

Travere Therapeutics Corporate Overview

January 2023



This presentation contains forward-looking statements, including statements about our prospects, products, growth projections, competitive position, potential regulatory filings, and agency actions, and the anticipated development, timing, data readouts, and therapeutic scope of programs in our clinical pipeline. These forward-looking statements may be accompanied by such words as "anticipate," "believe," "estimate," "expect," "forecast," "intend," "may," "plan," "project," "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, including the safety and efficacy of our product candidates, product competition, market acceptance, the occurrence of adverse safety events with our products or product candidates, clinical trials risk, adverse market and economic conditions, regulatory uncertainty, our dependence on collaborations and other third parties over which we may not always have full control, failure to comply with government regulation, our ability to protect our intellectual property rights, and have sufficient rights to market our products and services together with the cost of doing so, problems with our manufacturing processes and our reliance on third parties, the potential impact of the ongoing COVID-19 pandemic, our ability to attract and retain qualified personnel, our level of indebtedness, environmental risks, change of control provisions in our collaborations, 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.



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.





Travere is Positioned for Sustainable Growth and Rare Disease Leadership



Patient-inspired culture rooted in personal rare disease experience



Integrated operations with clinical development and commercial expertise in rare disease



Diversified pipeline of potential first-in-class programs targeting rare diseases with no or limited treatment options currently available



Commercial organization prepared for successful launches from pipeline

First launch of sparsentan expected in 1Q23*



Potential opportunity for multiple commercial products and significant value creation



Experienced Team with Distinguished Track Record of Successful Execution



Eric Dube, PhD President and Chief Executive Officer





Chris Cline Chief Financial Officer

élan



Peter Heerma Chief Commercial Officer

AMGEN abbvie



Jula Inrig, MD Chief Medical Officer



Casey Logan SVP, Corporate and Business Development





Elizabeth Reed, SVP General Counsel and Corporate Secretary





Bill Rote, PhD SVP, Research and Development





Charlotte Smith SVP, Public Affairs





Angela Giannantonio SVP, Human Resources





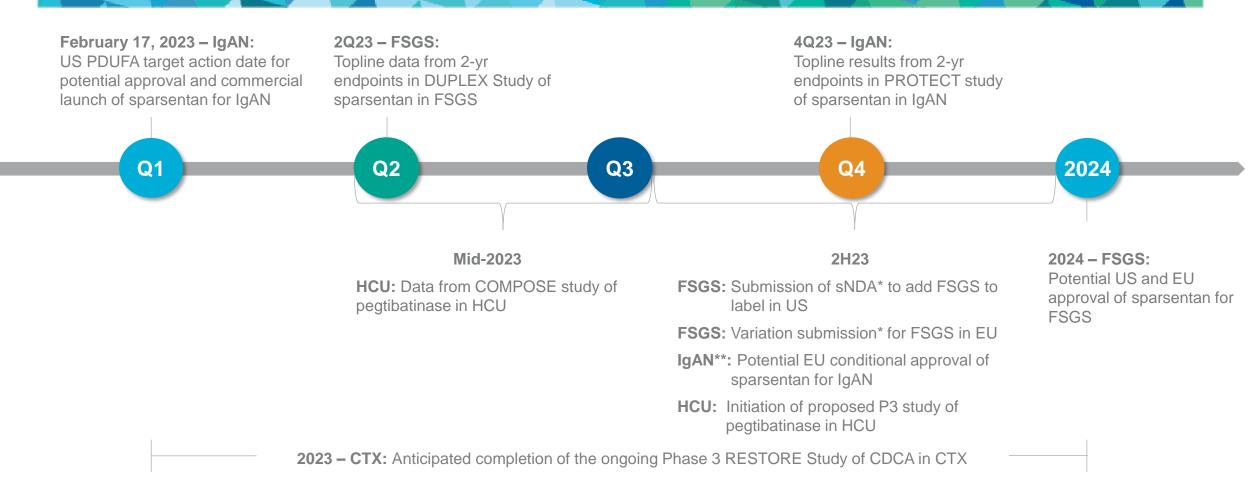
Pipeline of Potential First-in-Class Programs Targeting Rare Diseases

PROGRAM	THERAPEUTIC AREA	PRECLINICAL	PHASE 1	PHASE 2	PHASE 3
Sparsentan	<u>FSGS</u>				
Sparsentan	<u>IgAN</u>				
CDCA*	<u>CTX</u>				
Pegtibatinase (TVT-058)**	<u>HCU</u>				
ALGS Collaboration	<u>ALGS</u>				

*CDCA is not indicated for CTX but has received a medical necessity determination in the US by the FDA for CTX. Travere Therapeutics is conducting a Phase 3 clinical trial to examine the safety and efficacy of CDCA (Chenodal®) for the treatment of CTX. **Pegtibatinase is currently in a Phase 1/2 clinical study.



Upcoming Milestones in 2023 to Further Position Travere as a Leader in the Rare Disease Community



Furthering our mission of delivering life-changing therapies to people living with rare disease



*Pending supportive data and US/EU approval of sparsentan for IgAN ** In partnership with European collaborator CSL Vifor

Sparsentan

The first and only Dual Endothelin Angiotensin Receptor Antagonist (DEARA) in development for the treatment of rare kidney disease



There is a Serious Unmet Need for Patients with Rare Kidney Disease

With standard of care failing patients living with rare kidney disease, the global burden continues to grow as incidence and prevalence of IgAN and FSGS increase.

	IgA Nephropathy		
•	Most reported primary glomerulonephritis or inflammation of the glomeruli resulting in immunoglobulin A (IgA) accumulation in the blood, resulting in inflammation and damage to the kidney's filtering capabilities	•	A to P th

- ~30-40% of patients have a progressive course of IgAN resulting in ESKD
- Patients with persistent proteinuria (>1gr/day) have more dire prognoses and are at greater risk for progression to ESKD
- **50%** of IgAN patients are on max tolerated ACE/ARB and **continue to excrete proteinuria**
- Occurs at any age, with peak incidence at 25-39 years

