Travere Therapeutics Corporate Overview

November 2026

Forward-Looking Statements

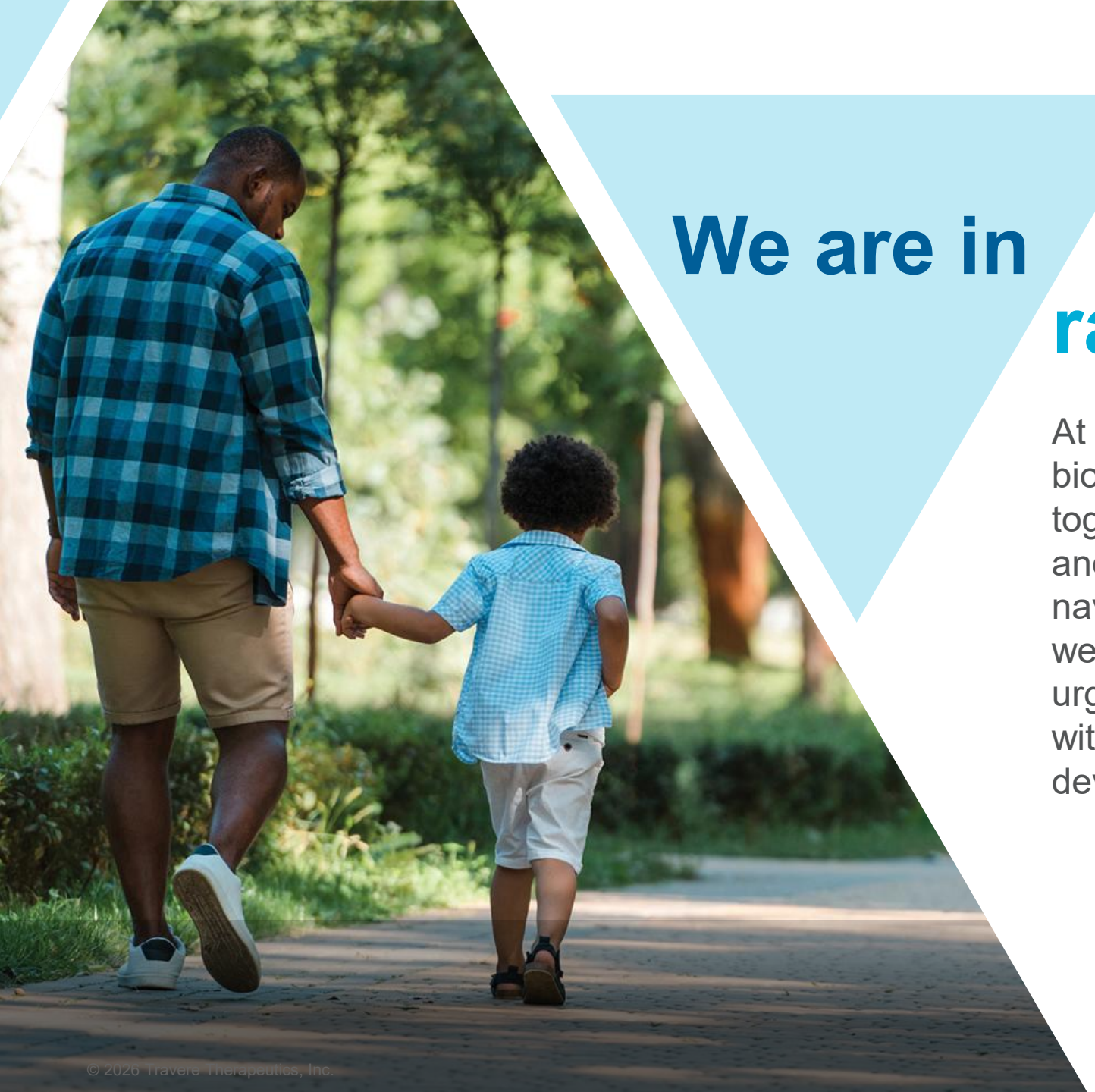
This presentation contains forward-looking statements, including but not limited to statements about: continued progress with FILSPARI in IgAN and in the commercial launch of FILSPARI in FSGS; statements regarding our products and products in development as potential best-in-class treatments, foundational treatments and/or treatment standards; estimates regarding the size of commercial opportunities; statements regarding the advancement of our pipeline throughout the year; statements and expectations regarding our pivotal Phase 3 HARMONY Study, including expectations regarding enrollment and data, and the timing and outcome thereof; statements and expectations regarding the other clinical studies and data described herein; statements regarding additional development and regulatory milestones; statements and expectations regarding civorebrutinib and our transaction with Everest Medicines, including statements regarding the characteristics and potential efficacy, safety profile, and differentiators of civorebrutinib with respect to the diseases and conditions described herein; statements regarding potential future milestone and royalty payments; statements regarding potential changes to treatment paradigms; statements and expectations regarding the activities of our partners and collaborators; statements regarding estimates of prevalence and potential addressable market sizes; and statements regarding financial metrics and expectations related thereto. These forward-looking statements may be accompanied by such words as “anticipate,” “believe,” “estimate,” “expect,” “forecast,” “intend,” “may,” “plan,” “project,” “schedule,” “target,” “will,” and other words and terms of similar meaning. You should not place undue reliance on these statements.

These statements involve risks and uncertainties that could cause actual results to differ materially from those reflected in such statements. Among the factors that could cause actual results to differ materially from those indicated in the forward-looking statements are risks and uncertainties related to our business and finances in general, the success of our commercial products, risks and uncertainties associated with our preclinical and clinical stage pipeline, risks and uncertainties associated with the regulatory review and approval process, risks and uncertainties associated with enrollment of clinical trials for rare diseases, and risks that ongoing or planned clinical trials may not succeed or may be delayed for safety, regulatory or other reasons. Specifically, we face risks associated with the commercial launch of FILSPARI in FSGS and the continued commercialization of FILSPARI in IgAN, the timing and potential outcome of our and our partners’ clinical studies, market acceptance of our commercial products including efficacy, safety, price, reimbursement, and benefit over competing therapies, risks related to the challenges of manufacturing scale-up, risks associated with the successful development and execution of commercial strategies for such products, including FILSPARI, and risks and uncertainties related to the current administration, including but not limited to risks and uncertainties related to tariffs and the funding, staffing and prioritization of resources at government agencies including the FDA. We also face the risk that we will not receive some or all of the potential future milestone and/or royalty payments described herein, the risk that our cash runway might not last as long as currently anticipated and the risk that we will be unable to raise additional funding that may be required to complete development of any or all of our product candidates, including as a result of macroeconomic conditions; risks relating to our dependence on contractors for clinical drug supply and commercial manufacturing; uncertainties relating to patent protection and exclusivity periods and intellectual property rights of third parties; risks associated with regulatory interactions; and risks and uncertainties relating to competitive products, including current and potential future generic competition with certain of our products, and technological changes that may limit demand for our products. We also face additional risks associated with global and macroeconomic conditions, including health epidemics and pandemics, including risks related to potential disruptions to clinical trials, commercialization activity, supply chain, and manufacturing operations, and the other risks and uncertainties that are described in the Risk Factors section of our most recent annual or quarterly report and in other reports we have filed with the SEC.

These statements are based on our current beliefs and expectations and speak only as of the date of this presentation. We do not undertake any obligation to publicly update any forward-looking statements.

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We are in rare for life.

At Travere Therapeutics, we are a biopharmaceutical company that comes together every day to help patients, families, and caregivers of all backgrounds as they navigate life with a rare disease. On this path, we know the need for treatment options is urgent — that is why our global team works with the rare disease community to identify, develop, and deliver life-changing therapies.

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Four Strategic Growth Pillars Support Durable Long-Term Value Creation

CORE GROWTH DRIVER

Continued growth of FILSPARI in IgAN

Established leadership with continued demand in evolving treatment landscape

FILSPARI (sparsentan) tablets 200 mg/400 mg

CORE GROWTH DRIVER

Successful launch of FILSPARI in FSGS

First approved medicine with rapid early adoption supported by high unmet need and prescriber familiarity

FILSPARI (sparsentan) tablets 200 mg/400 mg

POTENTIAL NEXT APPROVED MEDICINE

Pegtibatinase as potential first disease modifying therapy for classical HCU

Topline data from pivotal Phase 3 HARMONY Study expected in 2H27

Pegtibatinase

NEW GROWTH PLATFORM

Civorebrutinib complements the rare kidney disease portfolio

Pipeline-in-a-product potential with multi-billion-dollar global opportunity

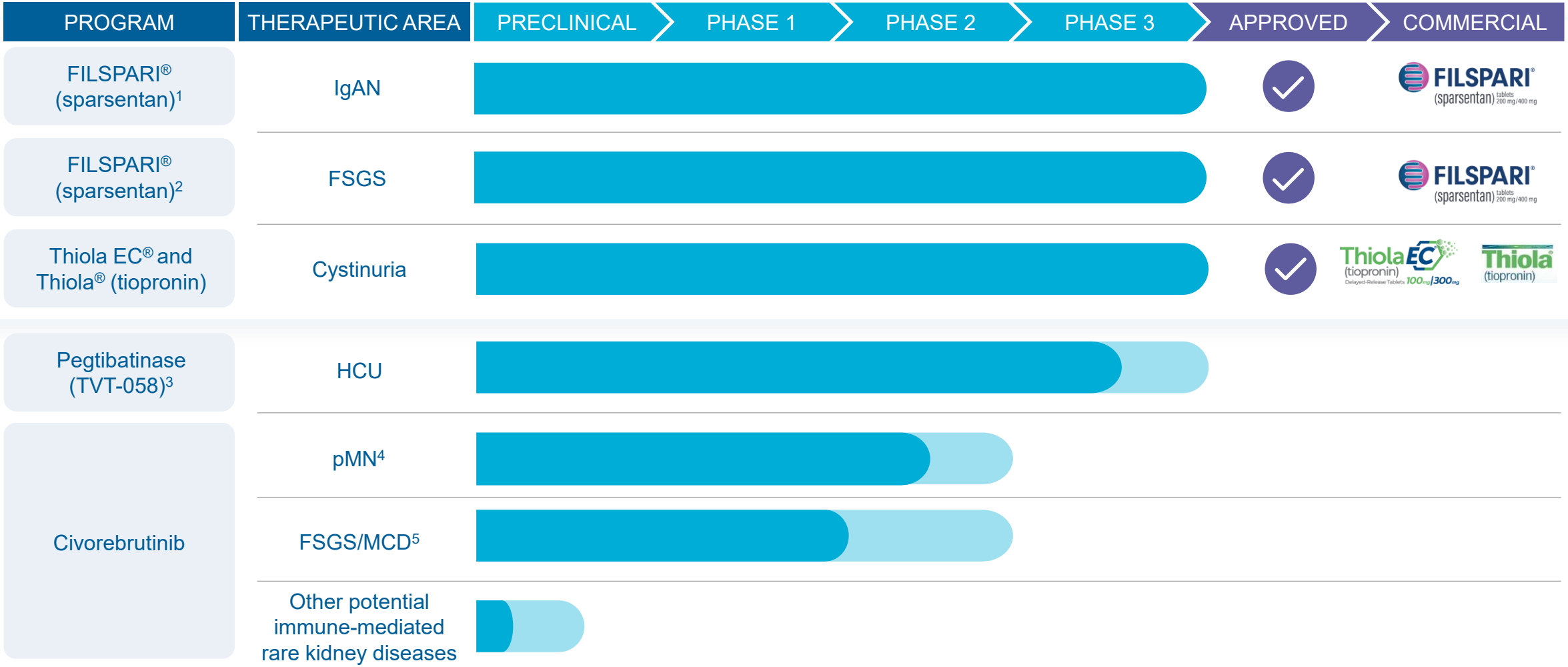
CIVOREBRUTINIB

Continued portfolio expansion through external innovation and disciplined investment

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Pipeline of Potential Best-in-Class Programs Targeting Rare Kidney and Metabolic Diseases



Abbreviations: IgAN: IgA nephropathy; FSGS: focal segmental glomerulosclerosis; HCU: classical homocystinuria, MCD: minimal change disease; pMN: primary membranous nephropathy.
[1] In September 2024, the FDA granted full approval of FILSPARI (sparsentan) to slow kidney function decline in adults with primary IgAN who are at risk for disease progression. [2] In April 2026, the FDA granted approval of FILSPARI (sparsentan) to reduce proteinuria in adult and pediatric patients aged 8 years and older with FSGS without nephrotic syndrome. CSL Vifor has exclusive commercial rights for sparsentan in Europe, Australia, New Zealand, Bahrain, Brazil, Chile, Israel, Kuwait, Oman, Qatar, Saudi Arabia and the United Arab Emirates. Chugai Pharmaceutical has exclusive commercial rights for sparsentan in Japan, South Korea, and Taiwan.
[3] Topline efficacy data from the Phase 3 HARMONY Study is anticipated in 2H 2027. [4] Current Everest Medicines study. [5] As part of the ongoing basket study conducted by Everest Medicines.

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FILSPARI: Clear Path to Durable Growth Through Leadership in Rare Kidney Disease

FILSPARI®

Multiple growth drivers support FILSPARI expansion

>$3B

Estimated sales opportunity in the U.S. by 2033

Large and Expanding Market Opportunity

>100,000 addressable patients across IgAN and FSGS in the U.S.[1]

Differentiated Clinical Profile

The only **foundational nephroprotective therapy** in IgAN and FSGS that can replace the historical standard of care

Designed for Long-Term Use

Once-daily oral medicine with growing long-term and real-world evidence

Scalable Commercial Platform

Leveraging cross-indication synergies to target **~7,000 nephrologists**

[1] Source: Independent market research, and data on file. FILSPARI is indicated to slow kidney function decline in adults with primary IgAN who are at risk for disease progression. FILSPARI is indicated to reduce proteinuria in adult and pediatric patients aged 8 years and older with FSGS without nephrotic syndrome.

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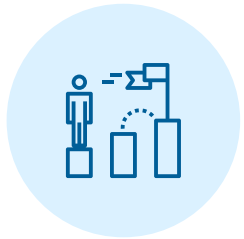
FILSPARI Commercial Execution Continues with New and Repeat Prescribers Driving Demand

$200M

U.S. net FILSPARI sales in 3Q26

120%

growth vs 3Q25



Evolving IgAN landscape expanding the market

High compliance and persistence rates

1,932

New PSFs in 3Q26

Strong demand adding to a growing base of patients with IgAN and FSGS



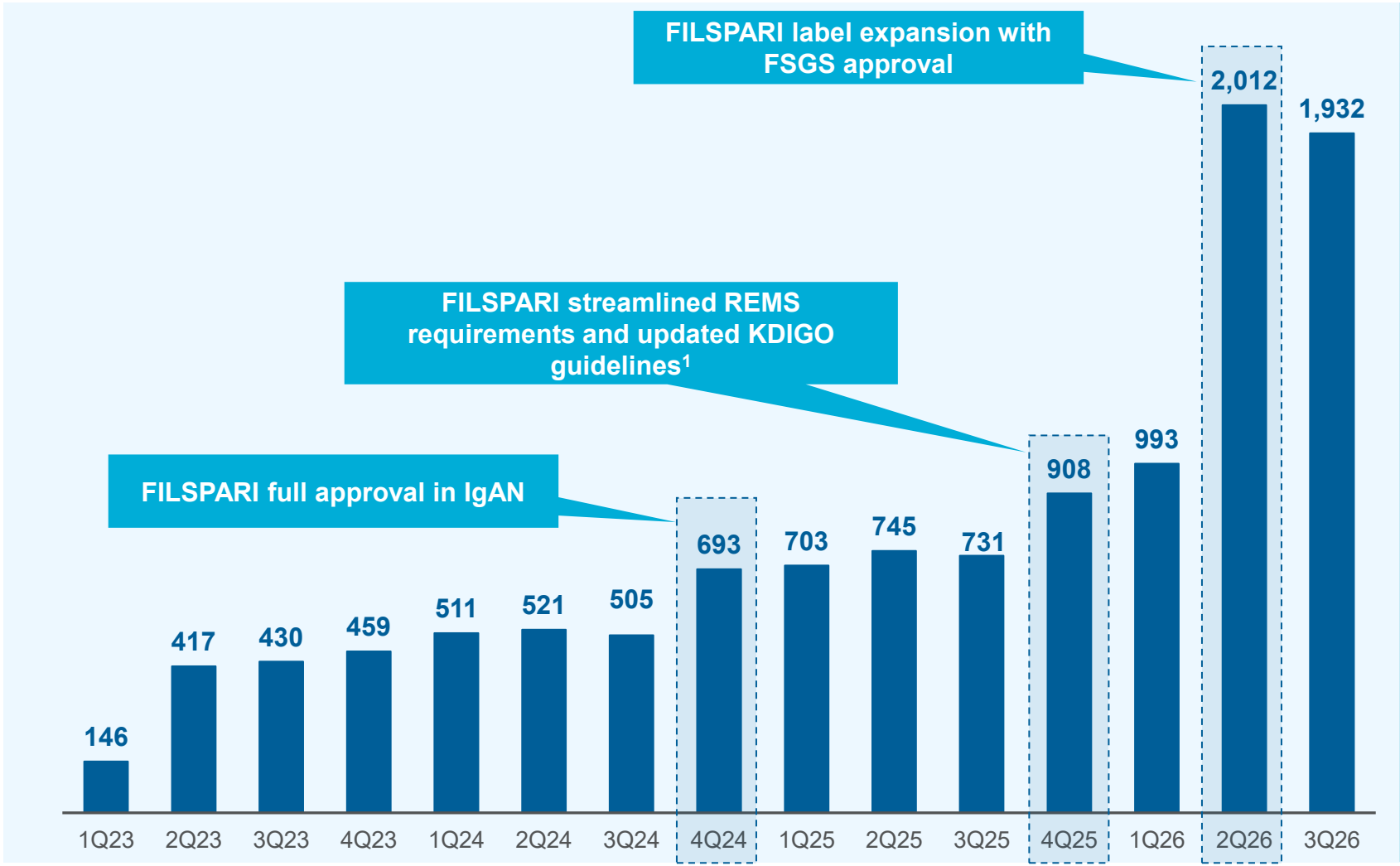
Increasing breadth and depth of prescriber base

High unmet need, urgency to treat, and familiarity with FILSPARI driving adoption in FSGS

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Strong Commercial Momentum Supported by Significant Long-Term Growth Drivers



- **~10% penetration** of the combined IgAN and FSGS addressable market provides significant runway for continued growth
- Strong execution across IgAN and FSGS is establishing FILSPARI as the **foundational nephroprotective therapy** in rare kidney disease
- Expanding clinical evidence and **evolving treatment paradigms** continue to strengthen FILSPARI's long-term growth trajectory

[1] KDIGO 2025 Clinical Practice Guideline for the Management of Immunoglobulin A Nephropathy (IgAN) and Immunoglobulin A Vasculitis (IgAV), October 2025.
* As measured by quarterly FILSPARI new patient start forms (PSFs).

