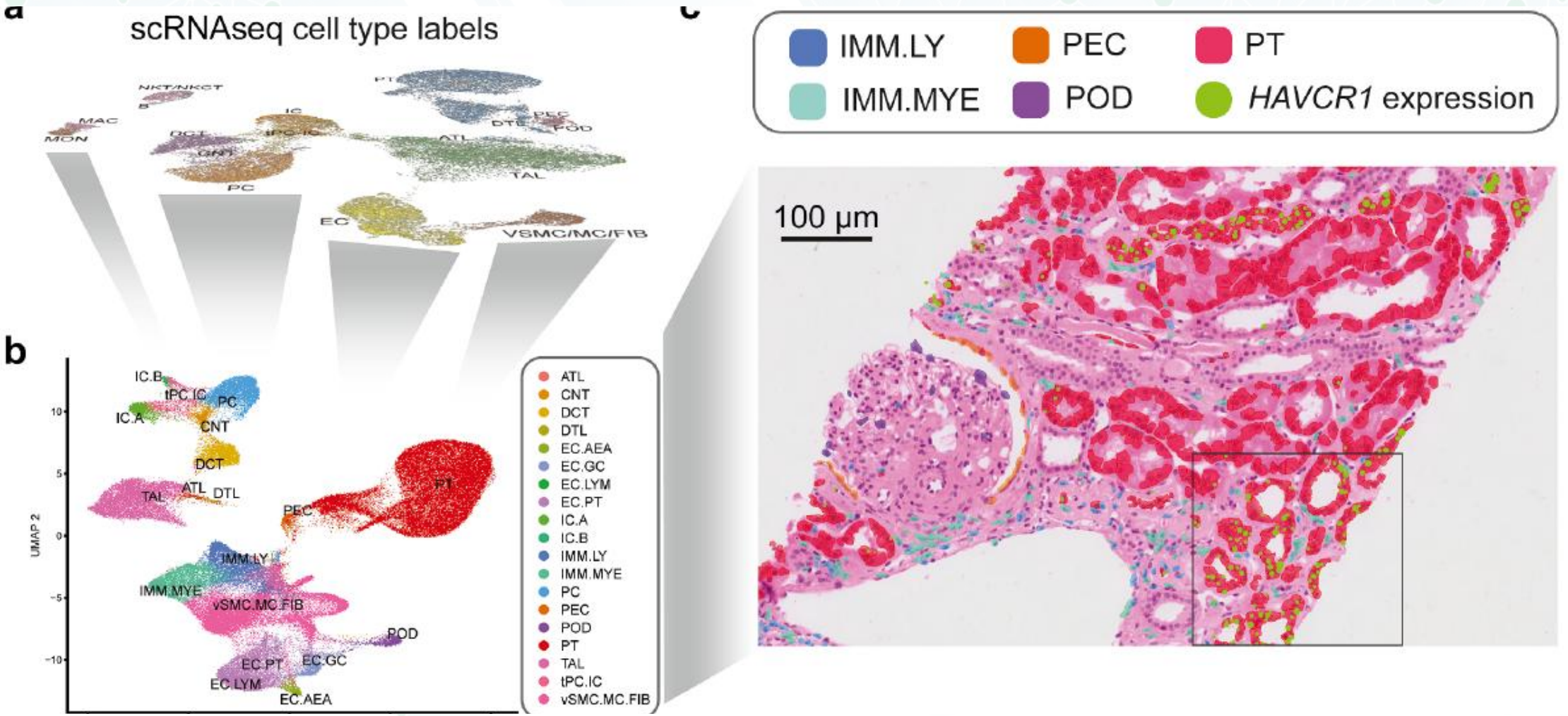
Correlate with morphologic changes (histology)



THE UNIVERSITY OF SYDNEY

Cherney et al./, *Nephrol Dial Transplant* , 2025, **40** , 2182–2192

Advance transcriptomic analysis for MOA in DKD



Pruijm et al Kidney International (2025)