Median time to kidney failure in high-risk patients is 10.7 years

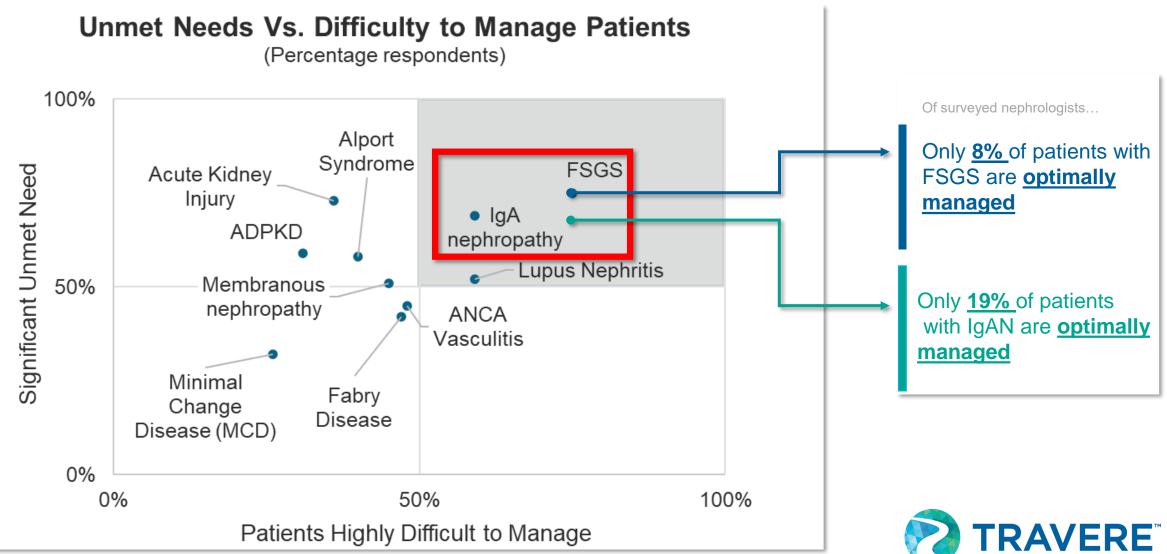
Focal Segmental Glomerulosclerosis (FSGS)

- Aggressive and progressive sclerosis of the glomeruli that can lead to ESKD
- Primary FSGS generally affects patients in their mid-twenties to thirties
- High proteinuria levels in (sub)nephrotic range is hallmark of disease
- More than 70% of newly diagnosed adult patients with primary FSGS have proteinuria in the nephrotic range
- Majority of patients relapse, many within 20-36 months
- **30-60%** progress to **ESKD with 5-10 years**; **recurrent disease** develops in **40%** of transplant patients



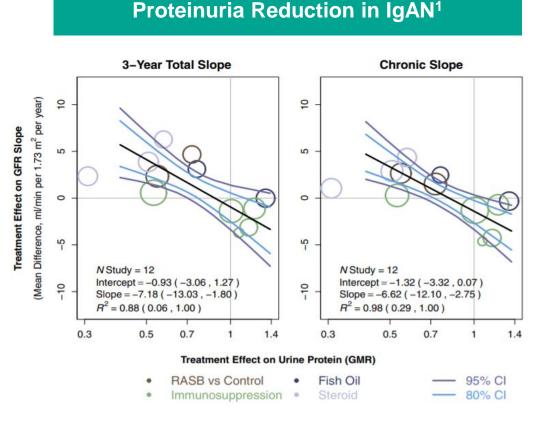
Sources: Gipson et al. *Kidney Int.* (2011); Healthagen 2007 – 2019; Korbet et al., *J Am Soc Nephrol.* (2012); Market Dynamix 2020; USRDS (2019); Rauen et al. *Kidney Int.* (2020); Moranne et al., *Q J Med* (2008) Jarrick et al., *JASN* (2019); Le et al., *Nephrol Dial Transplant* (2011); Selvaskandan et al., *Clin and Exp Nephrol* (2019); proprietary market research. ¹Estimated based on McGrogan et al. *Nephrol Dial Transplant* (2011); Sim et al., *AJKD* (2016); Sim on et al., 2004; Zara et al. *Nephrol Dial Transplant* (2013); Braun et al., *Int Urol Nephrol* (2011); data on file. Additional sources: Korbet et al., *J Am Soc Nephrol.* (2012); Kitiyakara et al., *Am J Kidney Dis.* (2004); USRDS (2019); KDIGO, *Kidney Int Suppl* (2011);

Nephrologists Consider Patients with IgAN and FSGS to Have Significant Unmet Needs and are Amongst the Most Challenging to Manage



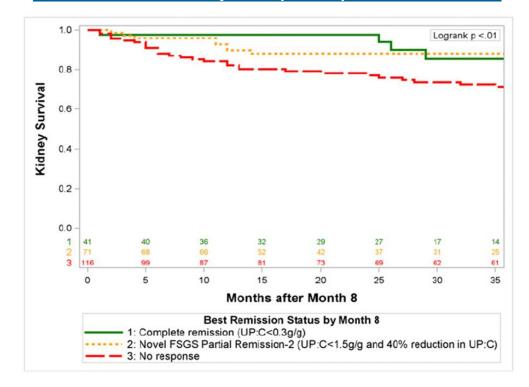
Source: Independent market research, data on file

Reductions in Proteinuria Have Been Tied to Improved Kidney Outcomes in Third-Party Studies Evaluating Patients with Both IgAN and FSGS



Individual-patient meta-analysis including data from 1,037 patients with IgAN across 12 trials showed that treatment effects on urine protein accurately predicted treatment effects on the total GFR slope at 3 years and on chronic GFR slope