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Caitlin, living with IgAN

01

FILSPARI in IgAN

Most commonly prescribed medicine approved for IgAN in evolving treatment landscape

02

FILSPARI in FSGS

Successfully launch first approved medicine in FSGS

03

Pegtibatinase

First disease-modifying therapy for classical HCU, if approved

04

Civorebrutinib

Pipeline-in-a-product potential with multi-billion-dollar global opportunity

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A Substantial Opportunity to Improve the Lives of Patients Living with IgAN

25-39 peak incidence age of IgAN[1]

~11 yrs median time to kidney failure in high-risk adult patients[2]

30-40% of transplants fail due to disease recurrence[3]

2025 KDIGO guidelines[4] to drive earlier treatment and combination therapy market

>70,000 addressable patients[5]

$1B+ potential for FILSPARI in IgAN[6]

[1] Nair R & Walker PD. Kidney Int 2006; 69:1455–1458. [2] Barratt J, et al., *Natural History of IgA Nephropathy: Analysis of a UK National RaDaR IgA Nephropathy Cohort*, ASN 2021; Poster presentation (Abstract P01577). [3] Uffing A et al., Clin J Am Soc Nephrol. 2021 Aug;16(8):1247-1255. [4] KDIGO 2025 Clinical Practice Guideline for the Management of Immunoglobulin A Nephropathy (IgAN) and Immunoglobulin A Vasculitis (IgAV), October 2025. [5] Independent market research and data on file. [6] Company estimates.

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Current KDIGO Guidelines: The IgAN Treatment Paradigm is Evolving

Earlier Treatment, Lower Proteinuria Targets and Simultaneous Therapy

1 Earlier diagnosis

Kidney biopsy in all adults with proteinuria ≥0.5g/d[1]

2 At risk of progressive loss of kidney function requiring treatment

At risk if proteinuria ≥0.5g/d while on or off treatment

FILSPARI included for earlier, first-line use to optimize nephroprotection

Simultaneous treatment in most patients

3 Treatment goal

Proteinuria should be maintained at <0.5g/d, and ideally at <0.3g/d

Proteinuria is the only validated early biomarker to help guide clinical decision-making

Source: KDIGO 2025 Clinical Practice Guideline for the Management of Immunoglobulin A Nephropathy (IgAN) and Immunoglobulin A Vasculitis (IgAV), October 2025.

[1] Or equivalent. In whom IgAN is a possible diagnosis and who do not have a contraindication for kidney biopsy.

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The Shifting IgAN Treatment Paradigm: Two Areas to Target; Two Treatment Categories

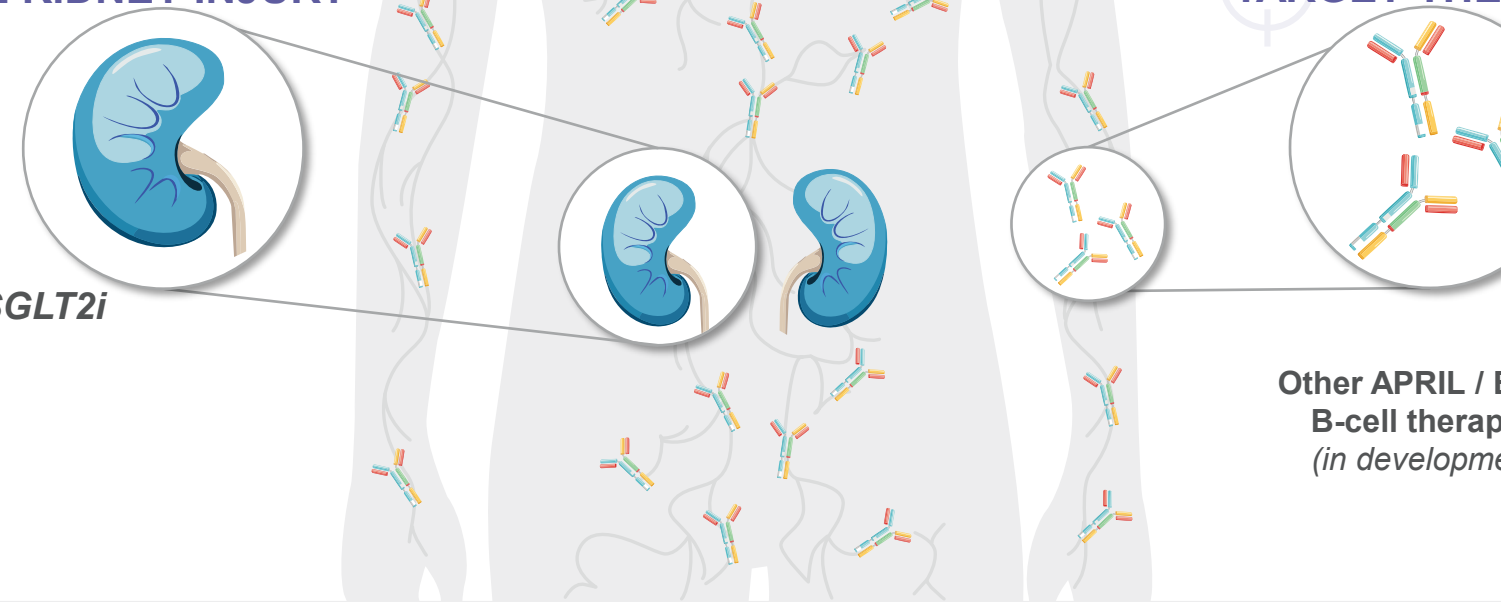
OVERACTIVATION IN THE KIDNEY | **OVERACTIVATION OF THE IMMUNE SYSTEM**

Foundational Treatment + **Immunosuppressant Treatment**

TARGET THE KIDNEY INJURY

- RASi
- FILSPARI®
- ETA + Supportive Care *(accelerated approval)*

± *SGLT2i*



TARGET THE IMMUNE SYSTEM

- Steroids
- Budesonide
- Complement Inhibitor
- APRIL / APRIL+BAFF *(accelerated approval)*

Other APRIL / BAFF, B-cell therapies *(in development)*

FILSPARI is the only oral non-immunosuppressive, long-term treatment positioned to replace historical standard of care for patients with IgAN*

Abbreviations: RASi: renin–angiotensin system inhibitor.
Source: KDIGO 2025 Clinical Practice Guideline for the Management of Immunoglobulin A Nephropathy (IgAN) and Immunoglobulin A Vasculitis (IgAV), October 2025.
* Indicated to slow kidney function decline in adults with primary IgAN who are at risk for disease progression.

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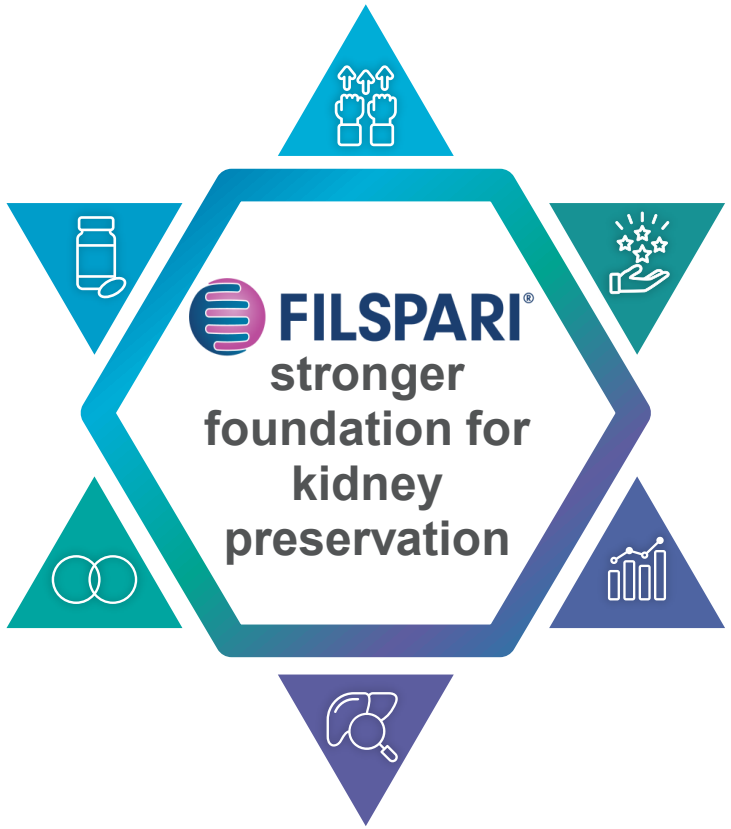
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FILSPARI Positioned as a First-in-Class Foundational Treatment in IgAN with Best-in-Class Features

Two-year safety data comparable to maximum-labeled dose irbesartan

One pill, once daily administration

Superior two-year proteinuria reduction vs active comparator irbesartan



Flexibility for combination use and newly diagnosed patients with IgAN

Only therapy to date to demonstrate statistically significant slowing of kidney function decline in a Phase 3 study vs active comparator irbesartan

Streamlined liver monitoring

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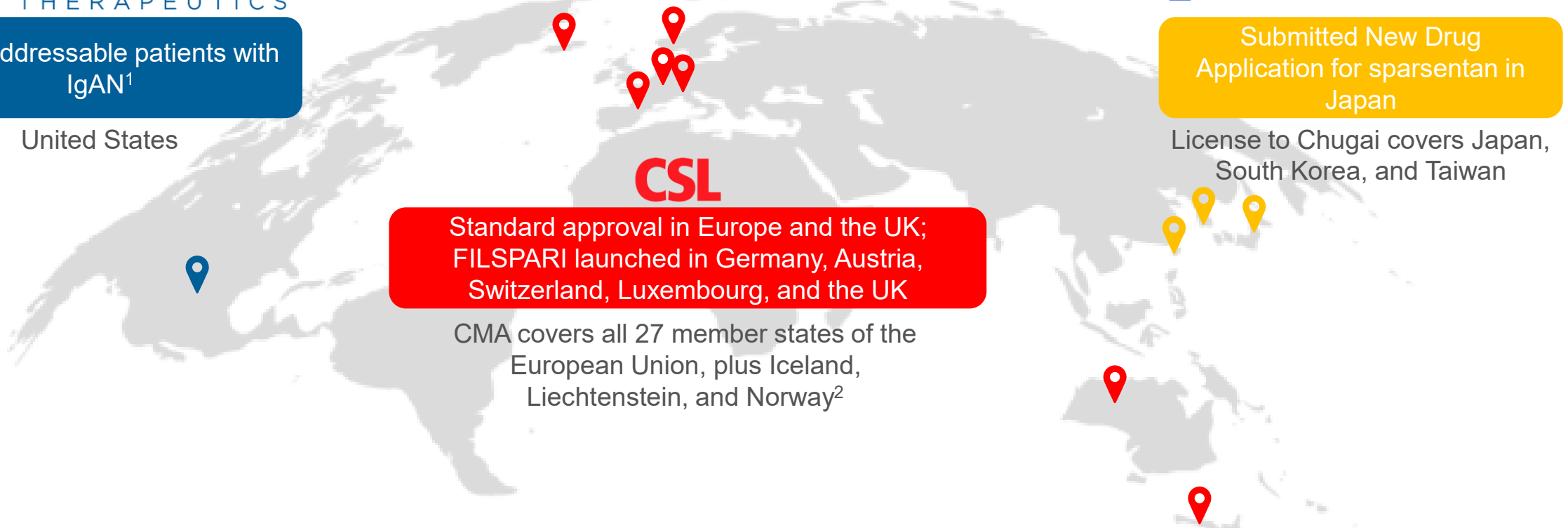
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Paving a Path to Global Access for FILSPARI in IgAN with Established Commercial Partners

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>70k addressable patients with IgAN[1]

United States



CSL

Standard approval in Europe and the UK; FILSPARI launched in Germany, Austria, Switzerland, Luxembourg, and the UK

CMA covers all 27 member states of the European Union, plus Iceland, Liechtenstein, and Norway[2]

CHUGAI PHARMACEUTICAL

A member of the Roche group

Submitted New Drug Application for sparsentan in Japan

License to Chugai covers Japan, South Korea, and Taiwan

Travere eligible to receive up to $910 million in potential milestone payments[3] + tiered double-digit royalties on global net sales of FILSPARI

Abbreviations: EC: European Commission; CMA: conditional marketing authorization. [1] Source: independent market research, data on file. [2] License to CSL covers Europe, Australia, New Zealand, Bahrain, Brazil, Chile, Israel, Kuwait, Oman, Qatar, Saudi Arabia and the UAE, with potential to expand. [3] Potential milestone payments include achievements for both IgAN and FSGS indications, as of the execution of the license agreements with CSL and Chugai Pharmaceutical. Through June 2026, the Company has received $63.5 million in disclosed milestone payments.
* Marks indicate licensed territories.

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01

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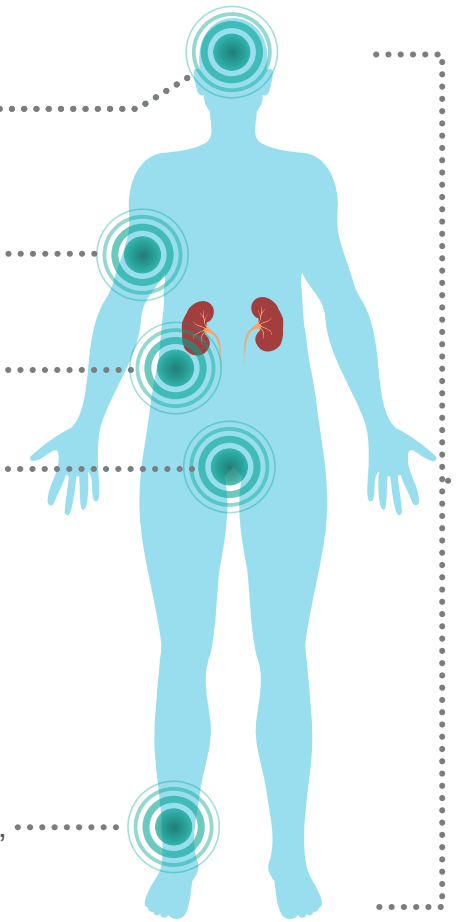
FSGS Represents Significant Patient and Community Burden

7-year
kidney survival rate
for FSGS is the lowest among primary glomerular diseases[1]

>4,000
people on the kidney transplant waitlist
due to FSGS in the U.S.[2]

~33,000
adults and children in the U.S. are currently experiencing kidney failure due to FSGS[3]

Symptoms of FSGS



Side effects from medications (IST)

Significant toxicity

- Infections
- Hypertension
- Diabetes
- Bone loss
- Mental health problems

5-10 years
median time to kidney failure for 30-60% of patients[4]

up to 55%
of transplant patients experience disease recurrence[5]

Abbreviations: FSGS: focal segmental glomerulosclerosis.
Sources: [1] Moranne O., Watier L., Rossert J., Stengel B., *GN-Progress Study Group Primary glomerulonephritis: an update on renal survival and determinants of progression,* Qjm. 2008;101(3):215–224. [2] Organ Procurement & Transplant Network (OPTN) data accessed December 2025 from HRSA website. [3] Bensink ME, Goldschmidt D, et al., *Kidney failure attributed to focal segmental glomerulosclerosis: a USRDS retrospective cohort study of epidemiology, treatment modalities, and economic burden,* Kidney Med. 2023;6(2):100760. doi: 10.1016/j.xkme.2023.100760. [4] Kiffel et al. Adv Chronic Kidney Dis. 2011;18:332-338. [5] Kienzl-Wagner et al. 2019; Trachtman et al. 2015; USRDS 2020.
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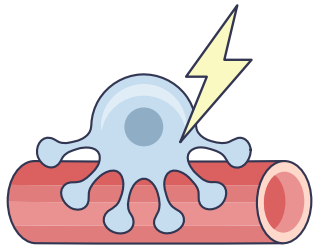
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Proteinuria is the Primary Predictor of Disease Severity and Kidney Failure in FSGS

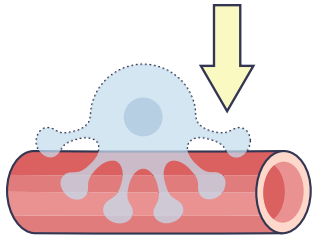
Role of Elevated and Persistent Proteinuria in FSGS

PROTEINURIA

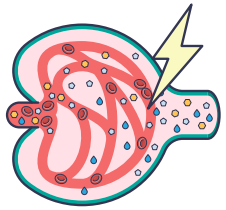
Podocyte injury[1,2]



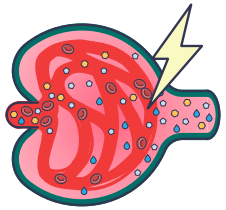
Podocyte Loss[3,4,5]



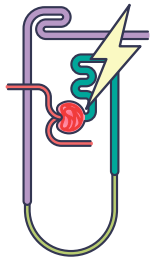
Glomerular hypertrophy hyperfiltration[6,7]



Glomerular sclerosis[1,2]



Tubulointerstitial fibrosis[1,2]



KIDNEY FAILURE

Abbreviations: FSGS: focal segmental glomerulosclerosis.

