Travere Therapeutics Corporate Overview

January 2021

Forward-Looking Statements

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.

TRAVERE™ THERAPEUTICS

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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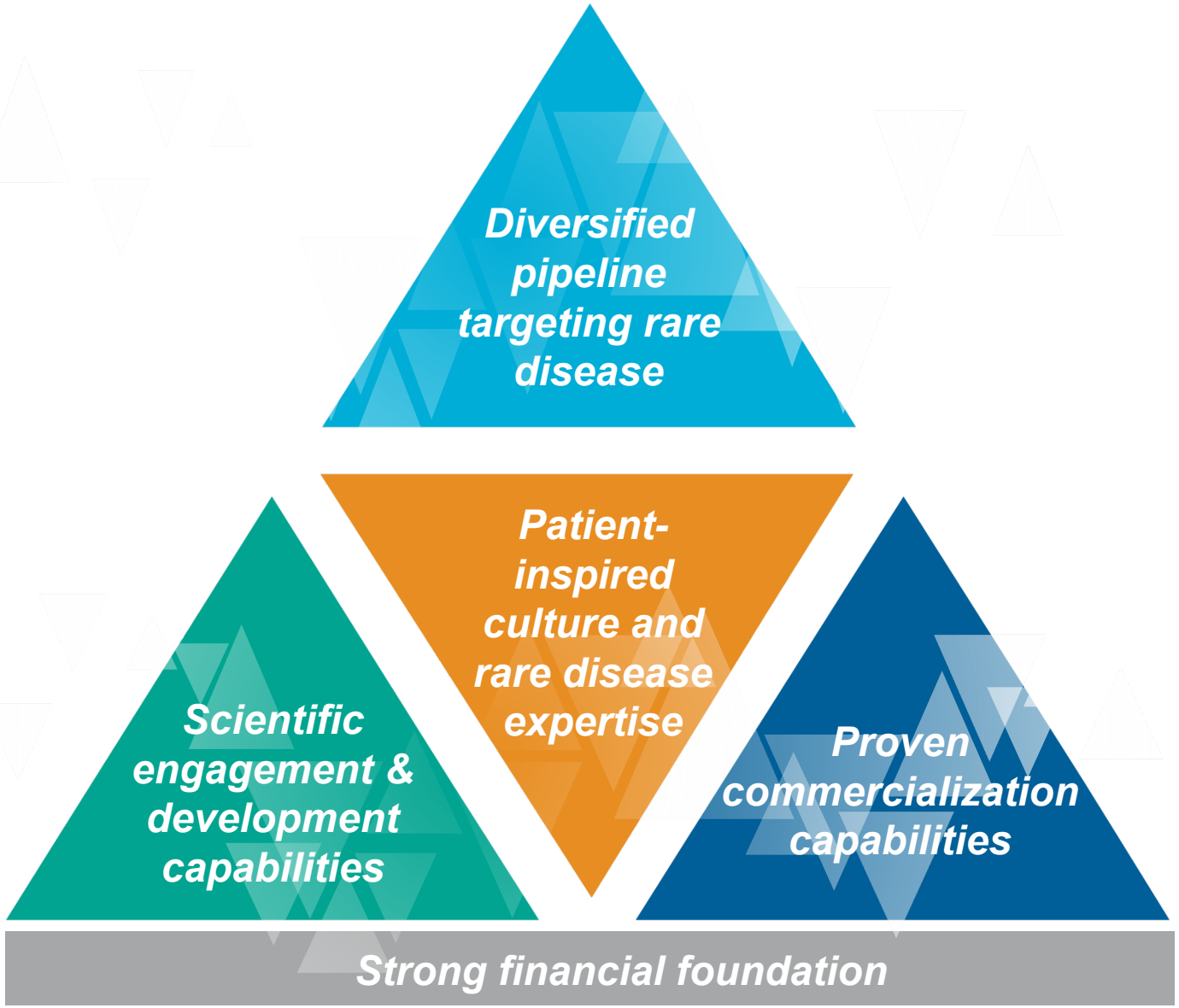
Key Strengths of Travere Therapeutics