FSGS Partial Remission of Proteinuria Endpoint (FPRE)²

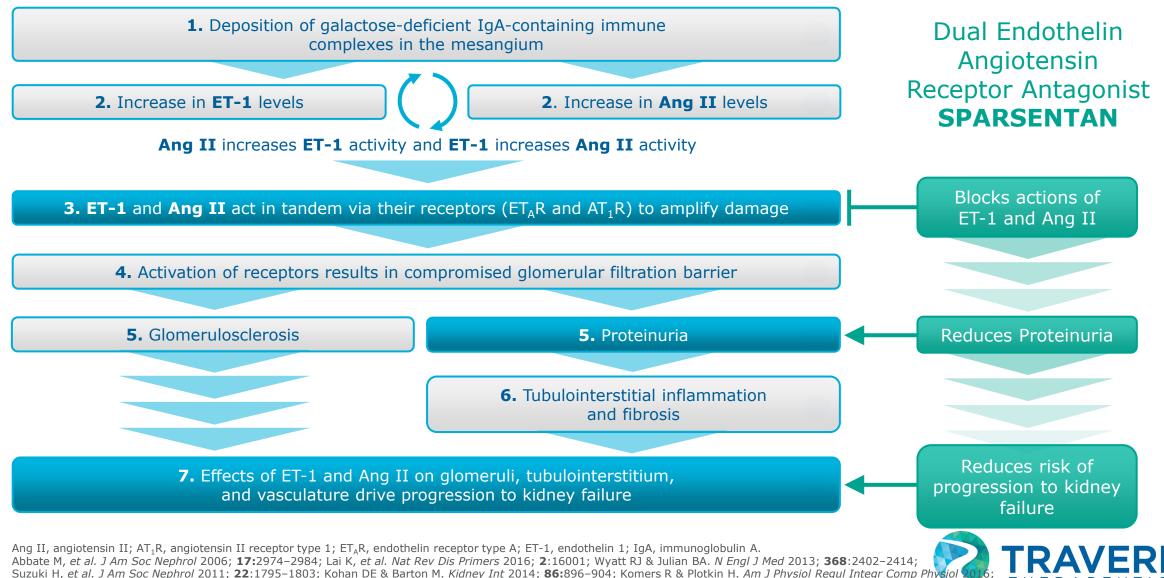


Data from five independent cohorts totaling 466 patients with primary FSGS showed that achieving a modified partial remission of proteinuria endpoint of <1.5 g/g accompanied with at least a 40% reduction in proteinuria was a robust correlate of kidney survival



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Effects of Sparsentan in the Pathophysiology of IgA Nephropathy



310:R877–R884; Raina R, et al. Kidney Dis 2020; **6**:22–34. Figure adapted from: Lai K, et al. Nat Rev Dis Primers 2016; **2**:16001.

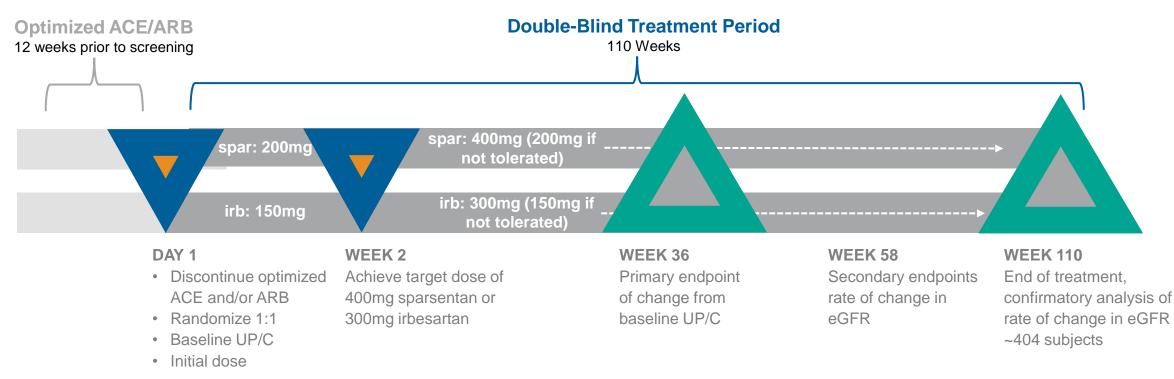


Leading Clinical Development in Rare Kidney Disease with Sparsentan

IgAN (IgA nephropathy)	 Ongoing Pivotal Phase 3 PROTECT Study in IgAN 404 patients 1:1 vs active control (irbesartan) ✓ Interim proteinuria endpoint evaluated at 36 weeks eGFR confirmatory endpoint will be evaluated at 110 weeks of treatment 	 >500 patients with IgaN and FSGS have received sparsentan in
FSGS (Focal segmental glomerulosclerosis)	 Ongoing Pivotal Phase 3 DUPLEX Study in FSGS 371 patients 1:1 vs active control (irbesartan) ✓ Interim proteinuria endpoint evaluated at 36 weeks eGFR confirmatory endpoint will be evaluated at 108 weeks of treatment 	 Clinical trials Earliest patients in the DUET OLE have been on
FSGS (Focal segmental glomerulosclerosis)	 Phase 2 DUET Study in FSGS 109 patients 2:1 vs active control (irbesartan) ✓ Proteinuria endpoint at 8 weeks eGFR observed throughout OLE (ongoing) 	sparsentan for more than seven years



Phase 3 PROTECT Study Designed to Support NDA & MAA Submissions for IgAN

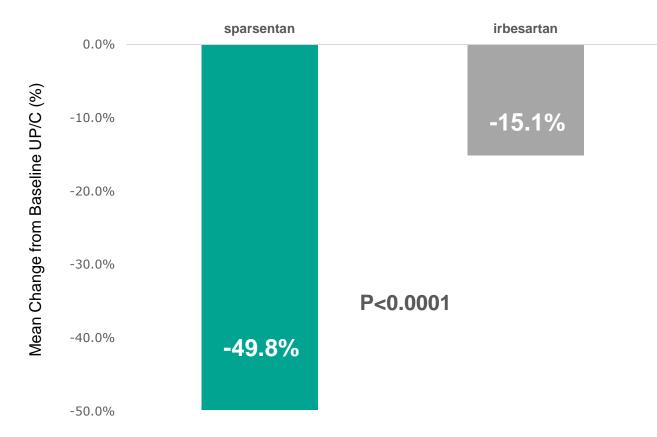


- Patients treated in the PROTECT study are those at high risk of progressing to renal failure; believed to be largest controlled study to date in IgAN (N=404)
- PROTECT is fully enrolled and is scheduled to continue on a blinded basis to assess the confirmatory eGFR endpoint after 110 weeks of treatment
- SGLT2i Combination Studies: the Company plans to expand data generation through a sub study in the open-label extension of the ongoing PROTECT Study, as well as an open-label clinical study to investigate the safety and efficacy of sparsentan in combination with sodium glucose cotransporter-2 inhibitors (SGLT2i) for the treatment of IgAN



IgAN: Ongoing Phase 3 PROTECT Study Met its Interim Reduction of Proteinuria Endpoint

Sparsentan demonstrated a greater than 3x reduction of proteinuria from baseline after 36 weeks of treatment, compared to the active control irbesartan