Sources: [1] De Zoysa, 2024. [2] Puelles, 2019. [3] Fogo, 1991. [4] Butt, 2020. [5] Kris, 2005. [6] Wiggins 2007. [7] Matsusaka, 2011.

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Regardless of the Underlying Etiology, All Types of FSGS Share a Similar Glomerular Lesion Initiated by Podocyte Injury



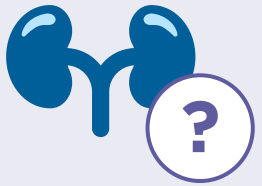
PRIMARY

FSGS lesions with diffuse foot process effacement due to putative circulating factors, possibly a cytokine originating from extrarenal sources[1,2]



GENETIC

Identified genetic variants of podocyte and basement membrane proteins as well as high risk alleles for FSGS[1,2]



UNDETERMINED CAUSE

FSGS lesions without diffuse foot effacement accompanied by proteinuria, in the absence of a genetic or identifiable secondary cause[1]



SECONDARY

Secondary to other disease processes. Podocyte damage caused by a known pathophysiologic process[1]

PODOCYTE INJURY

mediated by upregulation of ET-1 and Ang II[3]

Abbreviations: Ang II: angiotensin II; ET-1: endothelin-1; FSGS: focal segmental glomerulosclerosis.

[1] Kidney Disease: Improving Global Outcomes (KDIGO) Glomerular Diseases Work Group. Kidney Int. 2021;100(4S):S1-S276.
[2] De Vriese AS, et al. J Am Soc Nephrol. 2018;29(3):759-774.
[3] Kohan DE, et al. Clin Sci. 2024;138(11):645-662.

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KDIGO Guidelines Recommend FSGS Management Based on Nephrotic Syndrome Status

FILSPARI is indicated to reduce proteinuria in adult and pediatric patients aged 8 years and older with focal segmental glomerulosclerosis (FSGS) without nephrotic syndrome



Patient with FSGS lesion on kidney biopsy[1]

ABSENCE OF nephrotic syndrome

Nephrotic-range proteinuria >3.5 g/d
BUT
Serum albumin >3.0 g/dl
OR
Proteinuria <3.5 g/d
with or without low albumin

- Evaluate for underlying cause, exclude secondary forms of FSGS
- Consider genetic screening (where appropriate) to exclude genetic forms
- Do not start immunosuppression
- Supportive therapy
- Monitor proteinuria and serum albumin

Worsening proteinuria and reduction in serum albumin

PRESENCE OF nephrotic syndrome

Proteinuria >3.5 g/d
AND
Serum albumin <3.0 g/dl
with or without edema
Especially in the presence of diffuse foot process effacement

- Likely primary FSGS
- Treat with immunosuppression
- If no response, consider genetic testing

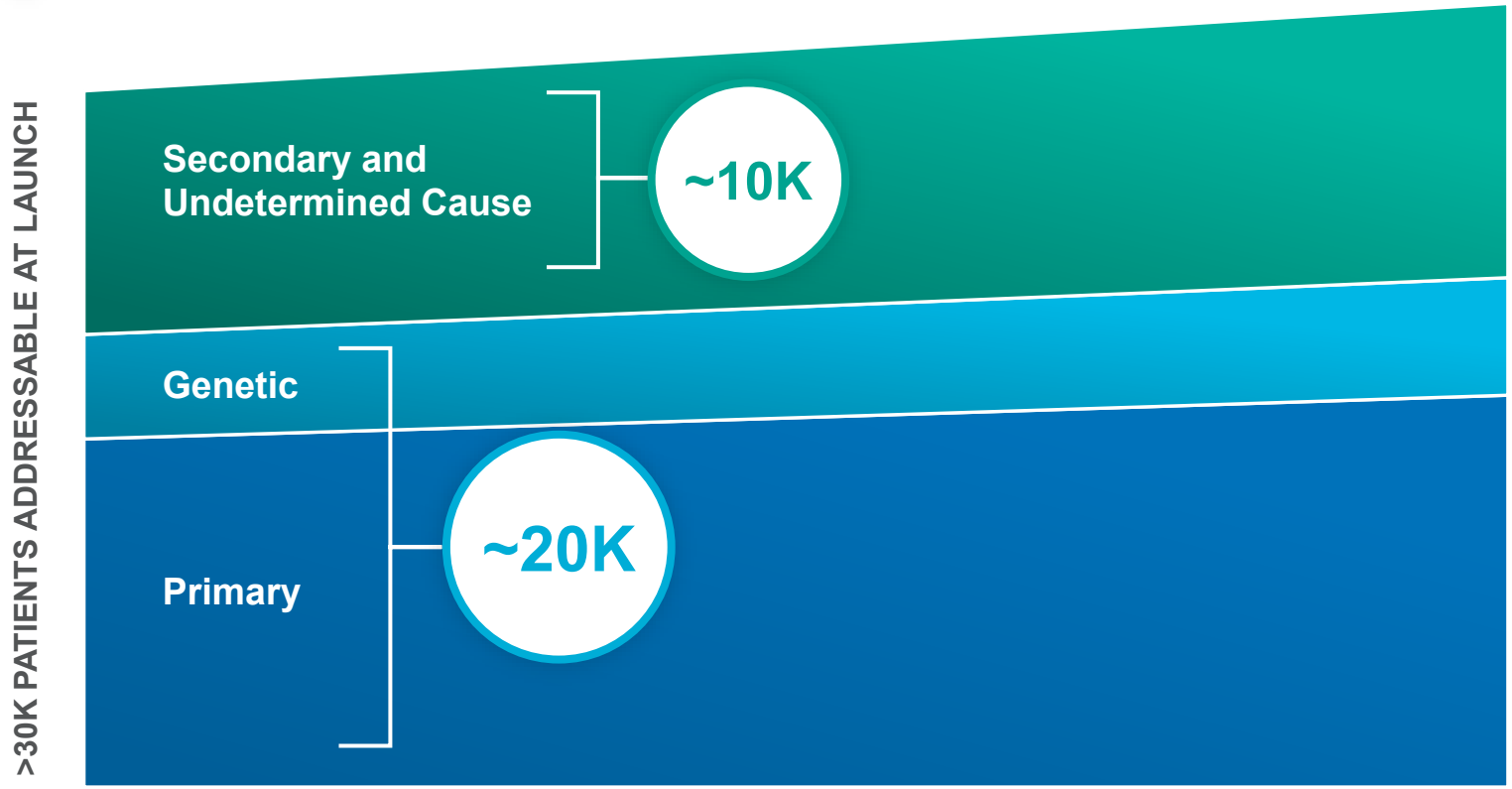
[1] Source: KDIGO 2021 Clinical Practice Guideline for the Management of Glomerular Diseases | Volume 100 | Issue 4S | October 2021.

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Addressable FSGS Population Across Disease Subtypes is Expected to Grow Over Time

>30,000 addressable patients with clear drivers of future growth



Future growth drivers:

- Population growth
- Earlier referral to nephrologist in light of approved treatment options
- Increased biopsies
- Lower treatment targets
- Expanding access

Source: Independent market research and data on file.

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Strong Anticipation and Clinical Familiarity with FILSPARI Expected to Drive Utilization in FSGS

>80% of nephrologists indicate novel non-immunosuppressives are highly desirable in FSGS[1]

~78% of nephrologists report being “highly familiar” with FILSPARI for FSGS[1]

~90% of patients are already on ACE/ARBs, positioning FILSPARI as a clear replacement option from supportive care to kidney-targeted therapy[2]

“In FSGS, there's no definitive therapy. We use steroids and then we move on to other things... But the effect is not great, we'd like something more definitive, more focused.”[1]

–Nephrologist

[1] Source: Spherix FSGS Market Dynamix Report, October 2025.
[2] Source:Spherix 2025 Patient Chart Dynamix.

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Leveraging FILSPARI's Established Commercial Platform to Accelerate FSGS Adoption



Patient & Physician Focus

Continuous patient and physician education on approved indication

Established Commercial Infrastructure

Experienced field team leveraging high prescriber overlap to accelerate uptake across both indications

Access & Reimbursement

Established payer access in IgAN positions FILSPARI for supportive access environment in FSGS

Focus on expanding policies to include FSGS

Comprehensive Support Program

Travere TotalCare infrastructure applied and customized to patients with FSGS

Product Available Day 1

Streamlined logistics with product already in channel

Strong commercial foundation that supports uptake across both indications

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Strategic Evidence Generation Expected to Further Support FILSPARI's Long-Term Growth Opportunity

	Evidence Generation	Data Generation	Potential Impact
IgAN	FILSPARI as **first-line therapy** in newly diagnosed patients *(SPARTAN Study)*	**~77% proteinuria reduction** & eGFR stabilization *(week 110)*[1] **~75% complete remission**[1]	**Supports foundational therapy utilization** in treatment-naïve patients
IgAN & FSGS	Evaluate FILSPARI in **post-transplant recurrent IgAN & FSGS** *(SPARX Study)*	Data presentations **expected in 2027**	To support use in **~7,000 addressable post-transplant patients**
FSGS	Generate evidence in **patients of African ancestry with FSGS** *(SPARLIGHT Study)*	Study initiation **in 2H 2026**	**To support broader FSGS adoption,** including high-risk patients

Abbreviations: eGFR: estimated glomerular filtration rate.
[1] Source: Cheung CK, et al. presented at ASN 2026; October 21-25, 2026; Denver, CO. TH-PO0453.

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Jamela, living with HCU

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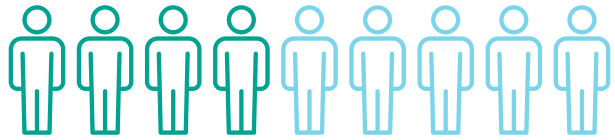
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HCU Market Represents Significant Unmet Need with Potential for Growth with Better Diagnostics, Awareness and Effective Treatment Options

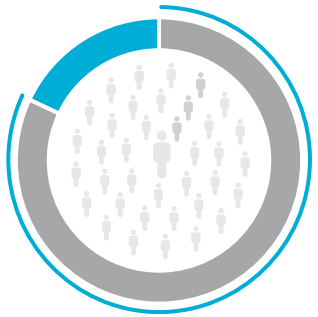
Disease education/awareness, enhanced diagnostics and better treatment options are expected to lead to **increased patient identification**, **earlier diagnosis, and better outcomes** – driving growth in addressable market

- **25% of HCU patients** by **age 16** and **50% by age 29** develop life-threatening thrombotic events, including heart attack and stroke[1,2]
- **7,000 to 10,000 patients** living with HCU in U.S.; similar number in Europe[3]
- **No approved treatments** to address the underlying genetic cause of HCU

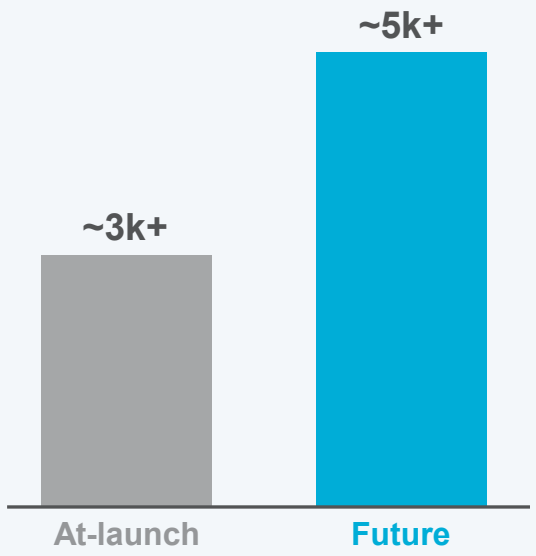


Despite newborn screening for HCU in the U.S., **fewer than 50% of people with HCU are diagnosed at birth**[4]

~80% of patients with HCU are partially or non-responsive to **B6 therapy** (current standard of care)[5]



Expected growth in addressable patients with HCU in U.S.



Pegtibatinase has the potential to become the **only disease-modifying therapy** in a market with significant growth expected

Sources: [1] Mudd et al., *Am J Hum Genet* (1985), [2] Yap et al., *J Am Heart Assoc*. (2001), [3] Data on file. [4] Levy H, et al., *Clin Chem*. 2023;69(5):433-434, [5] Kozich V, Sokolova J, Morris AAM, et al., *J Inherit Metab Dis*. 2021;44(3):677-692.

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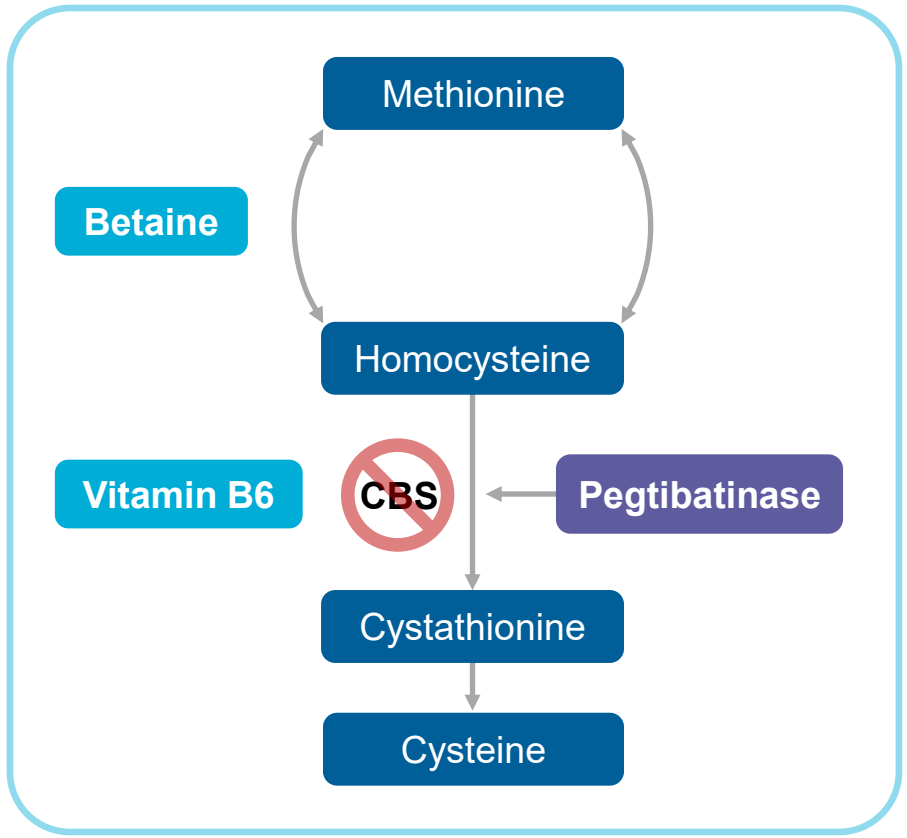
Pegtibatinase is an Investigational, Modified, Recombinant CBS Human Enzyme Therapy

Pegtibatinase is designed to address the underlying genetic cause of HCU

Mechanism of action is designed to have broad effect across HCU population

Administered subcutaneously and designed to be active and stable in plasma, unlike native CBS

Designed to introduce the CBS enzyme into circulation and reduce intracellular and plasma Hcy levels



Pegtibatinase has been granted multiple regulatory designations for the treatment of classical HCU

- FDA Breakthrough Therapy designation
- FDA Rare Pediatric Disease designation
- FDA Fast Track designation
- Orphan Drug designation in the U.S. and Europe

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Treatment with Pegtibatinase in the Phase 1/2 COMPOSE Study Showed Rapid and Sustained tHcy Reduction Through 12 Weeks of Treatment