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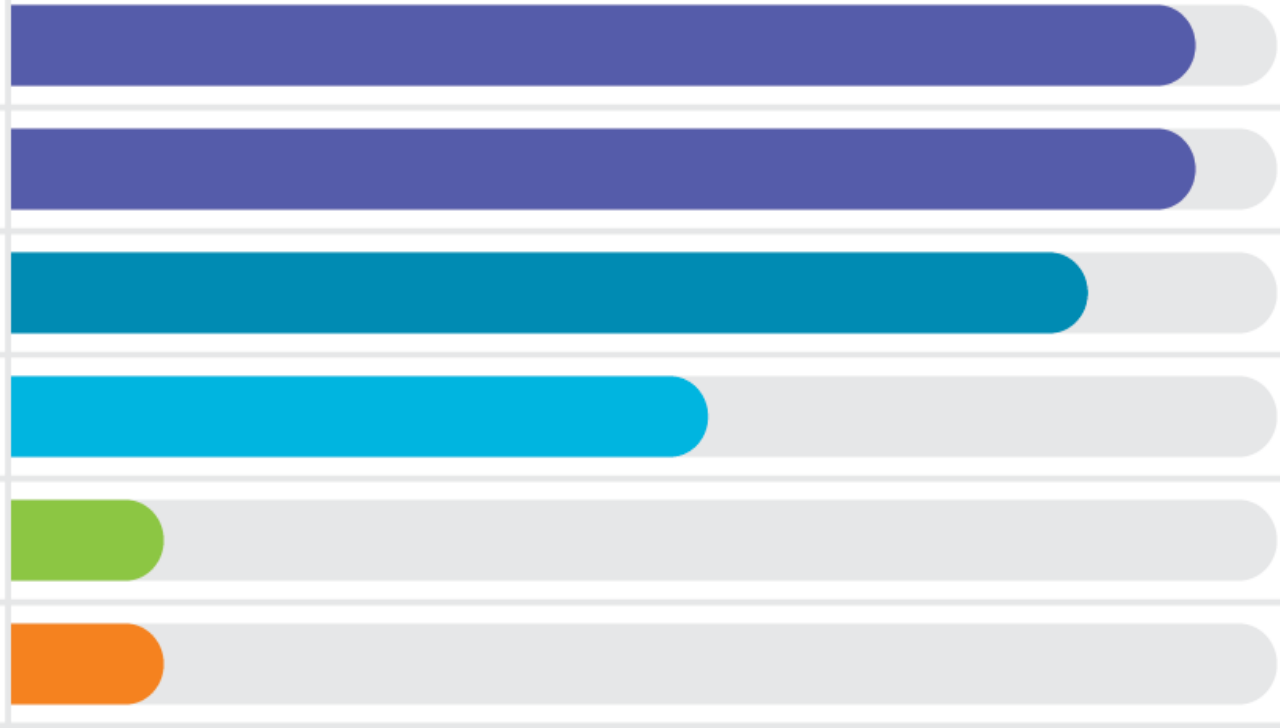
Pipeline of Potential First-in-Class Programs Targeting Rare Diseases

PROGRAM	THERAPEUTIC AREA	PRECLINICAL	PHASE 1	PHASE 2	PHASE 3
Sparsentan	Focal Segmental Glomerulosclerosis (FSGS)				
Sparsentan	IgA Nephropathy (IgAN)				
CDCA*	Cerebrotendinous Xanthomatosis (CTX)				
TVT-058**	Classical Homocystinuria (HCU)				
NGLY1 Collaboration	NGLY1 Deficiency				
ALGS Collaboration	Alagille Syndrome (ALGS)				



*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.
**TVT-058 is currently in a Phase 1/2 clinical study.