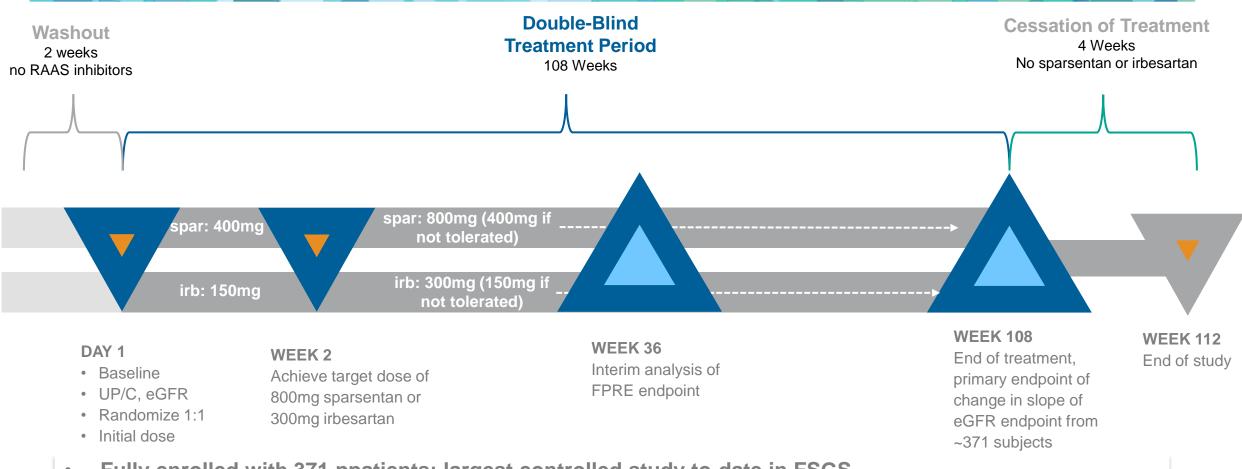


- **41% difference in GMR:** PROTECT Study was designed to detect a 30% difference in the geometric mean ratio (GMR) of proteinuria reduction between sparsentan and irbesartan
- **Preliminary eGFR data:** believed to be indicative of a potential clinically meaningful treatment effect after two years of treatment based on data available at the time of the interim analysis
- **Safety:** sparsentan was generally well-tolerated, and appeared consistent with the previously observed safety results with no new safety signals emerging



36 Week Interim Results in Ongoing 110 Week Study

FSGS: Phase 3 DUPLEX Study Designed to Support Potential NDA & MAA Submissions

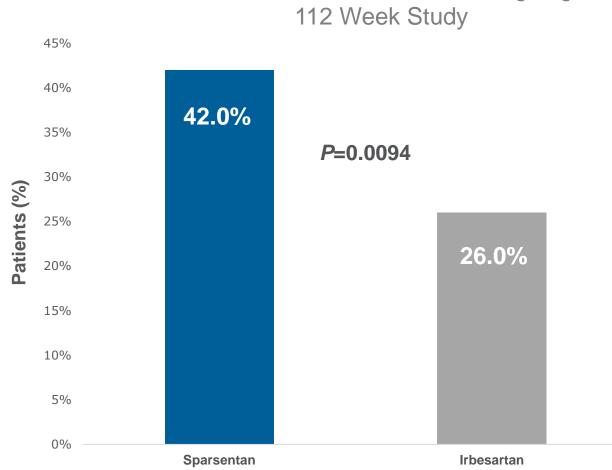


- Fully enrolled with 371 ppatients; largest controlled study to-date in FSGS
- DUPLEX is scheduled to continue as planned on a blinded basis to assess the confirmatory eGFR endpoint after 108 weeks of treatment



FSGS: Ongoing Phase 3 DUPLEX Study Achieved Interim FPRE **Proteinuria Endpoint**

The FSGS partial remission endpoint (FPRE) is defined as >40% reduction in proteinuria to a UP/C ≤1.5 g/g

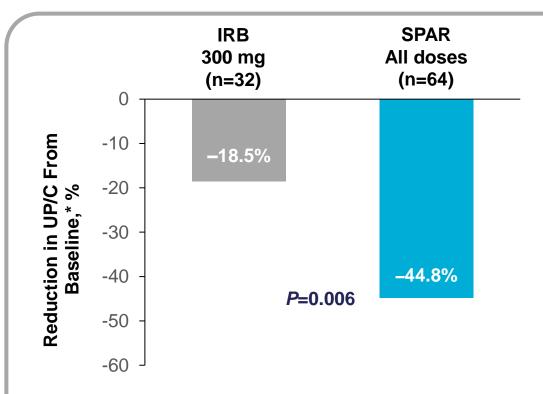


36 Week Interim Results in Ongoing

- Treatment with sparsentan resulted in a 60% greater relative likelihood of achieving FPRE when compared to irbesartan
- At the time of the interim assessment. sparsentan was generally welltolerated and had shown a comparable safety profile to irbesartan



FSGS: Phase 2 DUET Study - Overall Sparsentan Treatment Group Met Primary Endpoint; More Than Doubled Reduction of Proteinuria vs. Irbesartan



	Patients with TEAEs During the Double-Blind Period, %				
TEAE	IRB (n = 36)	SPAR, All Doses (n = 73)			
Any	72.2	76.7			
Drug-related	36.1	43.8			
Serious	2.8	2.7			
Leading to dose change or interruption	8.3	23.3			
Leading to drug discontinuation	2.8	4.1			
Leading to study withdrawal	2.8	2.7			
Death	0	0			

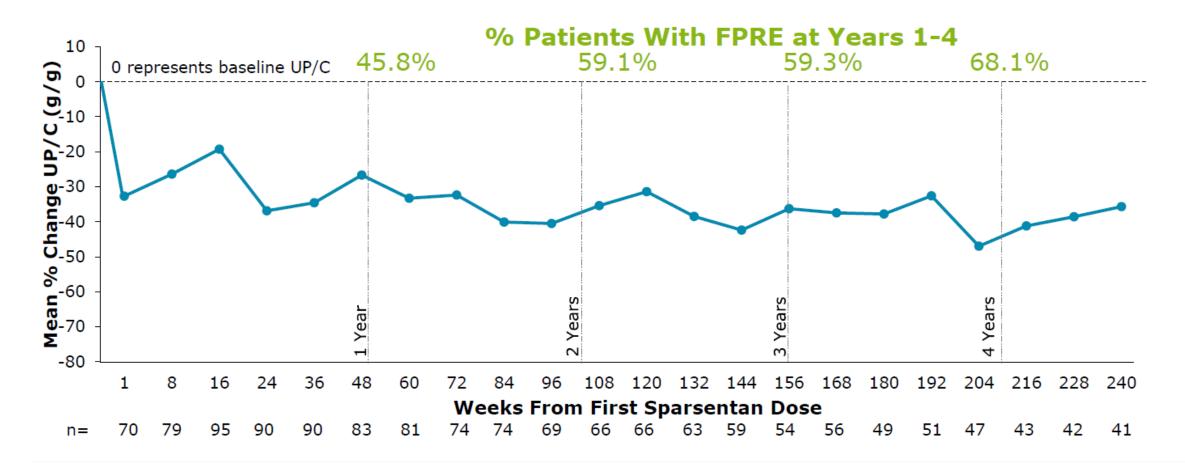
Significant reduction in proteinuria after 8 weeks of sparsentan vs irbesartan treatment in primary and genetic FSGS patients