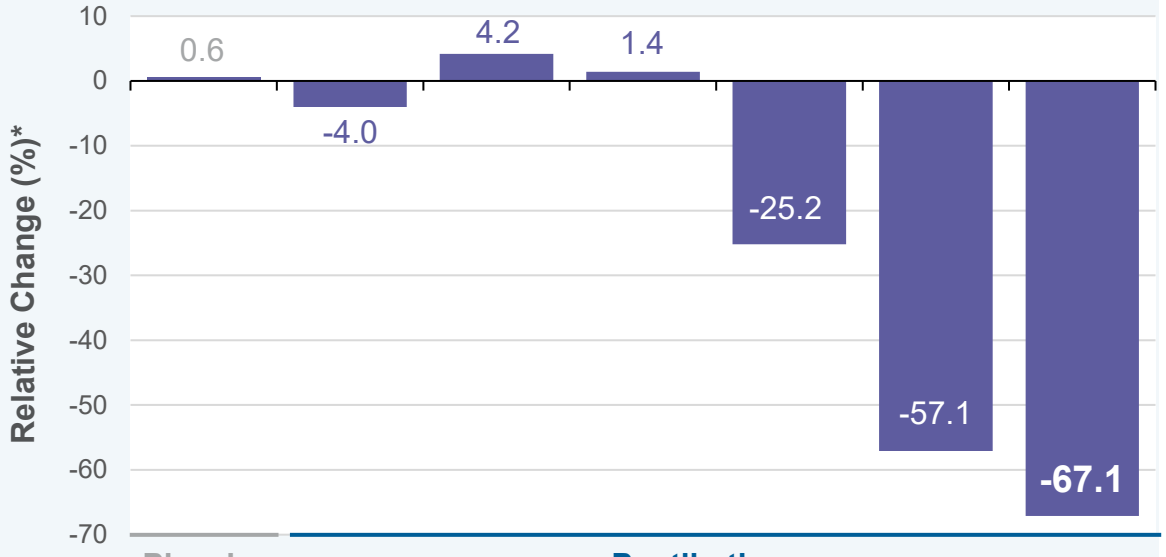
67.1% mean relative reduction in total homocysteine from baseline

All patients in highest dose cohort achieved a clinically meaningful threshold in mean tHcy over weeks 6 to 12 of treatment

Methionine and cystathionine biomarkers suggest that pegtibatinase acts similar to the native CBS enzyme and can restore the metabolic dysregulation in patients with HCU

Pegtibatinase was generally well-tolerated at all doses tested

Summary of relative reduction in geometric mean of total homocysteine from baseline from cohorts 1-6 in the Phase 1/2 COMPOSE Study



	Placebo	Pegtibatinase					
		Cohort 1 0.33 mg/kg QW	Cohort 2 0.66 mg/kg QW	Cohort 3 1.0 mg/kg QW	Cohort 4 1.0 mg/kg BIW	Cohort 5 1.5 mg/kg BIW	Cohort 6 2.5 mg/kg BIW
Sample Size (n)	6	3	3	3	2	3	4

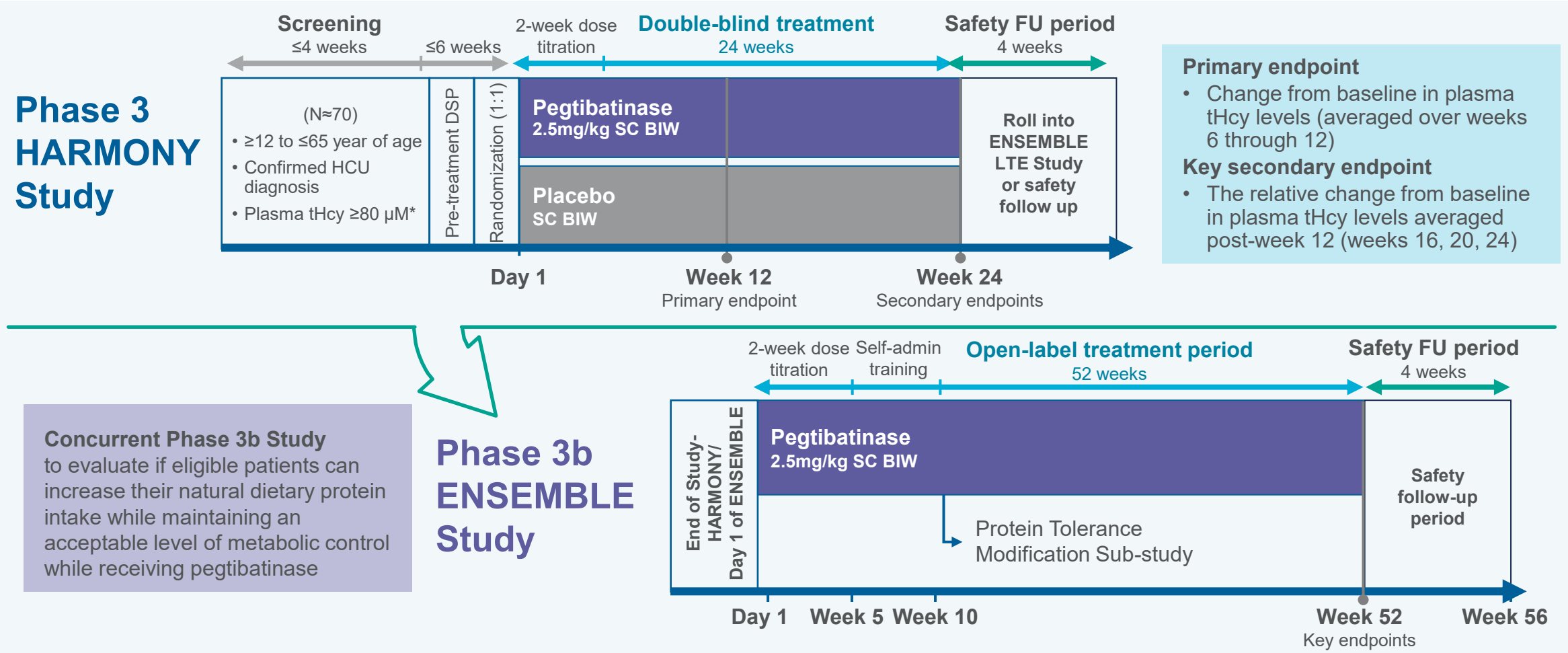
Abbreviations: BIW: twice weekly; QW: once weekly.

* The data referenced in the table above and the analysis conducted in cohort 6 assess the relative reduction in tHcy from baseline in the geometric mean by averaging tHcy over weeks 6, 8, 10, and 12. This measure improves the precision and reliability of assessment of the treatment effect and takes into account that there is some variability in tHcy depending on food intake and diurnal variation. The Company intends to use this measure moving forward.

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Innovative Pegtibatinase Phase 3 Program Designed to Enable a BLA Submission



Abbreviations: BIW: twice weekly, DSP: diet standardization period, LTE: long-term (open-label) extension, SC: subcutaneous, tHcy: total homocysteine, FU: follow up.
* Protocol allows for ~25% of patients with tHcy ≥50 to <80µM.
** ClinicalTrials.gov ID: NCT06247085.
*** In April 2026, the Company dosed the first new patient following the Phase 3 HARMONY Study restart. Topline efficacy data anticipated in 2H 2027.

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On Track to Position Pegtibatinase as the First Potential Disease-Modifying Therapy in HCU

Phase 3 HARMONY Study enrollment continues

Completed site activation

Leveraging patient identification efforts to build enrollment momentum

Topline data on track for 2H 2027

Patients continue to be followed in ENSEMBLE open label extension study

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01

FILSPARI in IgAN

Most commonly prescribed medicine approved for IgAN in evolving treatment landscape

02

FILSPARI in FSGS

Successfully launch first approved medicine in FSGS

03

Pegtibatinase

First disease-modifying therapy for classical HCU, if approved

04

Civorebrutinib

Pipeline-in-a-product potential with multi-billion-dollar global opportunity

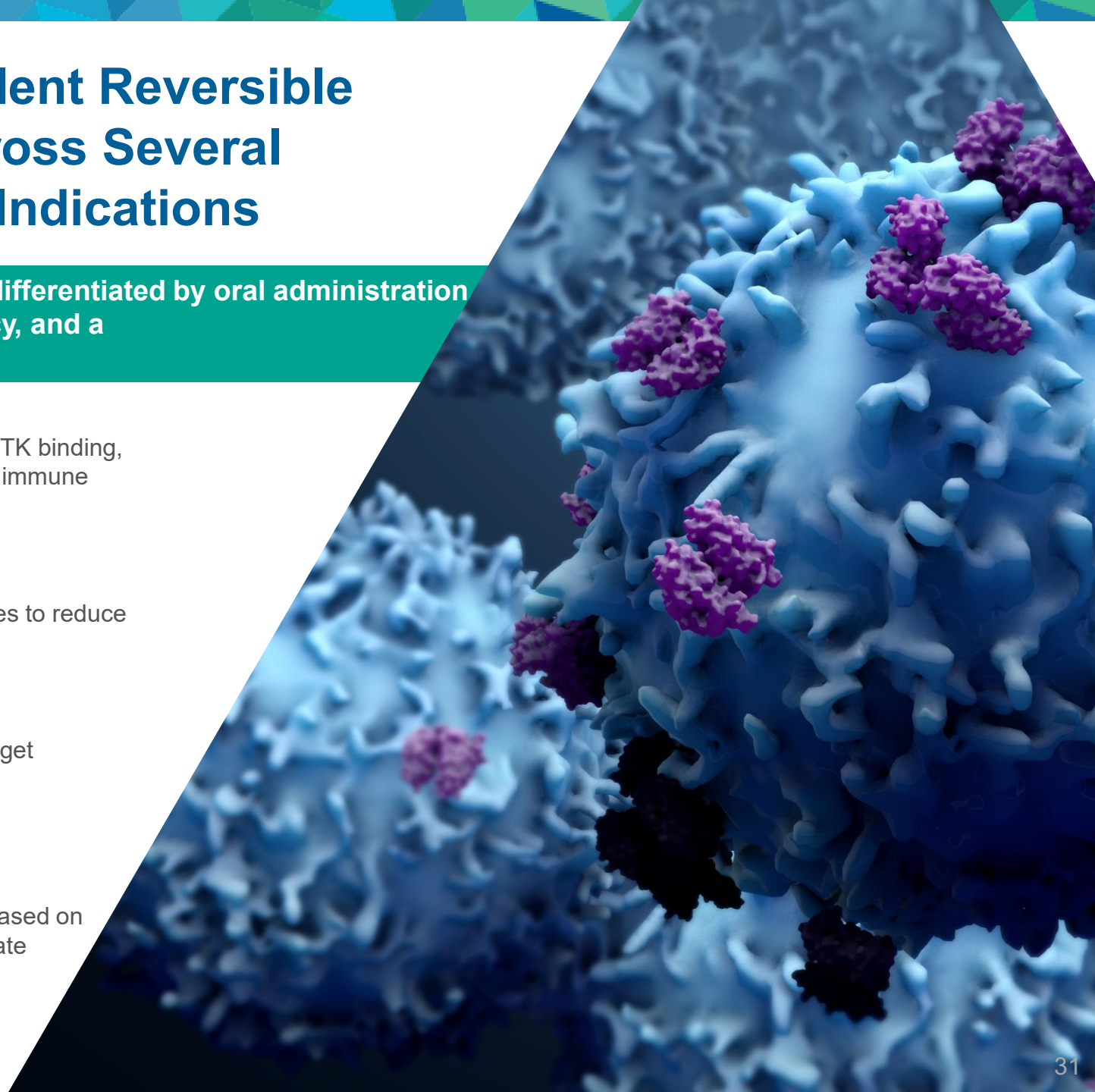
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Civorebrutinib is a Potent Covalent Reversible BTK Inhibitor with Potential Across Several Immune-Mediated Rare Kidney Indications

Civorebrutinib is a potential best-in-class BTK inhibitor differentiated by oral administration with rapid onset of action, high selectivity, strong potency, and a potentially favorable safety profile

1 Covalent reversible	Designed for strong yet reversible BTK binding, potentially enabling more controlled immune modulation vs other approaches
2 High selectivity	Highly selective against other kinases to reduce off-target toxicity
3 Potent target binding	Single digit nM potency and high target occupancy at clinical doses leads to sustained target inhibition
4 Potential best-in-class safety profile	Potentially favorable safety profile based on clinical and non-clinical studies to date



Abbreviations: BTK: Bruton tyrosine kinase.

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Civorebrutinib Offers Higher Selectivity and Potency Compared to Covalent Irreversible BTK inhibitors

1. Reversible covalency
2. Excellent selectivity
3. Potent target binding
4. Potential best-in-class safety profile

Compound	IC50 nM	Kinase IC50/BTK IC50 (fold)						
	BTK	LCK	SRC	LYN	EGFR	ITK	TEC	JAK3
Civorebrutinib	**1.8**	**3668x**	**4853x**	**1125x**	**714x**	**5178x**	**6x**	**>10000x**
Ibrutinib	1.5	4x	17x	17x	4x	3x	5x	21x

Civorebrutinib demonstrates potent and selective inhibition of BTK without affecting EGFR, ITK, or TEC family kinases—targets associated with potential adverse effects

BTKi	Selectivity (IC50 fold)		
	EGFR	ITK	TEC
Civorebrutinib	**714x**	**5178x**	**6.6x**
Ibrutinib	4x	3x	5x
Zanubrutinib	8.7x	187x	6.7x
PRN1008	400x	338x	0.6x
Acalabrutinib	>200x	>200x	18x

Source: Everest Medicines 2025 Interim Results Presentation, 8/29/2025.

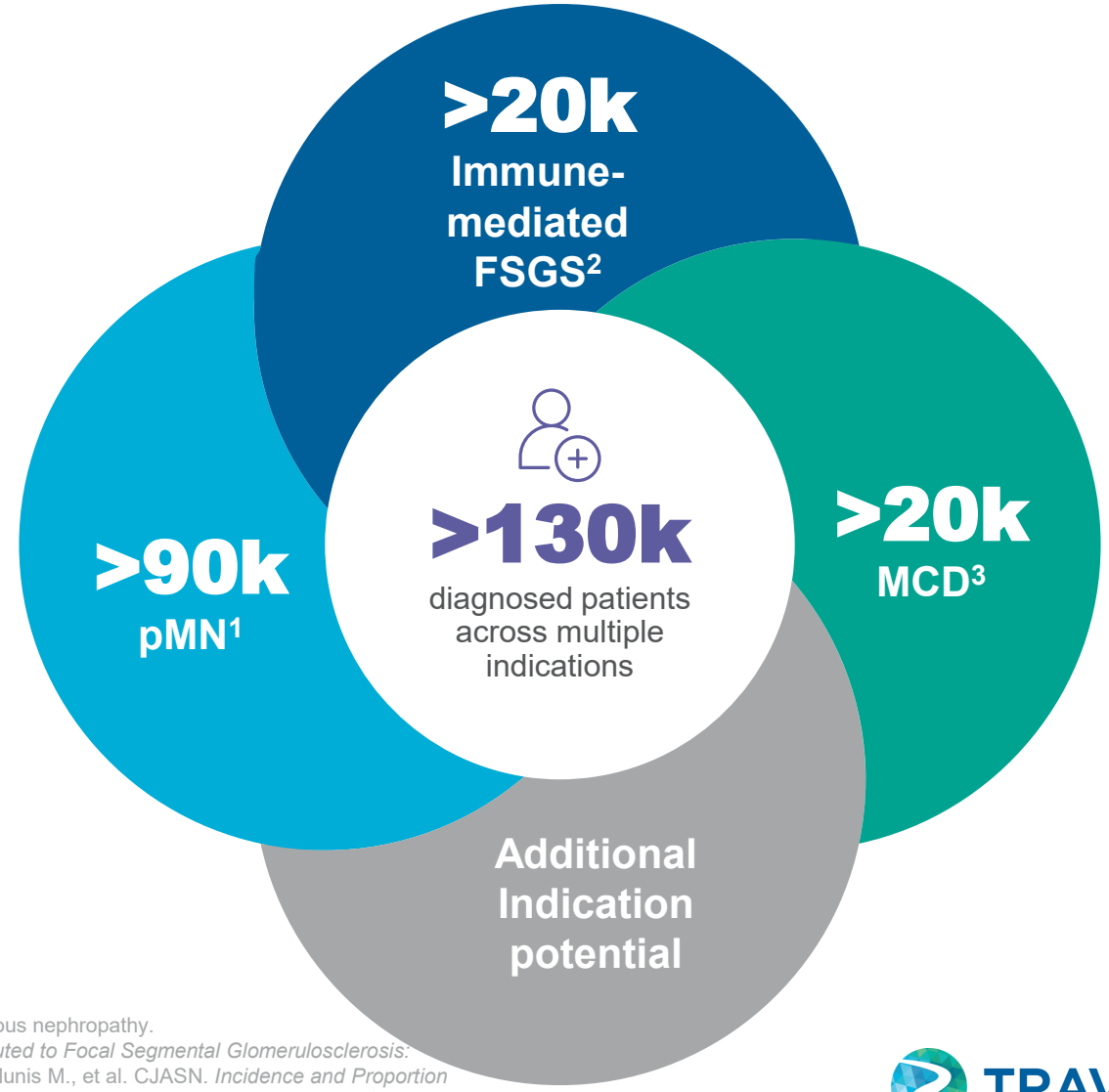
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TRAVERE® THERAPEUTICS

Significant Patient Population Across Potential Rare Kidney Disease Opportunities

Mechanistic rationale supports further evaluation in immune-mediated FSGS and MCD