SPARTAN (NCT0466320) Study Design

SPARTAN

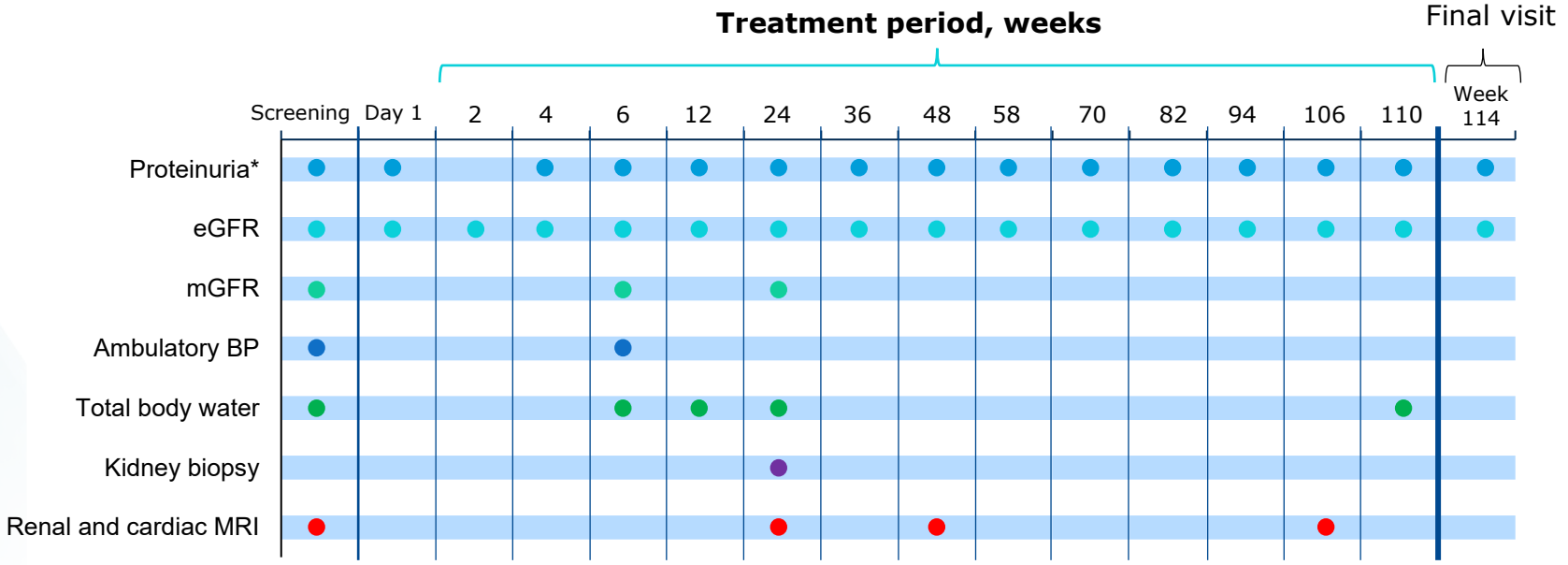
Screening	Day 1 to Week 2	Weeks 3 to 110	Weeks 110 to 114
	SPARSENTAN 200 mg	SPARSENTAN 400 mg	Follow-up
N=12			Start SOC RASB therapy

Key Eligibility Criteria

- Age ≥18 years
- Biopsy-proven IgAN within ≤6 months
- Proteinuria ≥0.5 g/day
- eGFR ≥30 mL/min/1.73 m^2
- No ACEIs/ARBs within ≤12 months