Similar incidence of TEAEs between irbesartan and sparsentan-treated patients

*Geometric least squares mean reduction. P values from analysis of covariance. Analyses based on the efficacy evaluable set. UP/C based on 24-hour urine. Individual dose cohorts showed clear signals of relative improvement, but did not reach statistical significance; H Trachtman, *et al. J Am Soc Nephrol* 2018; 29:2745–2754. Loreto G, *et al. ERA-EDTA* 2017 Oral presentation TO042



FSGS: Phase 2 DUET Study OLE: Treatment With Sparsentan Resulted in Sustained Reductions in Proteinuria



43% of patients experienced >1 complete remission of proteinuria at any time

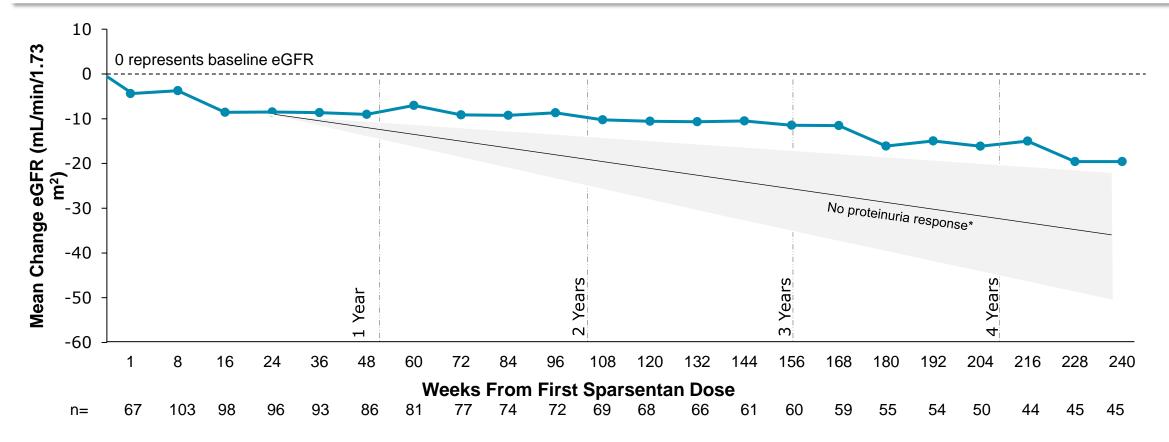
Sparsentan data presented at ASN Kidney Week 2022; Only on-treatment observations (defined as occurring within 1 day of last sparsentan dose) are included. FPRE (UP/C ≤1.5 g/g and 6 >40% reduction in UP/C from baseline). FPRE, FSGS partial remission endpoint.



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FSGS: Phase 2 DUET Study OLE: Treatment With Sparsentan is Associated With Slower Decline in eGFR vs Natural History Estimates

Chronic slope estimate through 108 weeks: -3.56 (95% CI: -5.6, -1.5) mL/min/1.73m²/year Chronic slope estimate all on-treatment data: -4.16 (95% CI: -5.8, -2.5) mL/min/1.73m²/year



Sparsentan data presented at ASN Kidney Week 2022; Only on-treatment observations (defined as occurring within 1 day of last sparsentan dose) are included. Chronic slope was assessed starting at Day 42 of starting sparsentan treatment. CI, confidence interval.

*Troost JP, et al. Am J Kidney Dis. 2021;77(2):216-225.



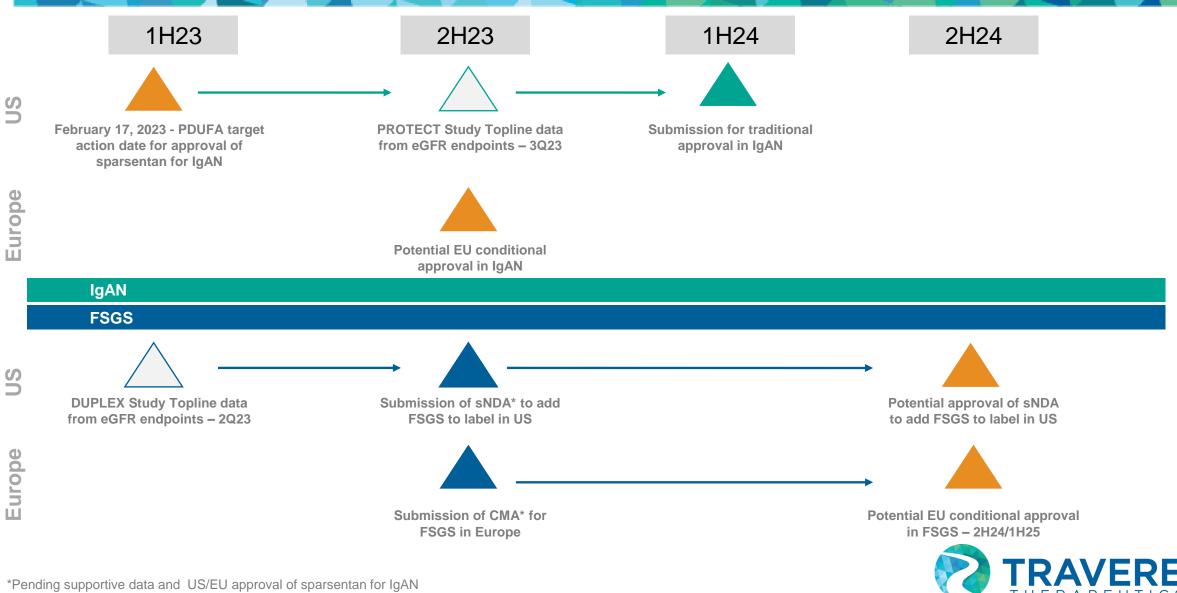
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Sparsentan has Demonstrated Consistent Anti-Proteinuric Responses Across Phase 2 and Phase 3 Clinical Trials