Estimated Prevalence Across Immune-Mediated Rare Kidney Diseases



Abbreviations: FSGS: focal segmental glomerulosclerosis; MCD: minimal change disease; pMN: primary membranous nephropathy.
[1] Source: Swaminathan S., et al. CJASN, 2006. [2] Source: Bensink M., et al. Kidney Medicine. *Kidney Failure Attributed to Focal Segmental Glomerulosclerosis: A USRDS Retrospective Cohort Study of Epidemiology, Treatment Modalities, and Economic Burden*, 2023, and Munis M., et al. CJASN. *Incidence and Proportion of Primary Focal Segmental Glomerulosclerosis (FSGS) among a Racially and Ethnically Diverse Adult Patient Population between 2010 and 2021*, 2014. [3] Source: Vivarelli M., et al. CJASN. *Minimal Change Disease*, 2016, and Waldman M., et al. CJASN. *Adult minimal-change disease: clinical characteristics, treatment, and outcomes*, 2007.
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TRAVERE® THERAPEUTICS

Primary Membranous Nephropathy Remains a High Unmet Need with Significant Patient Burden

>70%
of patients present with nephrotic syndrome[1,2]

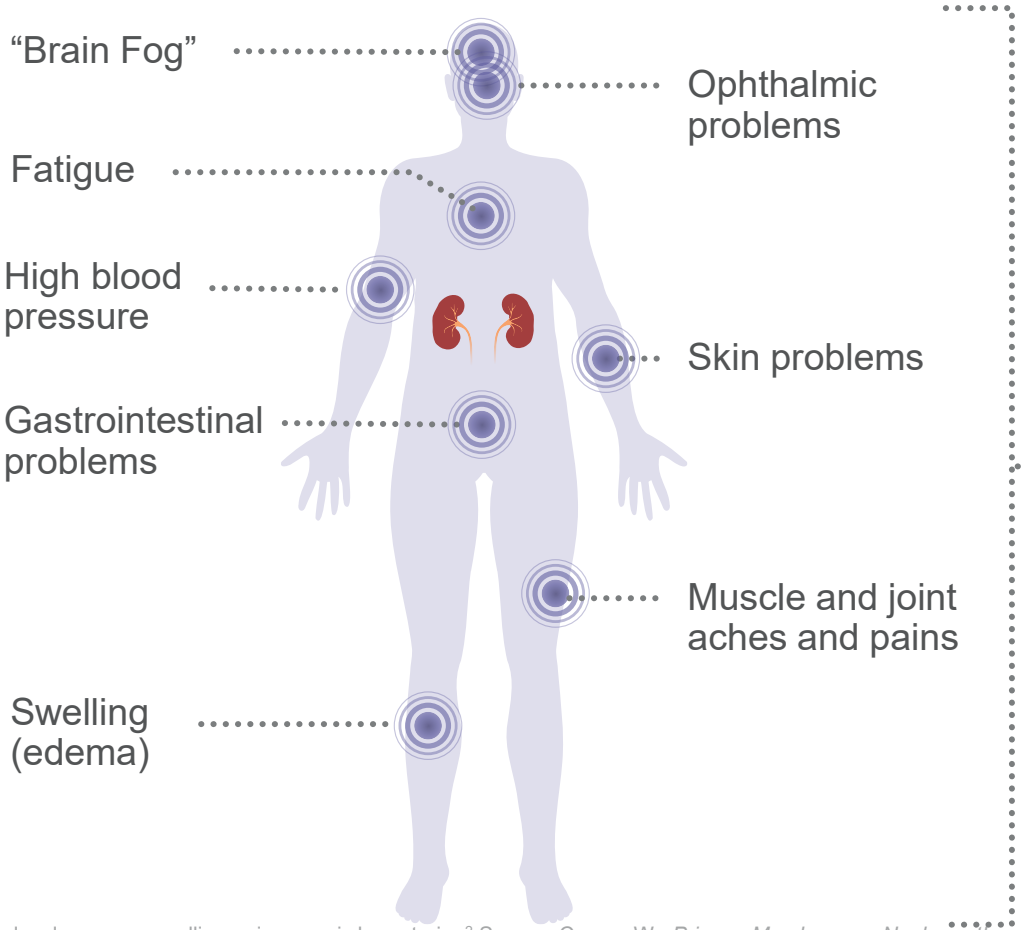
~60%
of patients presenting with sub nephrotic-range proteinuria will progress to full nephrotic syndrome within 1-2 years from presentation[3]

40-50%
of patients with persistent nephrotic syndrome will progress to kidney failure over a period of 10 years[4]

>70%
of patients with pMN report moderate or significant impacts on their daily life[5]

0
approved treatments

Symptoms of pMN



[1] Symptoms include high proteinuria (common range 3.5 g – 20 g/24h), decrease in serum albumin levels, severe swelling, microscopic hematuria. [2] Source: Couser W., *Primary Membranous Nephropathy*, CJASN, 2017. [3] Source: Hladunewich M., et al. *The Natural History of the Non-Nephrotic Membranous Nephropathy Patient*, CJASN, 2009. [4] Source: Ronco P., et al. *Membranous nephropathy*, Nature Reviews Disease Primers, 2021. [5] Source: National Kidney Foundation *"Voice of the Patient Report"*, Externally Led Patient-Focused Drug Development Meeting on Membranous Nephropathy. Public Meeting: August 27, 2021. Report Date: May 5, 2023.

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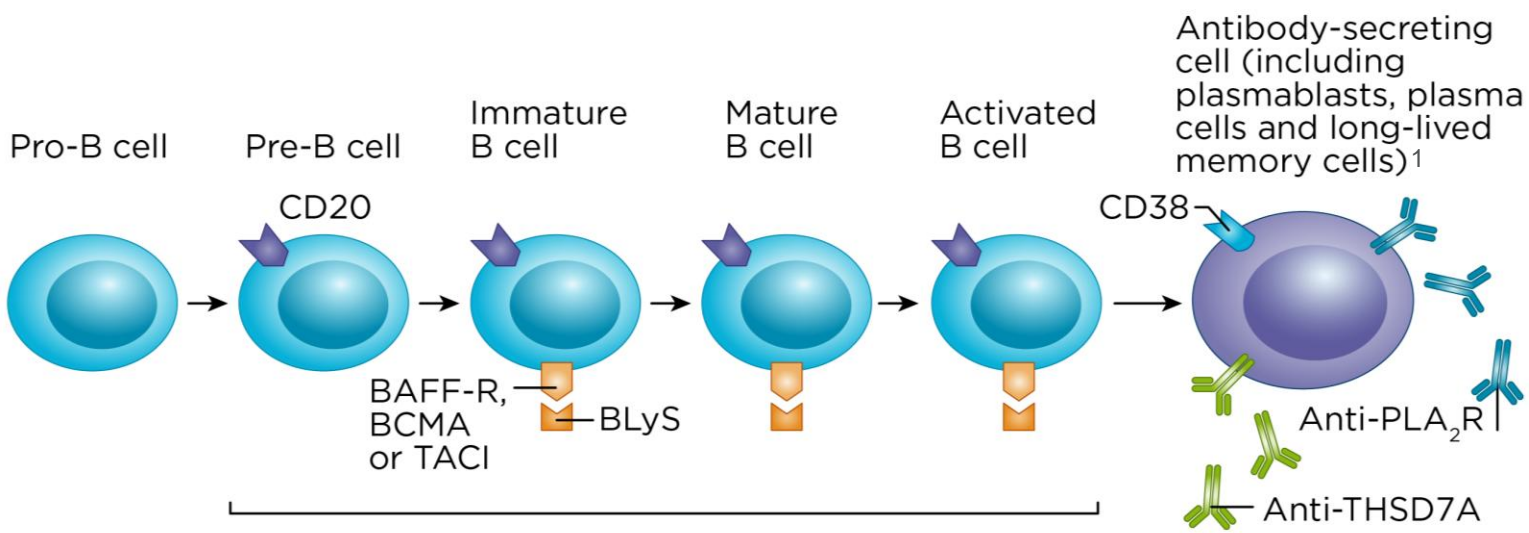
TRAVERE® THERAPEUTICS

BTK Inhibitors Target Multiple Key Nodes in the Pathogenesis of Primary Membranous Nephropathy

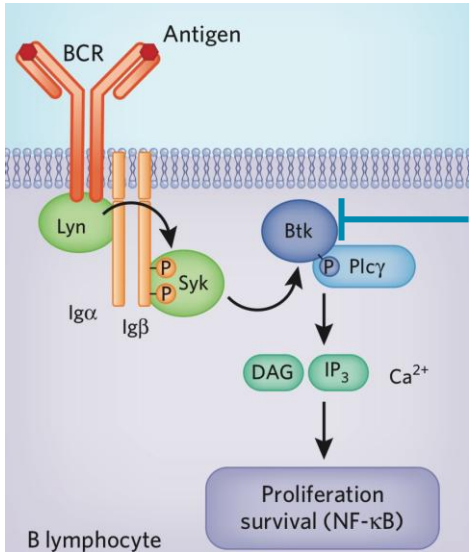
BTK inhibitors suppress B-cell signaling, reducing B-cell activation and proliferation and limiting differentiation into autoantibody-producing plasma cells

BTK inhibitors provide functional B-cell modulation without broad b-cell or immunoglobulin depletion

Oral BTK inhibitors may allow for more rapid recovery of B-cell function following treatment discontinuation, if needed



Intracellular site of action[2]



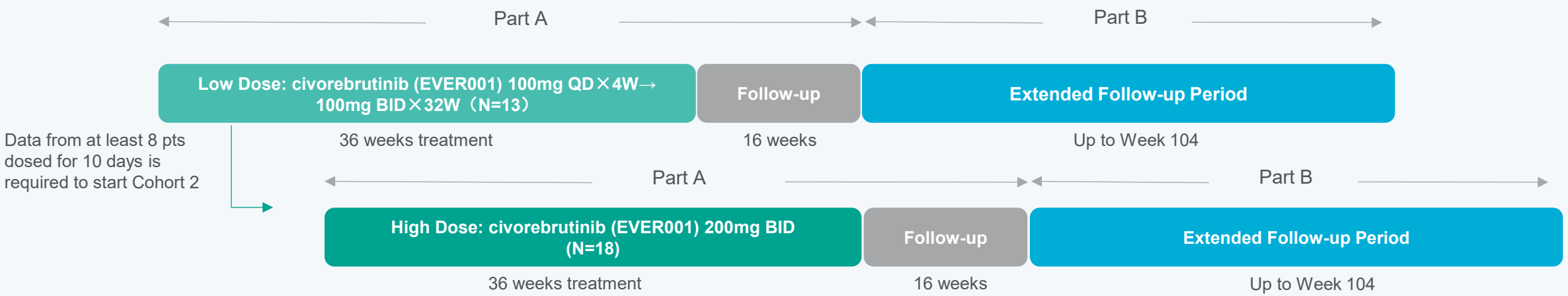
Abbreviations: anti-PLA2R: anti-phospholipase A2 receptor; anti-THSD7A: thrombospondin type-1 domain-containing protein 7A; BAFF: b-cell activating factor; BCMA: b-cell maturation antigen; BlyS: b-Lymphocyte stimulator; BTKi: Bruton tyrosine kinase inhibitor.

[1] Image adopted from Ruggenenti P., et al. *Nature Reviews Nephrology. Treatment of membranous nephropathy: time for a paradigm shift*, 2017. [2] Image adopted from Hendriks R. *New BTK inhibitor holds promise*, Nat Chem Biol 7, 4–5 , 2011.

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Phase 1b/2a Study of Civorebrutinib in Patients with pMN with Positive Anti-PLA2R Autoantibody



Key Inclusion and Exclusion Criteria

- Biopsy proven primary membranous nephropathy
- Anti-PLA2R autoantibody > 20 RU/mL
- Nephrotic range proteinuria >3.5 g/24h
- eGFR ≥ 45 mL/min/1.73m^2
- Serum albumin level ≥ 25g/L and IgG ≥ 3g/L
- No use of rituximab, cyclophosphamide and CNIs in 2 years, 6 months and 3 months before study treatment, respectively
- Exclude patients with positive TB test or positive HBsAg

Primary endpoint

- Safety and tolerability

Secondary endpoints

- Percentage change from baseline of 24h proteinuria, anti-PLA2R autoantibody level, UPCR and eGFR
- Complete or partial remission of 24h proteinuria
- Remission of anti-PLA2R autoantibody

Abbreviations: anti-PLA2R: anti-phospholipase A2 receptor; CNI: calcineurin inhibitor; eGFR: estimated glomerular filtration rate; HBsAg: hepatitis B surface antigen; TB: tuberculosis; UPCR: urine protein to creatinine ratio.

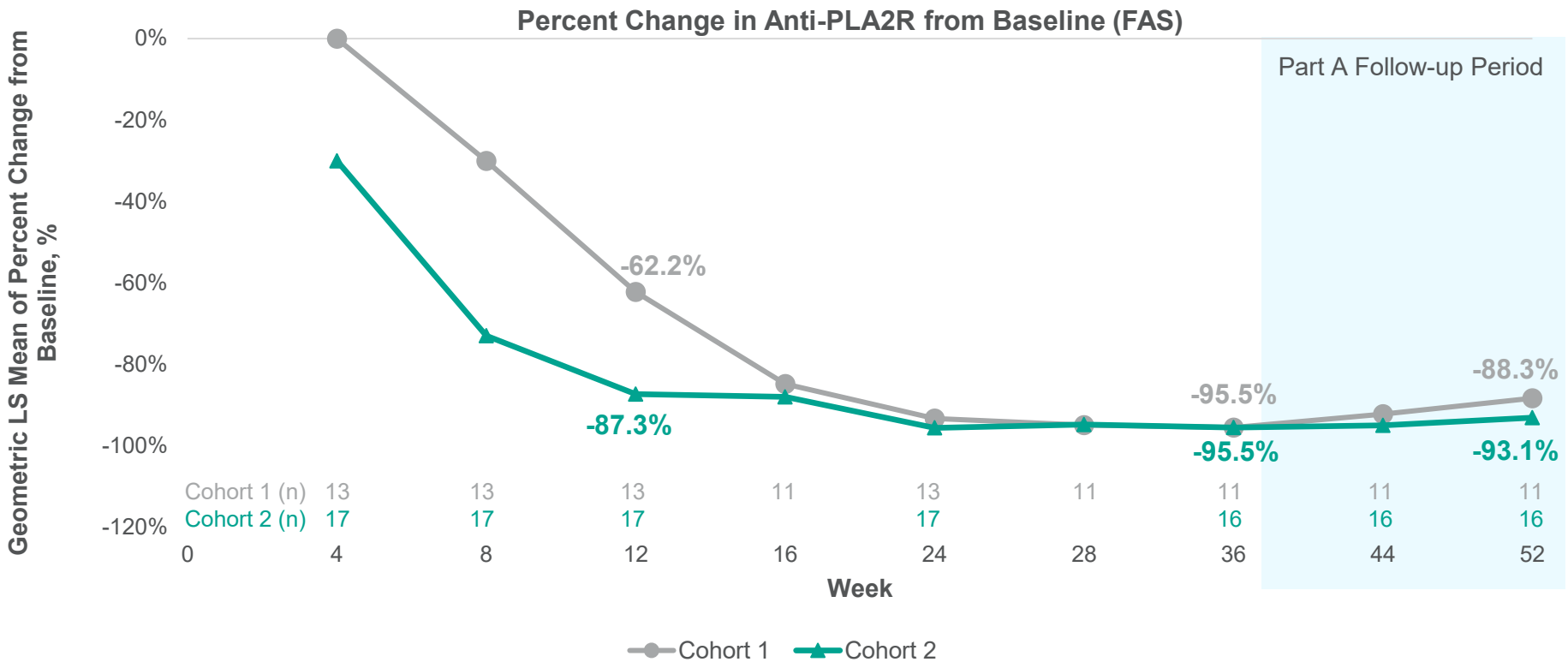
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Phase 1/2 Proof of Concept: Civorebrutinib Demonstrated Rapid and Sustained Reductions in Key Disease Biomarkers

~90%

reduction in anti-PLA2R by week 12 in a higher-dose cohort



Abbreviations: anti-PLA2R: anti-phospholipase A2 receptor.
Note: data cutoff date: September 5, 2025. Presented at 63rd ERA Congress in Glasgow, UK, on June 3-6, 2026.