Key Endpoints

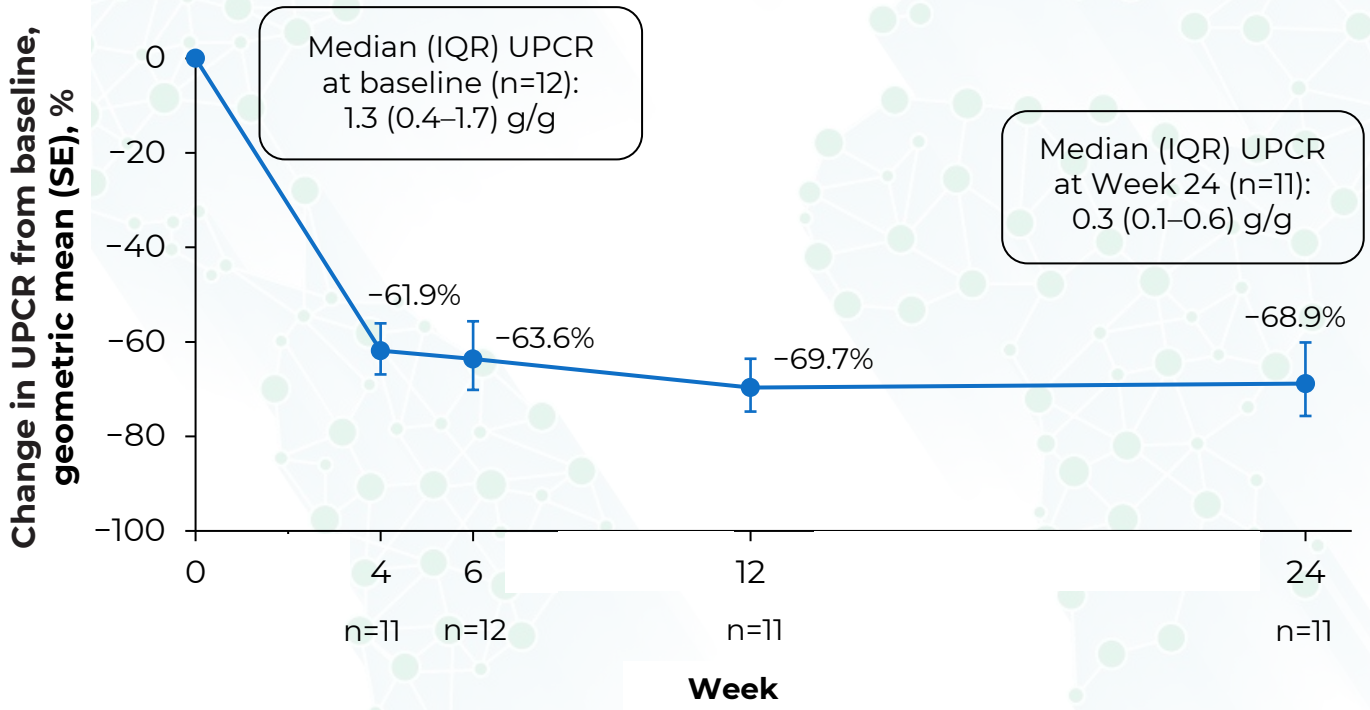
- Safety
- Change in proteinuria from baseline
- Complete remission of proteinuria (<0.3 g/day)
- Change in GFR and BP from baseline



ACEI, angiotensin-converting enzyme inhibitors; ARB, angiotensin receptor blocker; BP, blood pressure; eGFR, estimated glomerular filtration rate; IgAN, immunoglobulin A nephropathy; mGFR, measured glomerular filtration rate; MRI, magnetic resonance imaging; RASB, renin-angiotensin-system blockers; SOC, standard of care.
*24-hour collection.

SPARTAN trial: proteinuria results

Change in UPCR from baseline* to Week 24



- Proteinuria reductions were **rapid** (≈60% from baseline at Week 4) and **sustained** over 24 weeks of sparsentan treatment

Proteinuria results are comparable with those from the PROTECT study

Sparsentan is indicated for the treatment of adults with primary IgAN with urine protein excretion ≥1.0 g/day (or UPCR ≥0.75 g/g).
*On-treatment analysis; one patient discontinued after Week 6.
IgAN, immunoglobulin A nephropathy; IQR, interquartile range; SE, standard error; UPCR, urine protein-to-creatinine ratio.
Cheung CK, et al. ASN 2024; Oral presentation #FR-OR63.