PROTECT	sparsentan											49.8%
IgAN, mean change from baseline UP/C (%) at 36 weeks P<0.0001	irbesartan				15.1	۱%						
Output	sparsentan										42.0%	
FSGS, FPRE response at 36 weeks P=0.0094	irbesartan		-	-	-	-	2	6.0%				
FSGS overall treatment group, mean change from baseline UP/C (%) at 8 weeks	sparsentan irbesartan					18.5%					44.8%)
P=0.006		0.0%	5.0%	10.0%	15.0%	20.0%	25.0%	30.0%	35.0%	40.0%	45.0%	50.0%



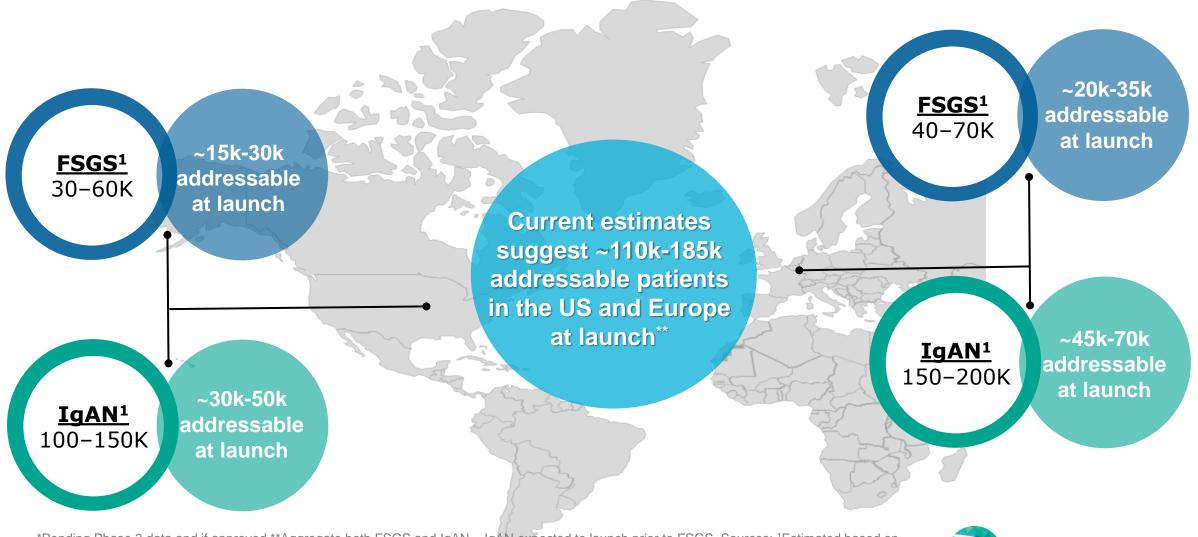
Expected Regulatory Pathways to Potential Submissions and Approvals of Sparsentan in the US and Europe



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** In partnership with European collaborator CSL Vifor

Significant Opportunity to Increase the Number of Patients Treated in the Coming Years if Sparsentan is Approved^{*}

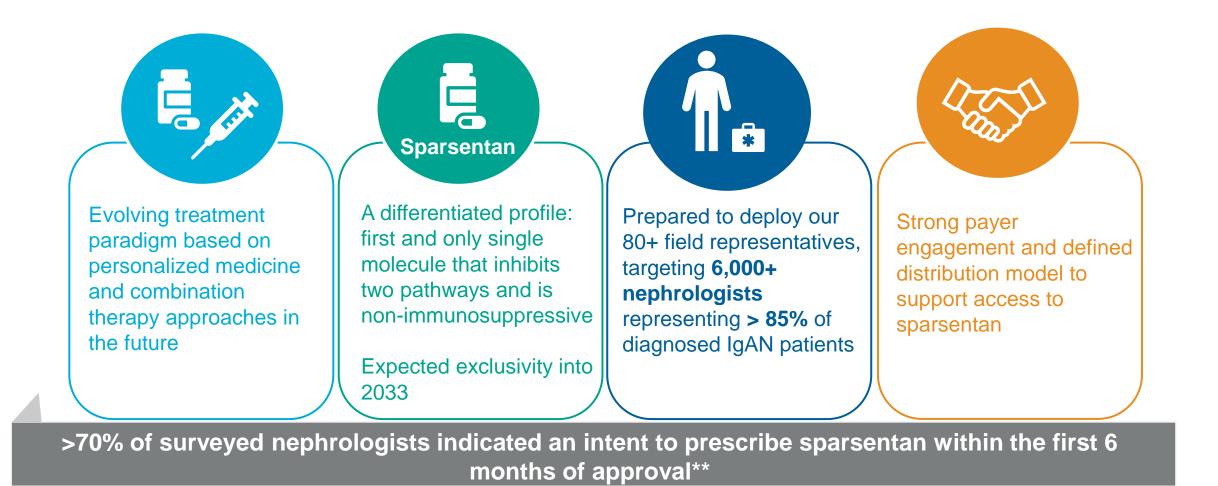


*Pending Phase 3 data and if approved **Aggregate both FSGS and IgAN – IgAN expected to launch prior to FSGS. Sources: ¹Estimated based on McGrogan et al. *Nephrol Dial Transplant* (2011); Sim et al., *AJKD* (2016); Simon et al., 2004; Zara et al. *Nephrol Dial Transplant* (2013); Braun et al., *Int Urol Nephrol* (2011); data on file. Additional sources: Korbet et al., *J Am Soc Nephrol*. (2012); Kitiyakara et al., *Am J Kidney Dis*. (2004); USRDS (2019); KDIGO, *Kidney Int Suppl* (2011);



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The IgAN Treatment Landscape is Evolving and Sparsentan has the Potential to Become a New Treatment Standard*





Joint Collaboration and Licensing Agreement with CSL Vifor; Two Leaders in Rare Nephrology to Deliver Sparsentan in the US and Europe, if Approved



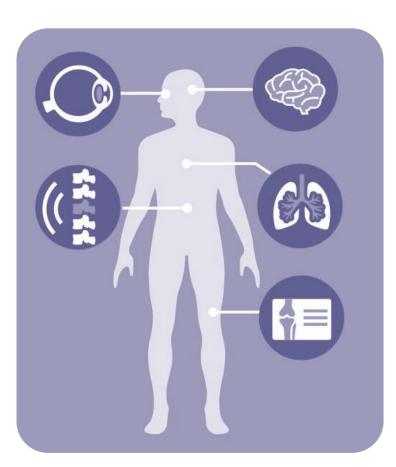
Travere to receive up to \$845 million in total milestone and upfront payments + tiered doubledigit royalties up to 40% on net sales of sparsentan in Europe, Australia, and New Zealand