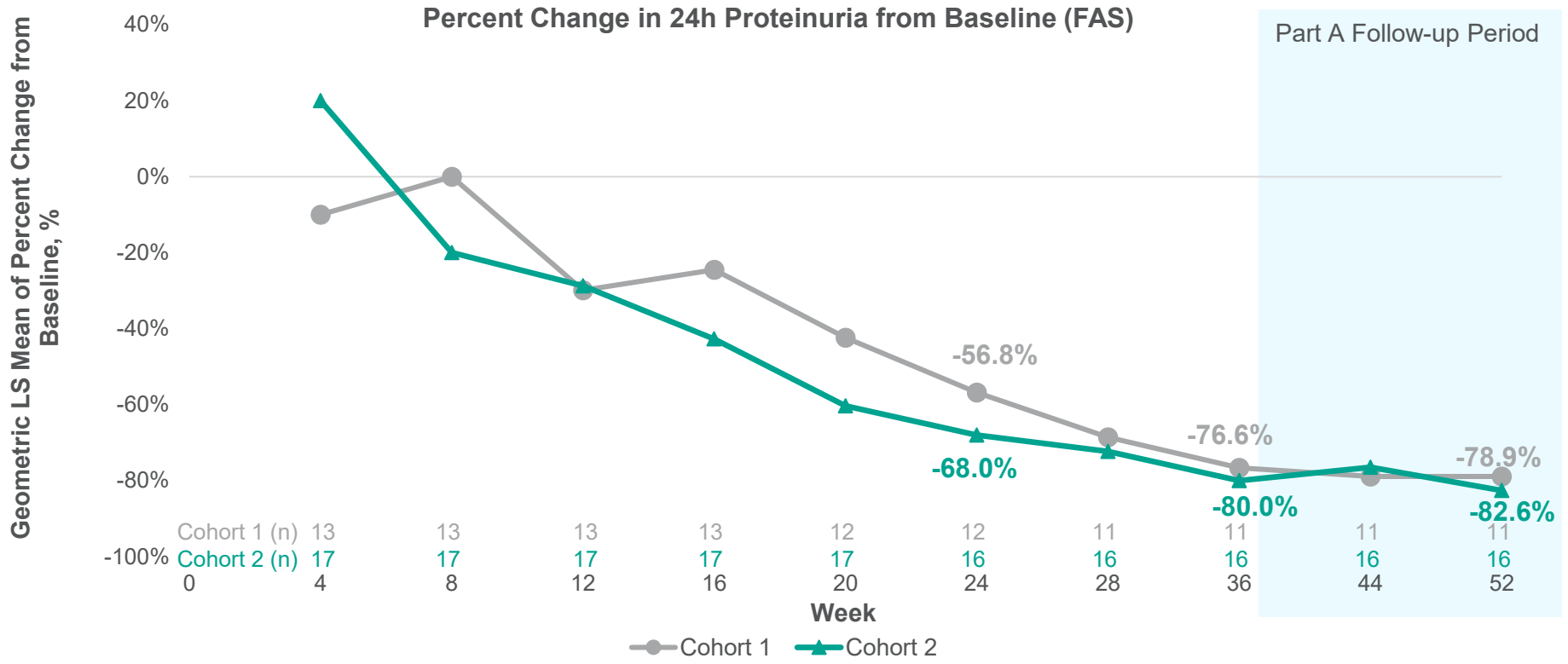
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TRAVERE® THERAPEUTICS

Phase 1/2 Proof of Concept: Civorebrutinib Demonstrated Rapid and Sustained Reductions in Key Disease Biomarkers

~80%

reduction in proteinuria in both cohorts by week 36 sustained after treatment discontinuation



Note: data cutoff date: September 5, 2025. Presented at 63rd ERA Congress in Glasgow, UK, on June 3-6, 2026.

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Favorable Safety and Tolerability Profile

No clinically meaningful AEs typically associated with BTK inhibitors, such as neutropenia, hemorrhage, cardiac arrythmia

Category of AEs, n (%)	Cohort 1 (N=13) 100mg QD→ 100mg BID	Cohort 2 (N=18) 200mg BID	Total (N=31)
Treatment emergent AE (TEAE)	12 (92.3)	15 (83.3)	27 (87.1)
Serious TEAE	3 (23.1)	3 (16.7)	6 (19.4)
≥CTCAE Grade 3 TEAE	3 (23.1)	3 (16.7)	6 (19.4)
Treatment-related TEAE	10 (76.9)	9 (50.0)	19 (61.3)
Treatment-related serious TEAE	2 (15.4)	2 (11.1)	4 (12.9)
≥CTCAE Grade 3 treatment-related TEAE	2 (15.4)	2 (11.1)	4 (12.9)
TEAE leading to discontinuation	0	2 (11.1)	2 (6.5)
TEAE leading to death	0	0	0

The most common treatment related TEAEs (>= 2 subjects) in all patients were anemia (12.9%), B-lymphocyte count decreased (9.7%), dizziness (9.7%), URTI (6.5%), hyperkalemia (6.5%), palpitations (6.5%), nausea (6.5%), and vomiting (6.5%).

Abbreviations: AE: adverse event; CTCAE: common terminology criteria for adverse events; TEAE: treatment-emergent adverse event; URTI: upper respiratory tract infection.
Note: TEAEs is defined as AEs occurred or worsened in severity or relationship to study treatment from first dose of study treatment throughout the treatment period and including the follow-up period.

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With the Potential for Rapid, Controllable Immune Modulation, Civorebrutinib is a Viable Candidate to Address Key Gaps in pMN Treatment

Civorebrutinib

Rapid and sustained reductions in key disease biomarkers seen in Phase 1/2 study (anti-PLA2R and proteinuria)

Oral administration
Convenient patient-friendly option

Safety & reversibility
Potential to avoid side effects traditionally associated with immunosuppression

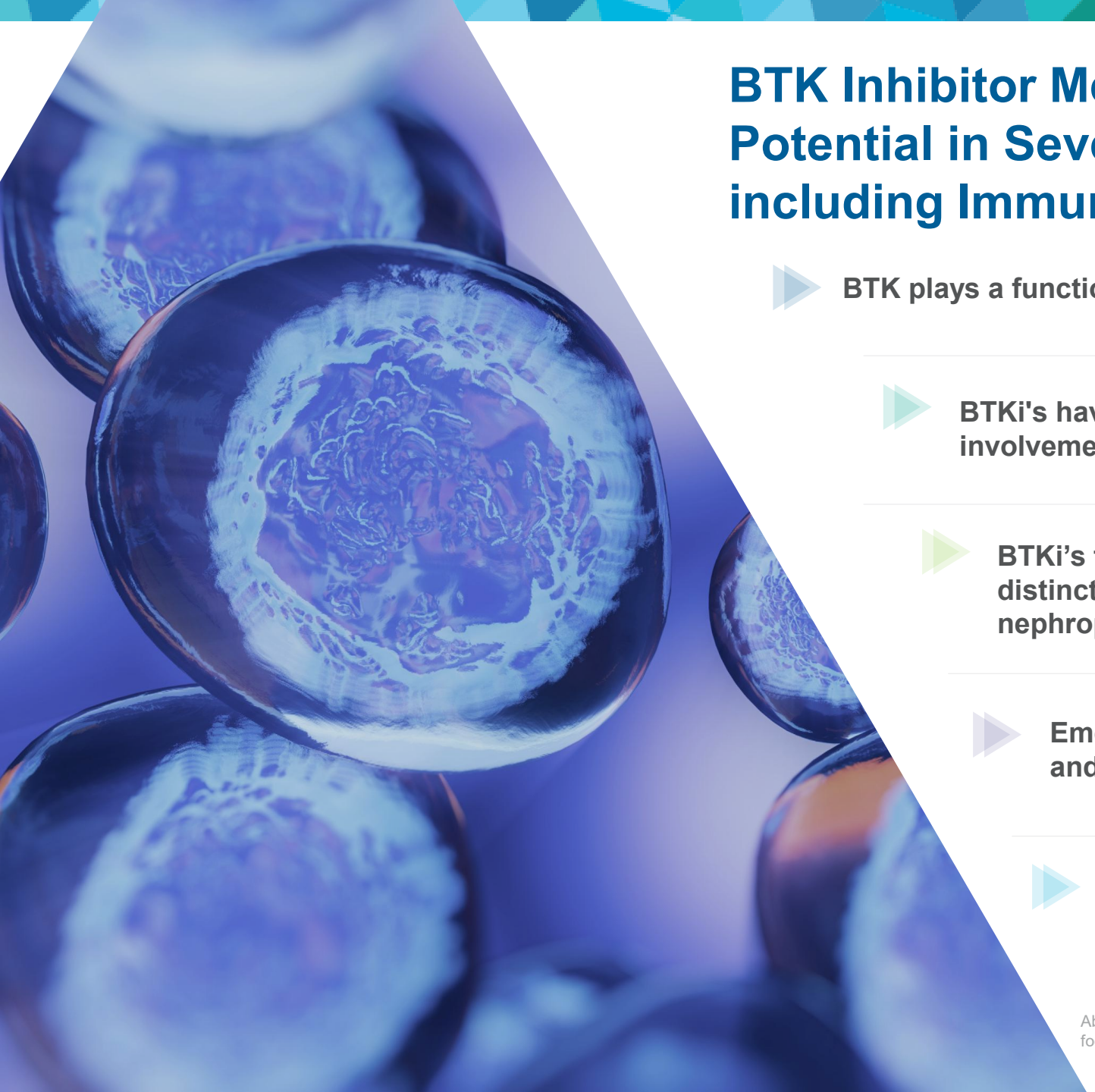
Potential to address **unmet need in patients who relapse or remain inadequately controlled** on current standard of care



Patients at high risk of progression, relapse, or inadequate response to current options need therapies with faster onset, favorable tolerability, and flexible immune modulation, representing an important opportunity for further evaluation of civorebrutinib

Abbreviations: anti-PLA2R: anti-phospholipase A2 receptor; pMN: primary membranous nephropathy.

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BTK Inhibitor Mechanism Has Promising Potential in Several Rare Kidney Diseases, including Immune-Mediated FSGS and MCD

- BTK plays a functional role in immune cell types involved in FSGS pathology
- BTKi's have a potential role in FSGS with immune cell involvement
- BTKi's target overactivation in the immune system, which is distinct and complementary to mechanisms that are nephroprotective for a heterogeneous disease like FSGS
- Emerging data suggests BTK is expressed in monocytes and macrophages in kidney biopsies of FSGS patients
- BTK biology supports further evaluation of civorebrutinib in selected immune-mediated rare kidney diseases, including FSGS and MCD

Abbreviations: BTKi: Bruton tyrosine kinase inhibitor; FSGS: focal segmental glomerulosclerosis; MCD: minimal change disease.

TRAVERE® THERAPEUTICS

A Strong Financial Foundation to Deliver New Treatment Standards in Rare Disease



~$220M

in total U.S. net product sales in 3Q26

Represents ~95% growth year-over-year



~95M

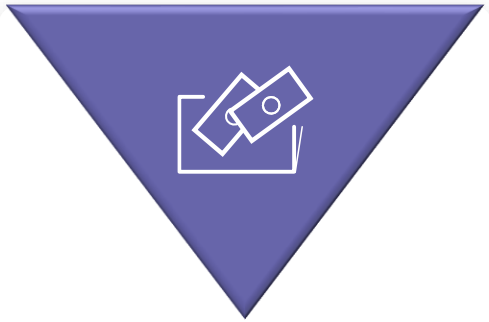
basic shares outstanding as of September 30, 2026

Diluted ~108mm[1]



~$472M

in cash and cash equivalents[2] as of September 30, 2026



~$95M

in convertible notes due March 2029

$525M due May 2032

[1] Diluted share count calculation includes all outstanding equity awards but excludes convertible notes. [2] Cash, cash equivalents and marketable securities as of September 30, 2026.

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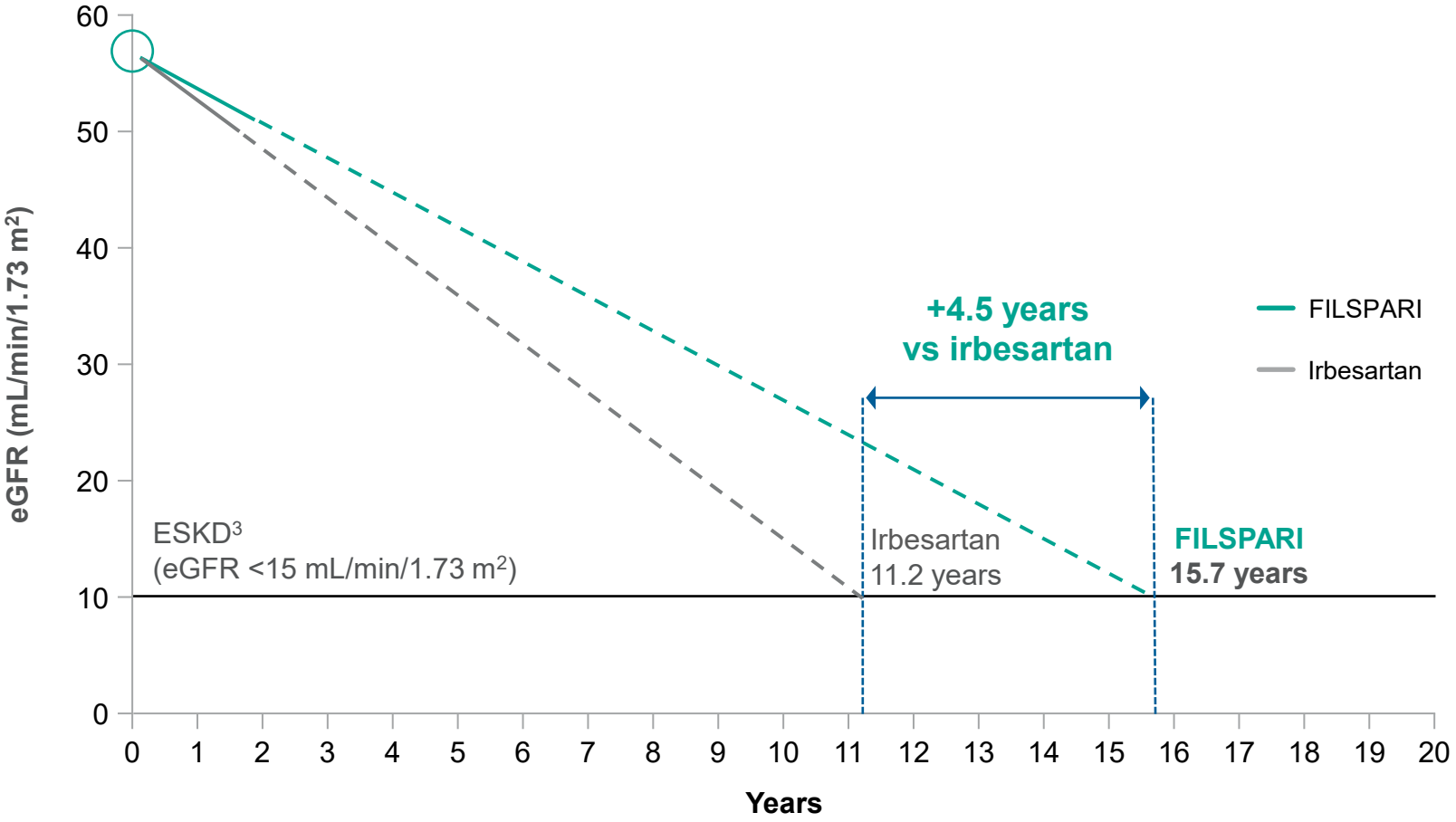
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Data Appendix

Treatment with FILSPARI May Potentially Delay Dialysis or Transplant

Potential long-term impact of preserved eGFR slope[1,2]



Based on extrapolation of eGFR slope data from PROTECT, FILSPARI may potentially **delay dialysis or transplant by 4.5 years** when compared to maximum-labeled dose irbesartan[1-3]

Abbreviations: eGFR: estimated glomerular filtration rate; ESKD: end-stage kidney disease.

[1] FILSPARI Prescribing Information.

[2] Data on file.

[3] United States Renal Data System. 2023 USRDS Annual Data Report: Epidemiology of kidney disease in the United States. NIH, NIDDK, Bethesda, MD, 2023.

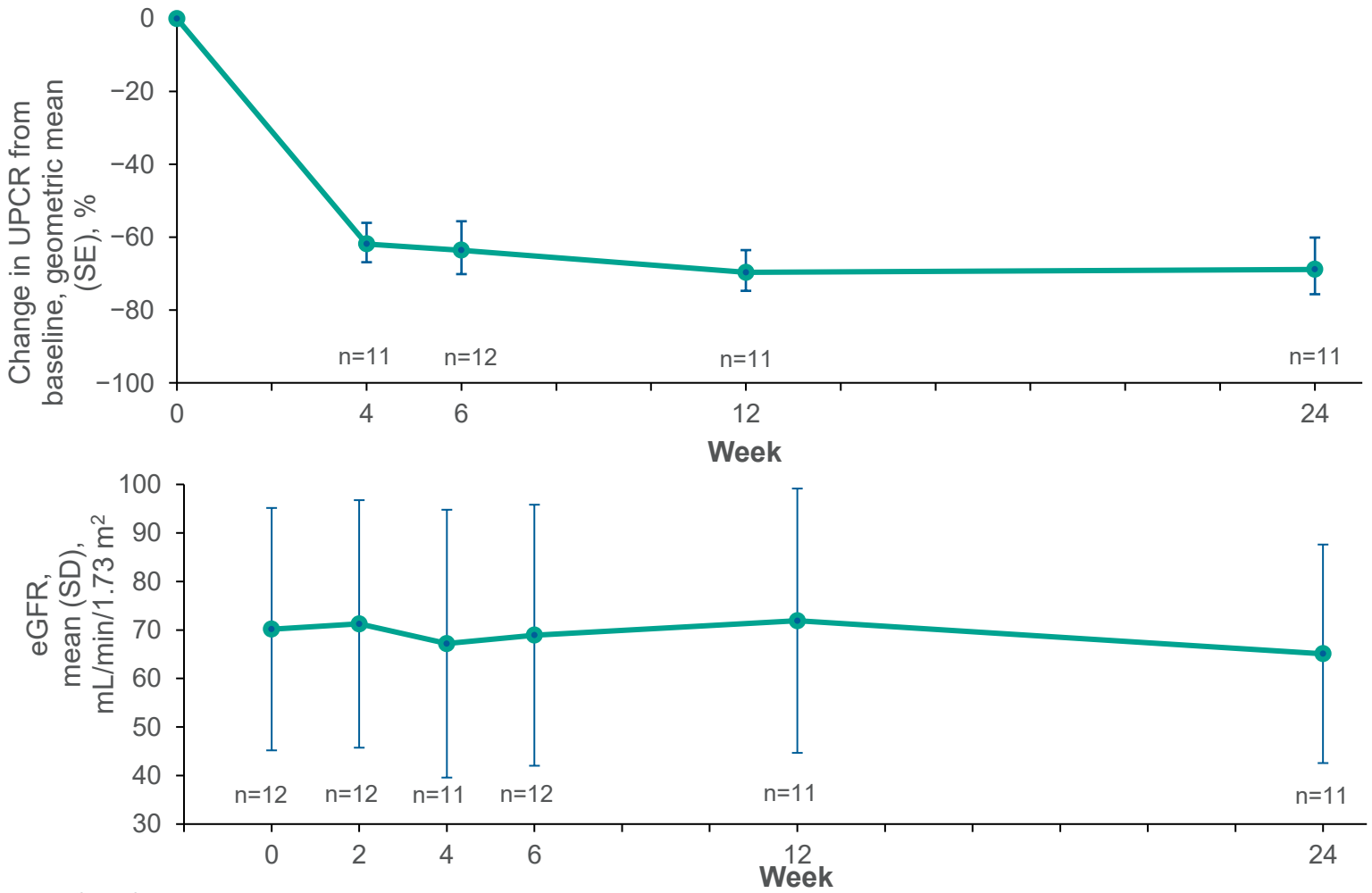
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Further Evidence Generation to Drive Broad Uptake: Supporting First-Line FILSPARI Use in Newly Diagnosed Patients

SPARTAN Study: Preliminary clinical findings at 24-weeks in treatment-naïve patients on FILSPARI

- FILSPARI led to rapid and sustained reductions in proteinuria (**~70%** from baseline) and stabilization of eGFR at week 24
- Within 24 weeks of starting FILSPARI, ~60% of patients achieved complete remission of proteinuria, a treatment goal recommended in the 2025 KDIGO guidelines[1]
- FILSPARI was generally well tolerated over 24 weeks of treatment, with no evidence of fluid retention. Safety results were consistent with the Phase 3 PROTECT Study[2,3]



Abbreviations: eGFR: estimated glomerular filtration rate; UPCR: urine protein-to-creatine ratio.
Source: Cheung CK, et al. presented at ASN 2024; October 23-27, 2024; San Diego, CA. FR-OR63.