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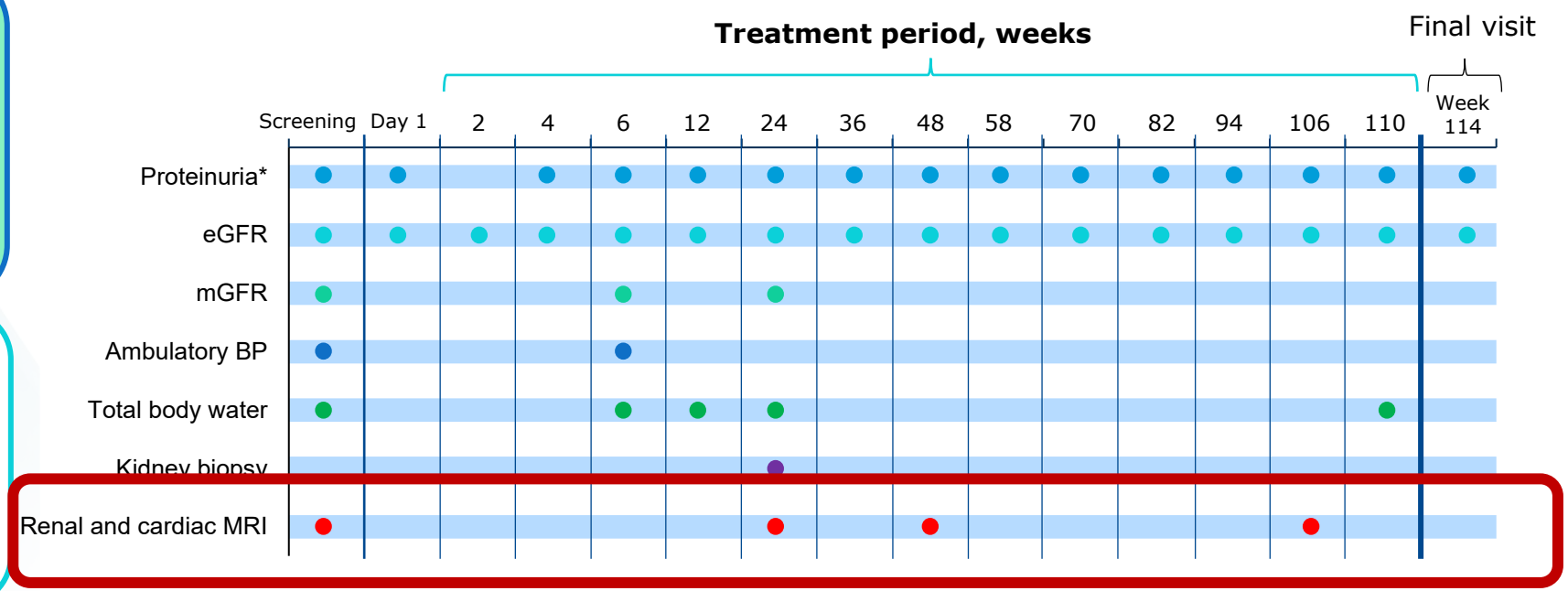
- Change in proteinuria from baseline
- Complete remission of proteinuria (<0.3 g/day)
- Change in GFR and BP from baseline



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*24-hour collection.