Pegtibatinase

The Potential First Disease Modifying Therapy for Classical Homocystinuria (HCU)



Classical Homocystinuria (HCU) is a Rare Disorder that can Lead to Life-Threatening Complications



Rare autosomal recessive disorder caused by mutations in cystathionine beta-synthase (CBS) gene, leading to deficient activity of CBS

- Metabolic deficiency of CBS leads to bodily buildup of toxic homocysteine (Hcy)
 Toxic levels of Hcy can lead to serious complications for people living with classical HCU
- Continuous risk of developing life-threatening thrombotic events including heart attack and stroke
- Other symptoms of classical HCU include dislocation of the eye lens and extreme nearsightedness, skeletal complications including osteoporosis, and developmental delay

There are no approved treatments that address the underlying genetic cause of HCU

 Current standard of care includes vitamin B6, low-protein diet + supplements, betaine

Estimates suggest at least 3,500 patients in US, similar number in Europe

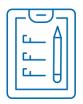
Thromboembolic events are observed in 25% of HCU patients by age 16, and 50% by age 29.



With Largely Ineffective Treatment Options, a Significant Unmet Need Remains for People Living with HCU



Generally accepted therapeutic goal is to reduce total homocysteine (tHcy) levels but current treatment options rarely sustain reductions in tHcy



Significant challenges for patients to maintain compliance; periods of poor metabolic control have a cumulative deleterious effect



Patients struggle with severe dietary protein restrictions as they age; liberalized diet is amongst top needs



Inability to sustain reductions in Hcy results in life-long risk of thrombotic and cardiac events + cognitive impairment



Pegtibatinase (TVT-058) is an Investigational, Modified, Recombinant CBS Human Enzyme Therapy

Pegtibatinase (TVT-058) is a pegylated, modified recombinant truncated human enzyme, designed to address the underlying genetic cause of HCU

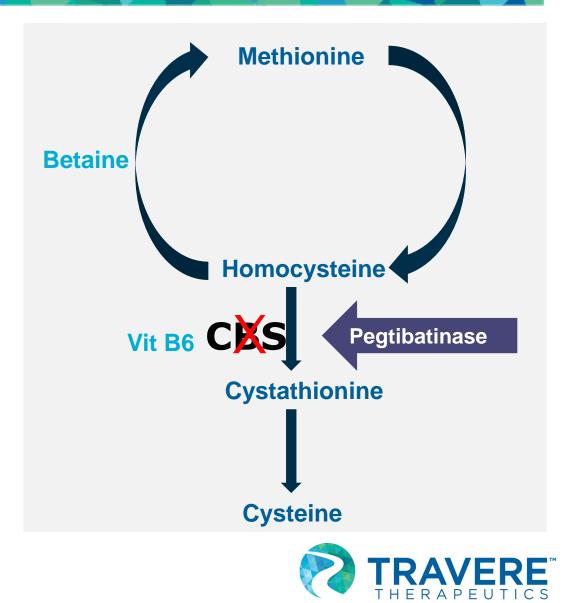
• Mechanism of action is pathology agnostic

Pegtibatinase is administered subcutaneously and designed to be active and stable in plasma unlike native CBS

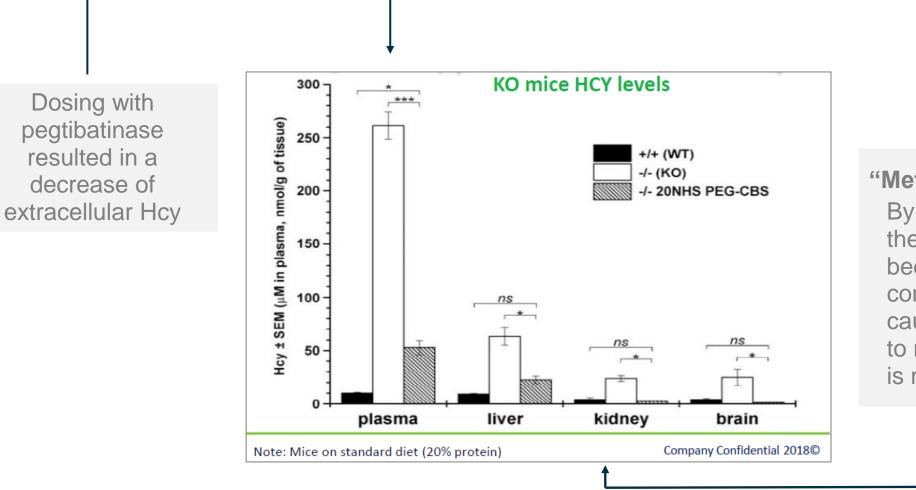
Designed to introduce the CBS enzyme into circulation and reduce intracellular and plasma Hcy levels

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 US and Europe.



Administration of Pegtibatinase Resulted in Up To 70-90% Reduction of Plasma and Tissue Hcy Levels in Mouse Models



"Metabolic Sink"

By lowering the Hcy levels in the blood, pegtibatinase has been shown to create a concentration gradient that causes excess Hcy in tissues to move to plasma, where it is metabolized



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Pegtibatinase is Advancing in the Phase 1/2 COMPOSE Study in HCU

Pegtibatinase is advancing in the Phase 1/2 COMPOSE Study – a double-blind, randomized, placebo-controlled study in patients with HCU

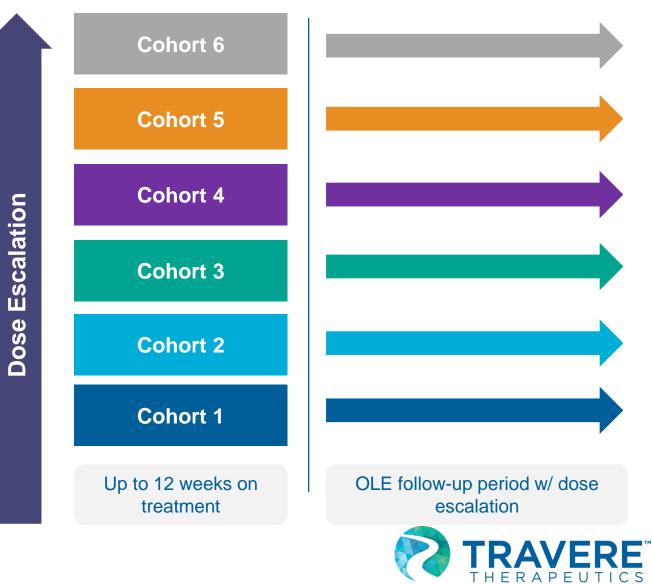
All 6 cohorts have completed enrollment. Cohorts were enrolled in a dose escalating fashion; following completion of each cohort, unblinded safety data are reviewed by DMC prior to activating next cohort