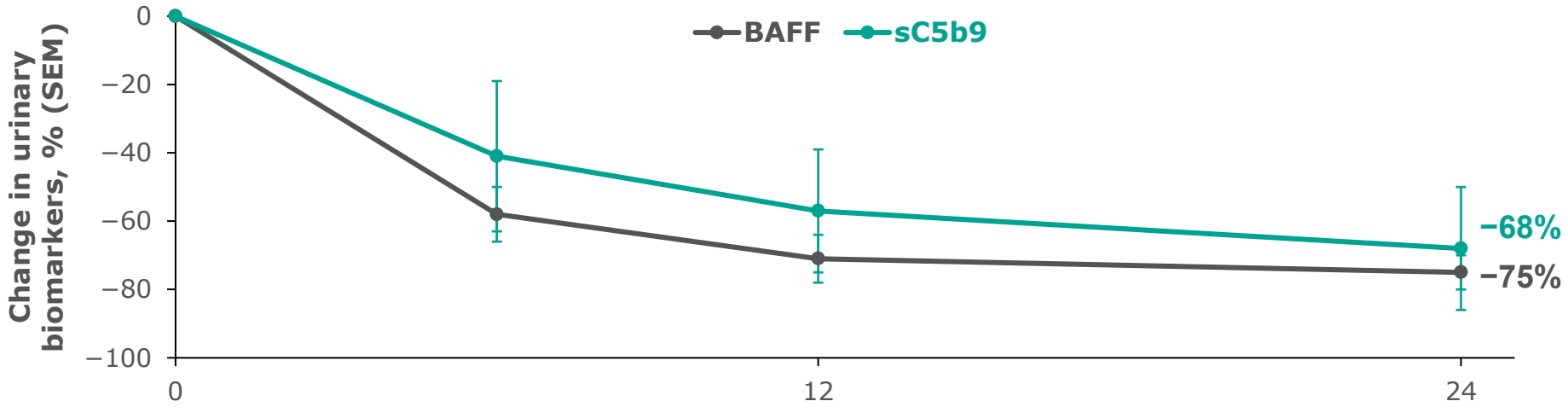
[1] KDIGO 2025 Clinical Practice Guideline for the Management of Immunoglobulin A Nephropathy (IgAN) and Immunoglobulin A Vasculitis (IgAV), October 2025.
[2] Heerspink HJL, et al. Lancet. 2023;401(10388):1584-1594.
[3] Rovin BH, et al. Lancet. 2023;402(10417):2077-2090.

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TRAVERE® THERAPEUTICS

SPARTAN Study: Urinary Biomarker Analysis Suggests Disease-Modifying Effects of FILSPARI in IgAN

Treatment with FILSPARI resulted in rapid and sustained reductions in urinary biomarkers of inflammation and fibrosis that reveal anti-inflammatory and anti-fibrotic effects of FILSPARI[1]



Change in urinary biomarkers from baseline to week 24

Inflammatory and profibrotic	α2M[2] **–83%**	CHI3L1 **–52%**	clusterin[2] **–47%**	GDF15 **–42%**	plasminogen[2] **–85%**	sCD163 **–50%**
Chemokine and cytokine	CXCL10 **–28%**	CXCL16 **–22%**	IL6 **–23%**	MCP-1 **–16%**		

Abbreviations: α2M: alpha-2-macroglobulin; BAFF: B-cell activating factor; CH13L1: chitinase-3-like protein 1; CXCL10: C-X-C motif chemokine ligand 10; CXCL16: C-X-C motif chemokine ligand 16; GDF15: growth/differentiation factor 15; IL6: interleukin 6; MCP-1: monocyte chemoattractant protein-1; sC5b9: soluble C5b9; sCD163: soluble CD163.

Source: Cheung, et al. presented at the National International Podocyte Conference & ISGD Meeting; June 10-13, 2025; Hamburg, Germany. Poster FR-11.

[1] One patient discontinued after week 6 and has been excluded from all urinary biomarker analysis (n=11).

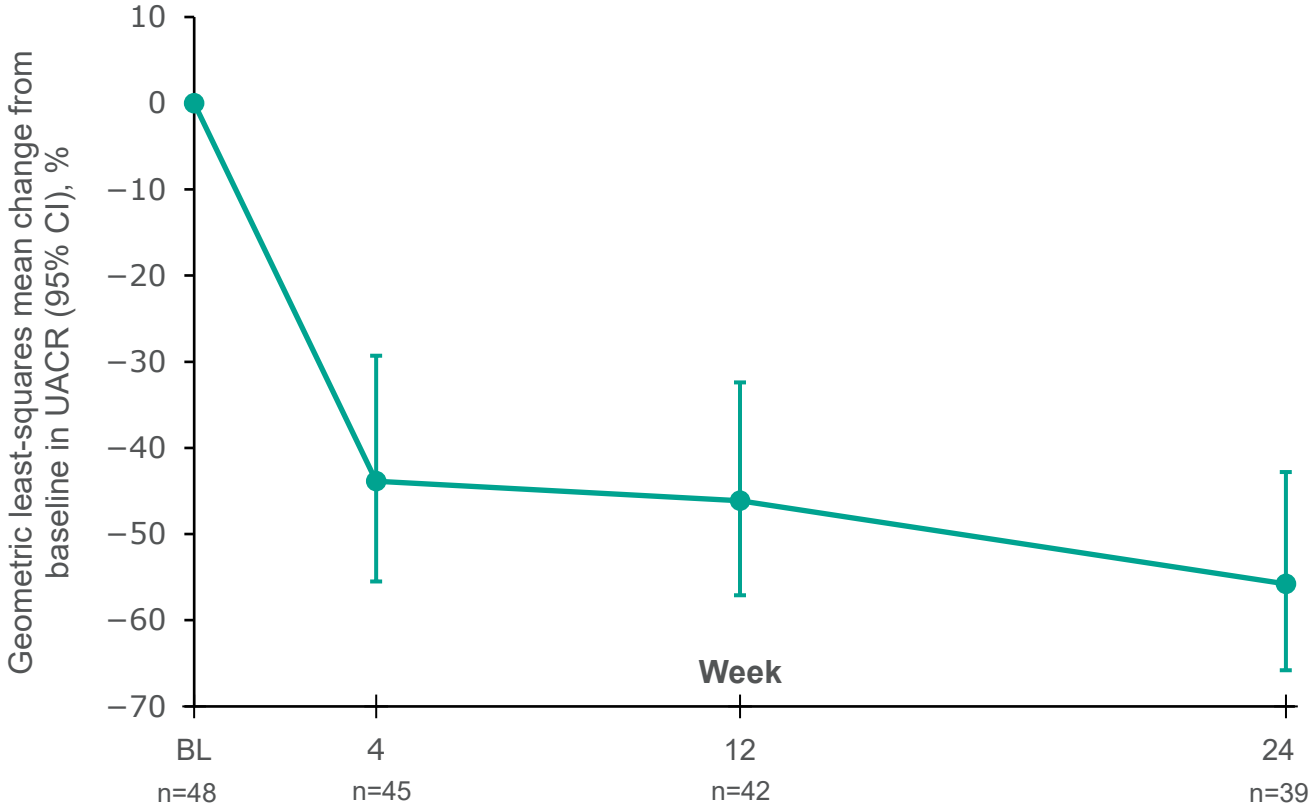
[2] α2M, clusterin and plasminogen analysis was performed only at baseline and week 12.

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SPARTACUS Study: FILSPARI Added to SGLT2i Resulted in Further Proteinuria Reduction and Was Generally Well Tolerated

Transitioning patients from RASi to FILSPARI resulted in a mean reduction in UACR of ~56% at 24 weeks



TEAEs	Patients (N=48)
Any TEAE, n (%)	**30 (63)**
SPAR related	10 (21)
SGLT2i related	2 (4)
Any TEAEs in >2 patients, n (%)	
Hypotension	7 (15)
Headache	4 (8)
Edema	4 (8)
Peripheral edema	4 (8)
Upper respiratory tract infection	4 (8)
Dizziness	6 (6)
Any severe TEAE, n (%)	2 (4)
Peripheral edema	1 (2)
Gout	1 (2)
Any serious AE, n (%)	4 (8)
Acute kidney injury*†	1 (2)
Cerebrovascular accident	1 (2)
Chemical burn	1 (2)
Deep vein thrombosis	1 (2)
Osteoarthritis*	1 (2)
Any abnormal liver function test results >3×ULN, n (%)‡	0 (0)
Any TEAE leading to SPAR discontinuation, n (%)	4§ (8)

Abbreviations: BL: baseline; RASi: renin-angiotensin system inhibitor; SGLT2i: sodium-glucose cotransporter-2 inhibitor; SPAR: sparsentan; UACR: urine albumin-to-creatinine ratio; TEAE: treatment-emergent adverse event; AE: adverse event; ULN: upper limit of normal.

Source: Ayoub I., et al. presented at ERA 2025, June 4-7, 2025; Vienna, Austria. Abstract: No. 1916.

* Reported in the same patient. †The incident of acute kidney injury was mild, deemed unrelated to SPAR or SGLT2i treatment, and was resolved after interruption of SPAR and SGLT2i. ‡Abnormal liver function test results met the following criteria: (1) new elevation in ALT or AST >3 × ULN with or without elevation of total serum bilirubin >2 × ULN and (2) 2-fold increase in ALT or AST above the baseline value in patients who had elevated values prior to taking study medication. § One patient each discontinued SPAR treatment due to a TEAE of vertigo, hypotension, peripheral edema, and Henoch-Schönlein purpura.

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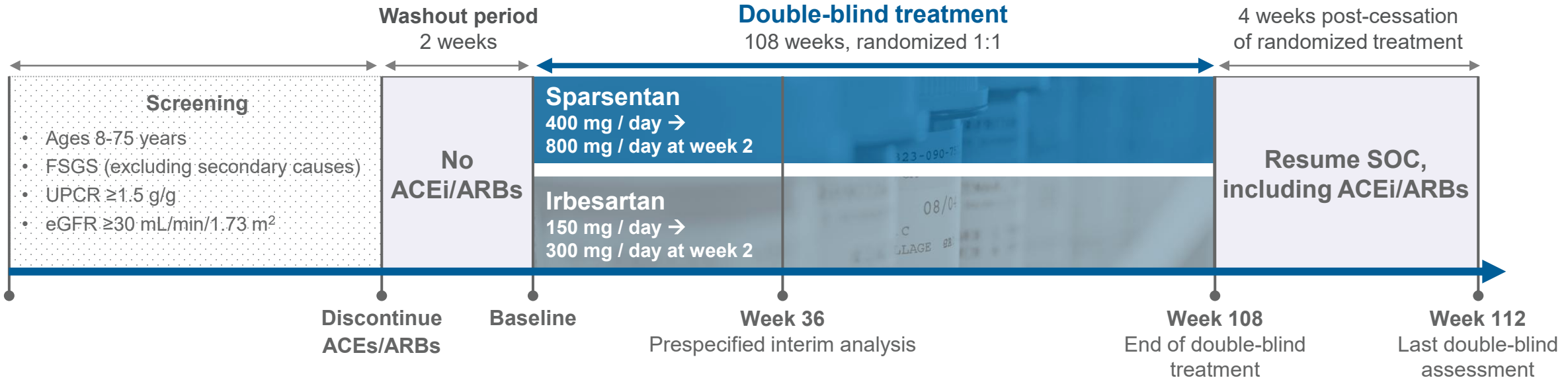
The DUPLEX Study of Sparsentan is the Largest Active-Controlled Interventional Phase 3 Trial in FSGS to Date

Objective

Evaluate the efficacy and safety of sparsentan vs. the active control irbesartan in patients with focal segmental glomerulosclerosis (FSGS)

Trial Design

- Phase 3, double-blind, active-controlled global trial in patients with biopsy-proven FSGS or genetic FSGS, N=371 patients (ages 8 to 75 years)*
- The only head-to-head Phase 3 study of its kind in FSGS
- **Surrogate efficacy endpoint:** (36-week interim analysis) = proportion of patients achieving FPRE at week 36 (UPCR ≤ 1.5 g/g and ≥ 40% reduction from baseline)
- **Primary endpoint:** eGFR total slope: From day 1 to week 108 of treatment (U.S. primary), eGFR chronic slope: From week 6 to week 108 of treatment (EU primary)



Abbreviations: ACEi: angiotensin converting enzyme inhibitors; ARBs: angiotensin receptor blockers; eGFR: estimated glomerular filtration rate; FPRE: FSGS partial remission endpoint; g/g: grams per gram; SOC: standard of care; UPCR: urine protein to creatinine ratio.

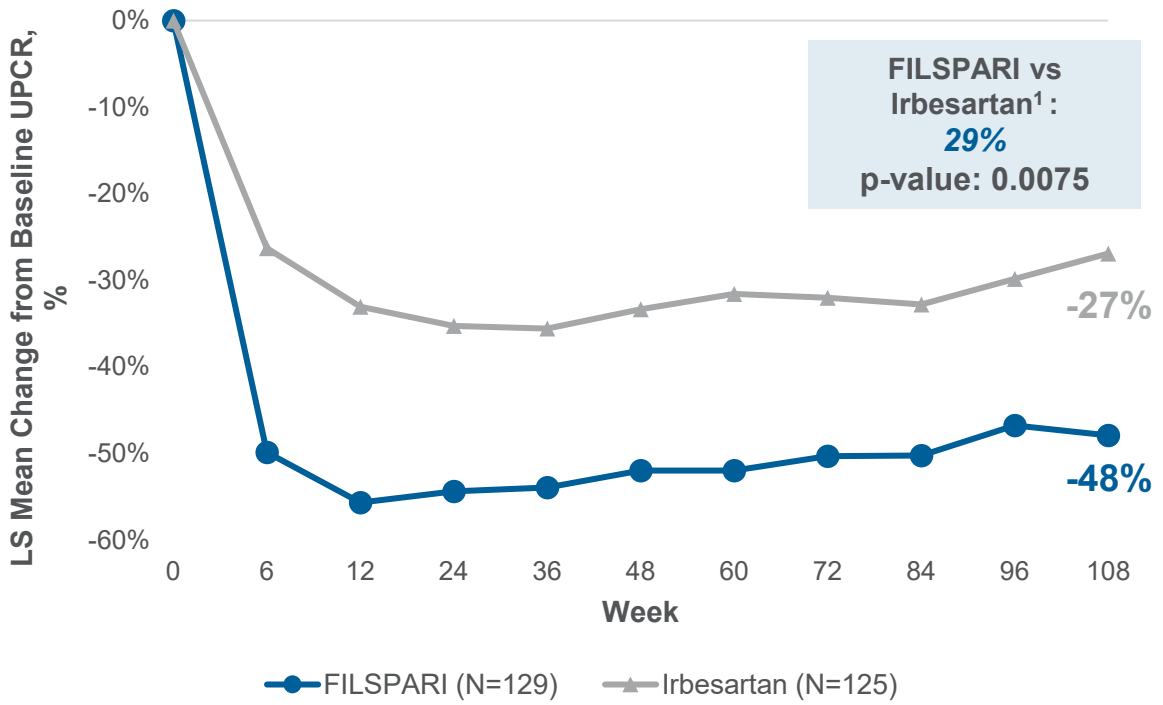
* ClinicalTrials.gov ID: NCT03493685.