Current and Emerging Therapies for IgA Nephropathy

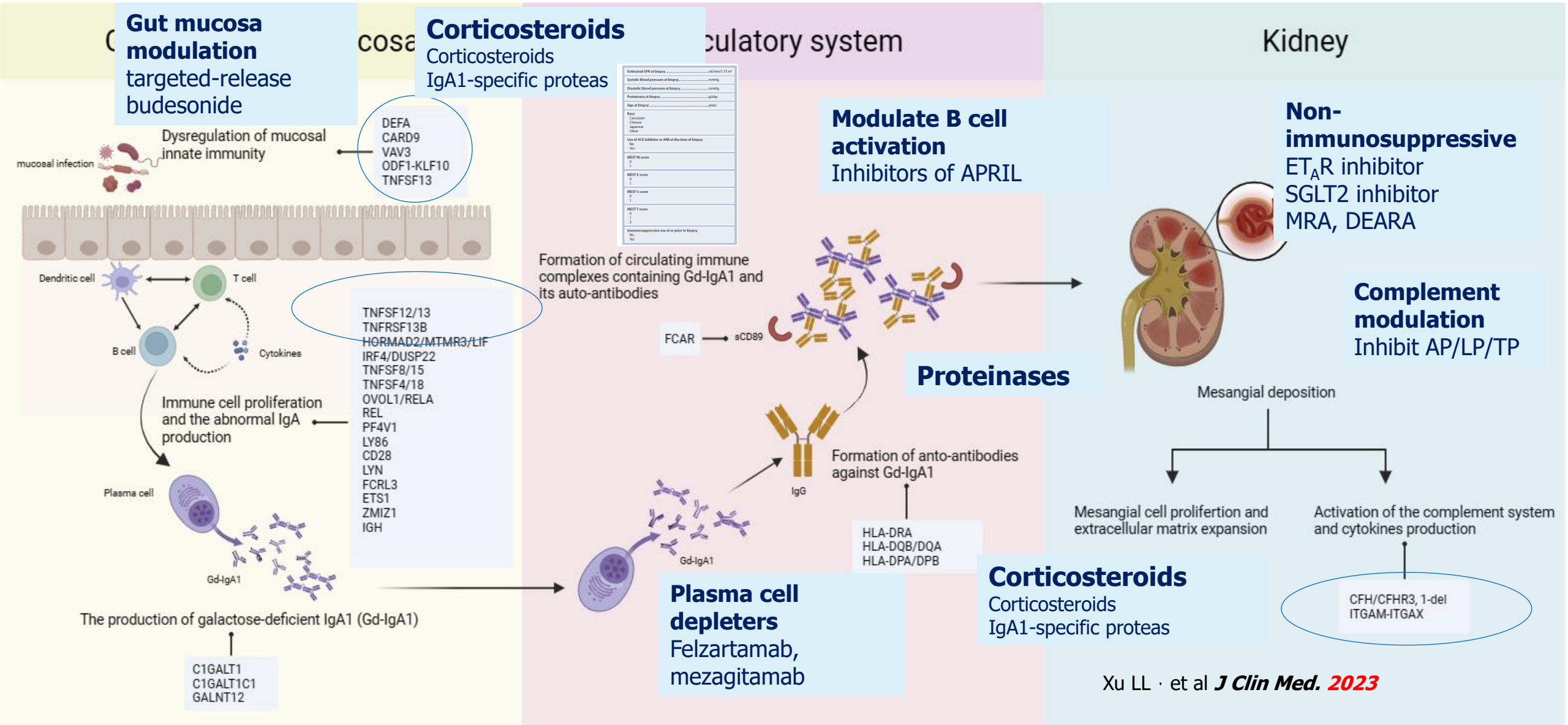
Trial Drug	Phase	Drug Class	Proteinuria Reduction	eGFR (mL/min/1.73 m^2) vs Placebo	Clinical Trial
Tarpeyo[1,2] (Nefecon)	Approved	Glucocorticoid	31% (*P*=0.0001)	-2.47 vs -7.52 (*P*<0.0001)	NefIgArd (Phase 3)
Filspari[3,4] (sparsentan)	Approved	Dual ET_A/ANG-II antagonist	49.8% (*P*<0.0001)	-2.9 vs -3.9 with irbesartan (*P*=0.058)	PROTECT (Phase 3)
Fabhalta[5-7] (iptacopan)	Approved	Complement factor B inhibitor	38% (*P*<0.001)	+2.4 vs -3.3[a]	APPLAUSE-IgAN (Phase 3) NCT03373461 (Phase 2)
Vanrafia[8,9] (atrasentan)	Approved	ET_A-1 receptor antagonist	36.1% (*P*<0.001)	Pending results	ALIGN (Phase 3)
Voyxact[10,11] (sibeprenlimab)	Approved	APRIL inhibitor	51.2% (*P*<0.0001)	+0.2 vs -7.4[a]	VISIONARY (Phase 3, IA)[b] ENVISION (Phase 2)
Atacicept[12,13]	Phase 3	Dual APRIL/BAFF inhibitor	42% (*P*<0.0001)	Absolute difference of 5.7[a]	ORIGIN (Phase 3, IA) ORIGIN (Phase 2b)
Povetacicept[14,15]	Phase 3	Dual APRIL/BAFF inhibitor	64%	Absolute change of +3.3[a]	RUBY-3 (Phase 1b/2a) RAINIER (Phase 3)
Telitacicept[16,17]	Phase 3 *(China only)*	Dual APRIL/BAFF inhibitor	55%	-1.0 vs -7.7	Phase 3
Felzartamab[18]	Phase 3	Anti-CD38	24%	Treatment difference of 8.8[a]	IGANZ (Phase 2)
Mezagitamab[19]	Phase 1b	Anti-CD38	55%	Mean change of +2.5[c]	Phase 1b Phase 3