Primary endpoint

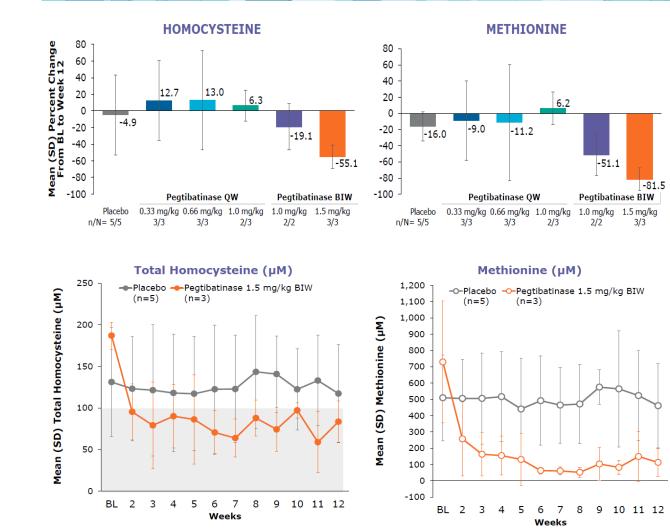
Incidence of treatment-emergent adverse events

Secondary endpoints

- Total plasma homocysteine (tHcy)
- Cognitive function changes
- Ocular assessment
- Bone mineral density
- Patient QoL scales: Neuro-QoL, EQ-5D & SF-36



Positive Topline Results from the Ongoing Phase 1/2 COMPOSE Study Provide Clinical Proof of Concept for Pegtibatinase



Shaded area indicates total homocysteine levels associated with improved long-term clinical outcomes (Morris 2017). BIW, twice weekly; BL, baseline; SD, standard deviation.

Levy H, *et al.* SIMD 2022 [poster presentation - Pegtibatinase, an Investigational Enzyme Replacement Therapy for the Treatment of Classical Homocystinuria: Initial Results From the Phase 1/2 COMPOSE Study]

- Pegtibatinase demonstrated dose-dependent reductions in tHcy during 12 weeks of treatment in the Phase 1/2 COMPOSE Study
- In the 1.5mg/kg BIW dose cohort, treatment with pegtibatinase resulted in a mean relative reduction from baseline of ~55% (n=3, mean baseline tHcy = 187.0 µmol), compared to a mean relative reduction from baseline of ~5% for all patients receiving placebo in the study (n=5, mean baseline tHcy = 131.1 µmol)
- In the 1.5mg/kg BIW dose cohort, treatment with pegtibatinase resulted in rapid and sustained reductions in tHcy, resulting in a maintenance of tHcy below a clinically meaningful threshold of 100 µmol from week 2 through week 12 of treatment
- In a dose-dependent manner, methionine levels were substantially reduced, and cystathionine levels were substantially elevated following treatment with pegtibatinase, suggesting that pegtibatinase acts in a manner similar to the native CBS enzyme
- The topline results suggested pegtibatinase had been generally well-tolerated



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Financial Snapshot

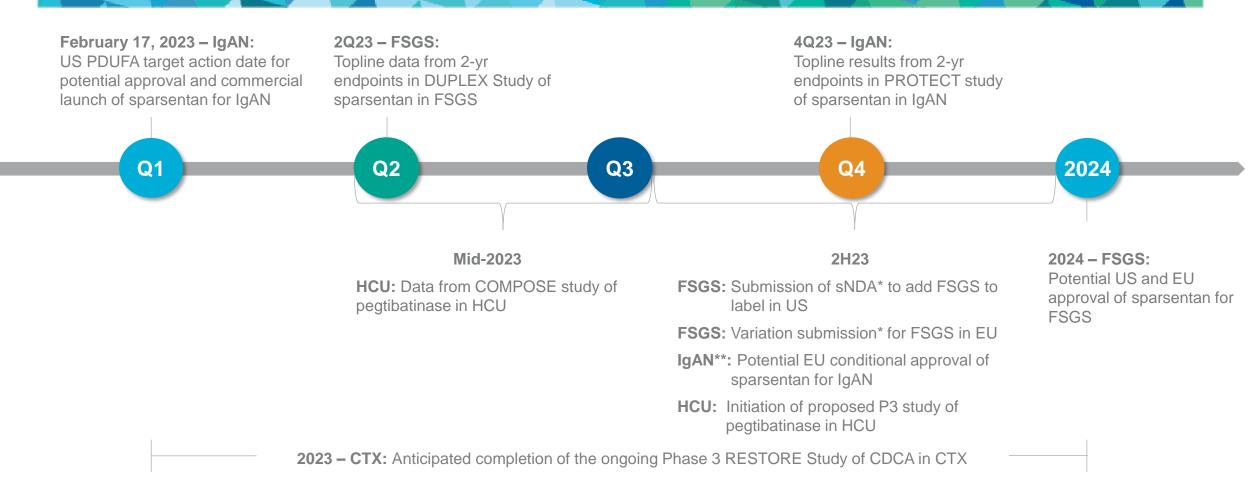
GAAP Reported Financials	FY2022*	3Q22	FY2021	FY2020	FY2019
Net Product Sales	~ \$201mm (~\$52mm Q422)	\$50.8mm	\$210.8mm	\$198.3mm	\$175.3mm
Operating Expenses	-	\$121.6mm	\$389.3mm	\$374.5mm	\$312.7mm
Operating Income / (Loss)	-	(\$68.1mm)	(\$161.8mm)	(\$176.2mm)	(\$137.4mm)
Net Income / (Loss)	-	(\$69.7mm)	(\$180.1mm)	(\$169.4mm)	(\$146.4mm)
Cash, Cash Equivalents and Marketable Debt Securities	~\$450mm	\$506.3mm	\$552.9mm	\$361.6mm	\$398.5mm

- Shares outstanding as of September 30, 2022: basic ~64mm, diluted ~76mm
- Convertible notes: \$69 million due 2025, \$316 million due March 2029



*Based upon preliminary, unaudited 2022 financial data

Upcoming Milestones in 2023 to Further Position Travere as a Leader in the Rare Disease Community



Furthering our mission of delivering life-changing therapies to people living with rare disease



*Pending supportive data and US/EU approval of sparsentan for IgAN ** In partnership with European collaborator CSL Vifor



In Rare For Life.