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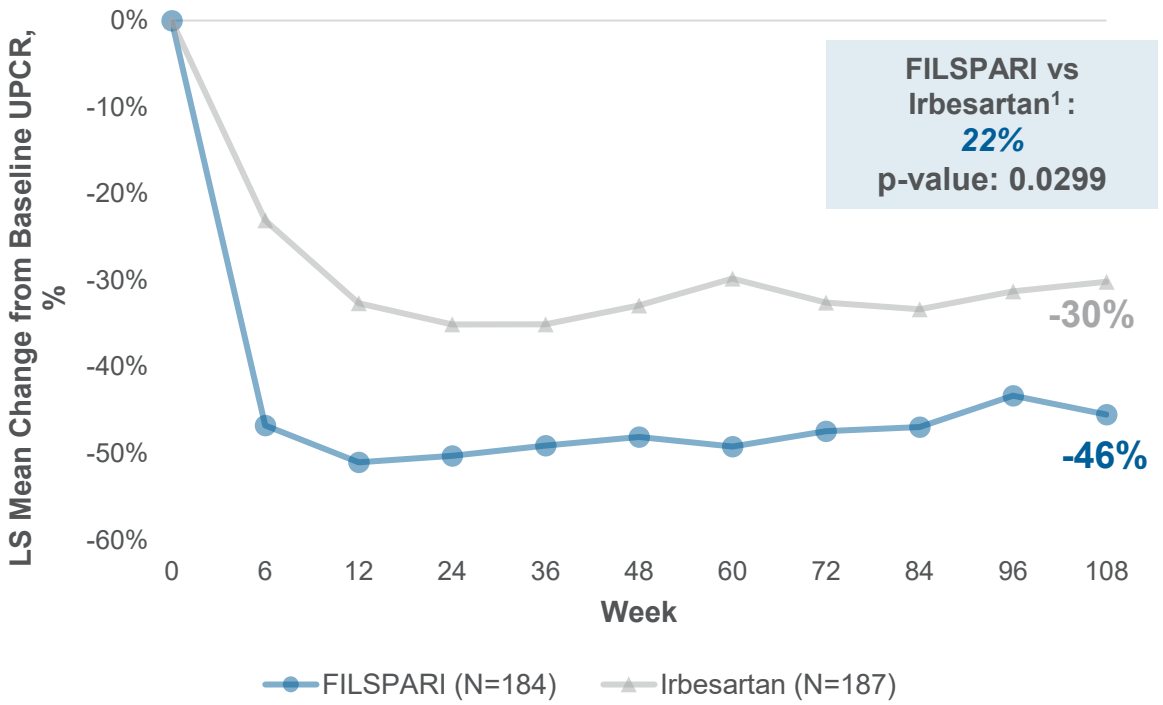
TRAVERE® THERAPEUTICS

In DUPLEX, Treatment with FILSPARI Demonstrated Even Greater Reduction in Proteinuria in FSGS without Nephrotic Syndrome

DUPLEX Population without Nephrotic Syndrome*
% reduction in UPCR from baseline by visit



Overall DUPLEX Population
% reduction in UPCR from baseline by visit



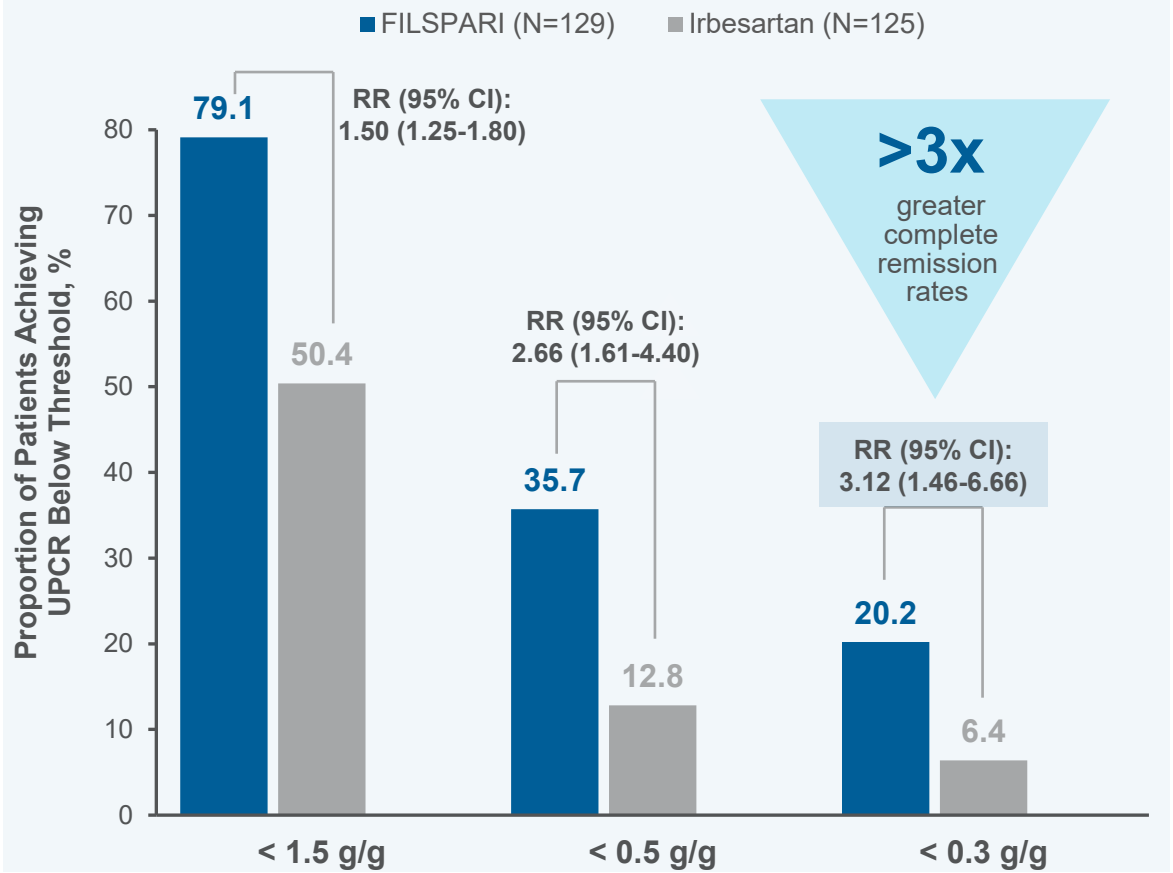
Abbreviations: CI: confidence interval; LS: least squares; UPCR: urine protein to creatinine ratio.
[1] 95% CI. All p-values are nominal.
* In the DUPLEX Study, nephrotic syndrome was defined as (a) documentation of nephrotic syndrome in the medical history, or (b) the concurrent presence of proteinuria >3.5 g/24 hours (adults) or UPCR >2.0 g/g (pediatric patients <18 years of age), serum albumin <3.0 g/dL, and edema at baseline. Patients without nephrotic syndrome did not meet both criteria (a) and (b).

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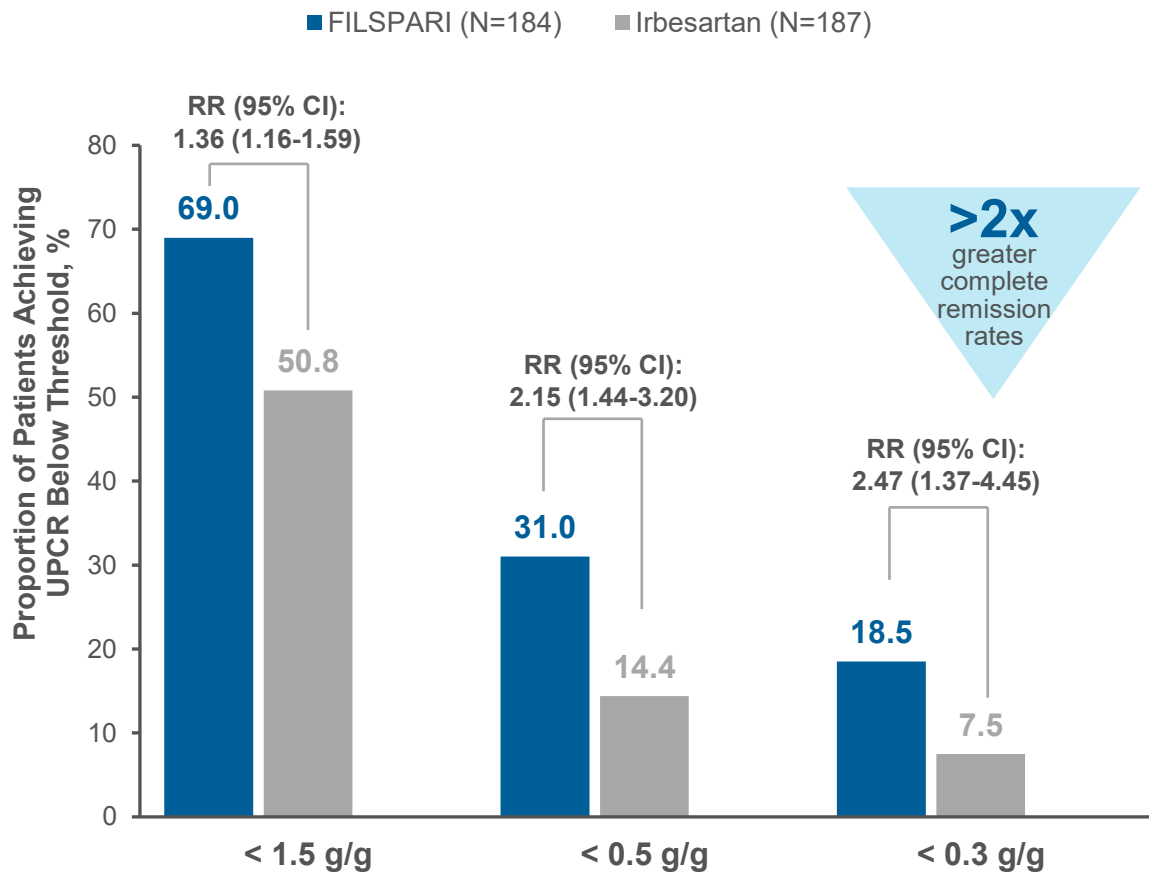
TRAVERE® THERAPEUTICS

In DUPLEX, FILSPARI Demonstrated Even Greater Relative Proteinuria Reduction Across Thresholds in FSGS without Nephrotic Syndrome

DUPLEX Population without Nephrotic Syndrome*
Patients achieving UPCR thresholds[1], %



Overall DUPLEX Population
Patients achieving UPCR thresholds[1,2], %



Abbreviations: CI: confidence interval, RR: relative risk, UPCR: urine protein to creatinine ratio.

[1] At any time during the double-blind period. All threshold analyses have nominal p-value <0.05.

[2] Source: Rheault MN, et al., *Sparsentan versus Irbesartan in Focal Segmental Glomerulosclerosis*, The New England Journal of Medicine and Supplement, 2023.

* In the DUPLEX Study, nephrotic syndrome was defined as (a) documentation of nephrotic syndrome in the medical history, or (b) the concurrent presence of proteinuria >3.5 g/24 hours (adults) or UPCR >2.0 g/g (pediatric patients <18 years of age), serum albumin <3.0 g/dL, and edema at baseline. Patients without nephrotic syndrome did not meet both criteria (a) and (b).

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TRAVERE® THERAPEUTICS

In DUPLEX, FILSPARI Demonstrated a Clinically Meaningful Effect on eGFR in FSGS without Nephrotic Syndrome

DUPLEX Population without Nephrotic Syndrome*

	FILSPARI (N=129)[1]	Irbesartan (N=125)[1]	Difference	p-value[5]
Adjusted Mean Change in eGFR (mL/min/1.73 m²) at Week 108 from baseline[2]	-11.3	-12.4	1.1	NS
Total eGFR slope (mL/min/1.73 m² per year)[3]	-4.1	-6.2	2.1	0.031
Chronic eGFR slope (mL/min/1.73 m² per year)[4]	-3.7	-6.2	2.5	0.015

Overall DUPLEX Population

	FILSPARI (N=184)[1]	Irbesartan (N=187)[1]	Difference	p-value[5]
Adjusted Mean Change in eGFR (mL/min/1.73 m²) at Week 108 from baseline[2]	-14.3	-13.3	-1.0	NS
Total eGFR slope (mL/min/1.73 m² per year)[3]	-5.4	-5.7	0.3	NS
Chronic eGFR slope (mL/min/1.73 m² per year)[4]	-4.8	-5.7	0.9	NS

Abbreviations: CI: confidence interval; eGFR: estimated glomerular filtration rate; LS: least squares; NS: not significant; UPCR: urine protein to creatinine ratio.

[1] On treatment. [2] Adjusted mean change from baseline in eGFR is obtained from the mixed model repeated measures analysis. The comparison between FILSPARI and irbesartan was based on the difference in adjusted mean change in eGFR at Week 108 with baseline on the absolute scale. [3] LS mean of annualized slope from Day 1 to Week 108. [4] LS mean annualized slope from Week 6 to Week 108. [5] Nominal p-values.

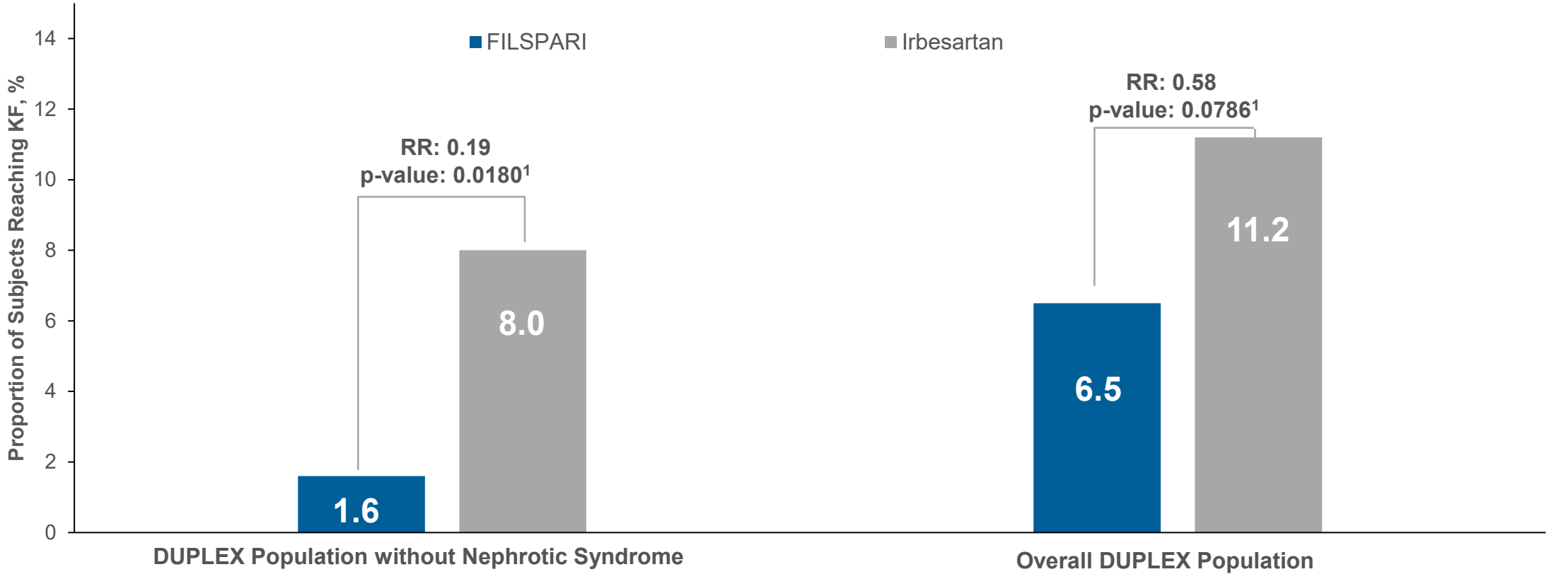
* In the DUPLEX Study, nephrotic syndrome was defined as (a) documentation of nephrotic syndrome in the medical history, or (b) the concurrent presence of proteinuria >3.5 g/24 hours (adults) or UPCR >2.0 g/g (pediatric patients <18 years of age), serum albumin <3.0 g/dL, and edema at baseline. Patients without nephrotic syndrome did not meet both criteria (a) and (b).

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TRAVERE® THERAPEUTICS

In DUPLEX, FILSPARI Demonstrated Even Stronger Benefit on Exploratory Kidney Outcome Endpoints in FSGS without Nephrotic Syndrome

FILSPARI showed clinically meaningful benefit in the broad DUPLEX population, with enhanced efficacy in population without nephrotic syndrome*



Abbreviations: CI: confidence interval; eGFR: estimated glomerular filtration rate; KF: kidney failure; RR: relative risk; UPCR: urine protein to creatinine ratio.

[1] Nominal p-values for odds ratio for rates of no events.

* In the DUPLEX Study, nephrotic syndrome was defined as (a) documentation of nephrotic syndrome in the medical history, or (b) the concurrent presence of proteinuria >3.5 g/24 hours (adults) or UPCR >2.0 g/g (pediatric patients <18 years of age), serum albumin <3.0 g/dL, and edema at baseline. Patients without nephrotic syndrome did not meet both criteria (a) and (b).

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