Note: All data presented are based solely on published and cited scientific sources and avoid any comparison of the listed drugs. At present, there have been no head-to-head studies comparing any of the listed drugs.

[a]Data from Phase 2 trial. [b]Phase 3 trial is ongoing. [c]Data from Phase 1b trial.
ANG-II, angiotensin II; APRIL, a proliferation-inducing ligand; BAFF, B-cell activating factor; eGFR, estimated glomerular filtration rate; ET_A, endothelin receptor A; IA, interim analysis; IgA, immunoglobulin A.

1. Lafayette R, et al. *Lancet*. 2023;402(10405):859-870. 2. Tarpeyo® [prescribing information]. Stockholm, Sweden: Calliditas Therapeutics AB; 2024. 3. Rovin BH, et al. *Lancet*. 2023;402(10417):2077-2090. 4. Filspari® [prescribing information]. San Diego, CA: Travere Therapeutics, Inc.; 2024. 5. Perkovic V, et al. *N Engl J Med*. 2025;392(6):531-543. 6. Fabhalta® [prescribing information]. East Hanover, NJ: Novartis Pharmaceuticals Corporation; 2025. 7. Zhang H, et al. *Kidney Int*. 2024;105(1):189-199. 8. Heerspink HJL, et al. *N Engl J Med*. 2025;392(6):544-554. 9. Vanrafia® [prescribing information]. East Hanover, NJ: Novartis Pharmaceuticals Corporation; 2025. 10. Mathur M, et al. *N Engl J Med*. 2024;390(1):20-31. 11. VOYXACT (sibeprenlimab-szsi) [prescribing information]. Rockville, Maryland; Otsuka America Pharmaceutical, Inc; 2025. 12. Lafayette R, et al. *Kidney Int*. 2024;105(6):1306-1315. 13. Vera Therapeutics announces atacicept achieved 46% proteinuria reduction in ORIGIN Phase 3 trial in adults with IgA nephropathy [press release]. Vera Therapeutics. June 2, 2025. Accessed July 30, 2025. https://ir.veratx.com/news-releases/news-release-details/vera-therapeutics-announces-atacicept-achieved-46-proteinuria. 14. Tumlin J, et al. ASN Kidney Week. 2025 (abstr SA-OR091). 15. Evaluation of efficacy of povetacicept in adults with immunoglobulin A nephropathy (IgAN). ClinicalTrials.gov. Accessed July 30, 2025. https://clinicaltrials.gov/study/NCT06564142. 16. Efficacy and Safety of Telitacicept in IgAN. ClinicalTrials.gov. Accessed November 19, 2025. https://clinicaltrials.gov/study/NCT06654596. 17. Lv, Jicheng, et al. ASN Kidney Week. 2025. (abstr SA-OR083). 18. Floege J, et al. *Kidney Int*. 2025:S0085-2538(25)00488-0. 19. Takeda Presents New Data Showing Mezagitamab (TAK-079) Sustained Effect on Kidney Function 18 Months After Treatment in Primary IgA Nephropathy [pres release[. Takeda. November 7, 2025. https://www.takeda.com/newsroom/newsreleases/2025/new-data-mezagitamab/.

Personalised medicine in IgA nephropathy



Xu LL · et al ***J Clin Med.*** ***2023***

Summary

There are needs for personalized approach in management of IgA nephropathy

Evolving field in risk prediction tool, genomic, pathomic and biomarkers.

No single biomarker yet but any marker/s needs to risk stratify and predict treatment response

Collaborative future research with industry partnership is the way to move forward


Thank you for your attention