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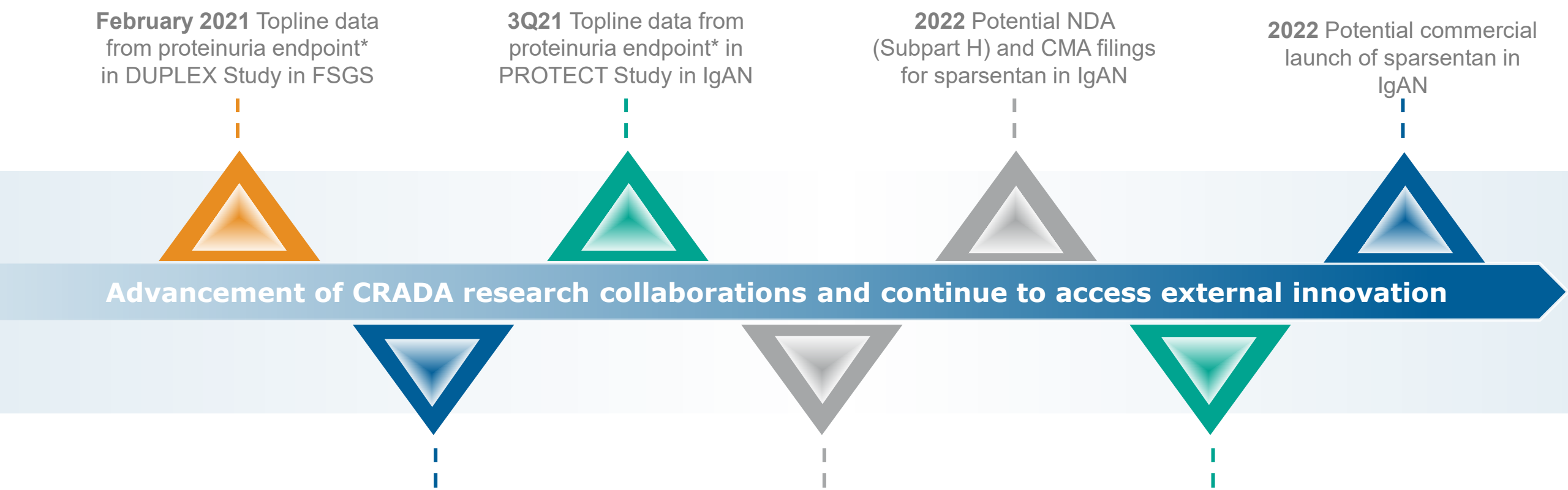
Path to Potential Breakthrough Growth for Travere Therapeutics



*Interim endpoint; confirmatory endpoint is slope of eGFR

Sparsentan - A Potential First-in-Class Molecule for FSGS and IgAN

Progressive Kidney Disease Has A Devastating Impact on Patients, Caretakers and Society

The devastating impact of progressive kidney disease:

- Kidney disease is one of the leading causes of death
- About 1-in-4 patients die within first year of dialysis
- High mortality rate prior to ESKD due to kidney related vascular events

Progressive kidney disease has a dramatic impact on healthcare cost:

- CKD accounts for ~20% in traditional Medicare spending upwards of $114B/year
- Total Medicare expenditures $49.2B for beneficiaries with ESKD in the US
- >125,000 patients start dialysis each year in the US; this number is growing

Results in disproportional number of patients with FSGS and IgAN in ESKD due to progressive nature of disease

ESKD = End Stage Kidney Disease
Xu et. al, NCHS Data Brief No. 355, January 2020; USRDS 2020 Annual Report; HHS Press Office, 2019;

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The Burden of FSGS and IgA Nephropathy

FSGS

- Primary FSGS generally affects patients in their mid-forties to fifties
- High proteinuria levels in (sub)nephrotic range is hallmark of disease
- 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

- Growing incidence and prevalence

IgAN

- Most commonly reported primary glomerulonephritis
- Affects young adults, often leading to dialysis or transplant in their 40s or early 50s
- ~30-40% of patients have a progressive course to ESKD
- The prognosis is worse for those with persistent proteinuria >1gr/day

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

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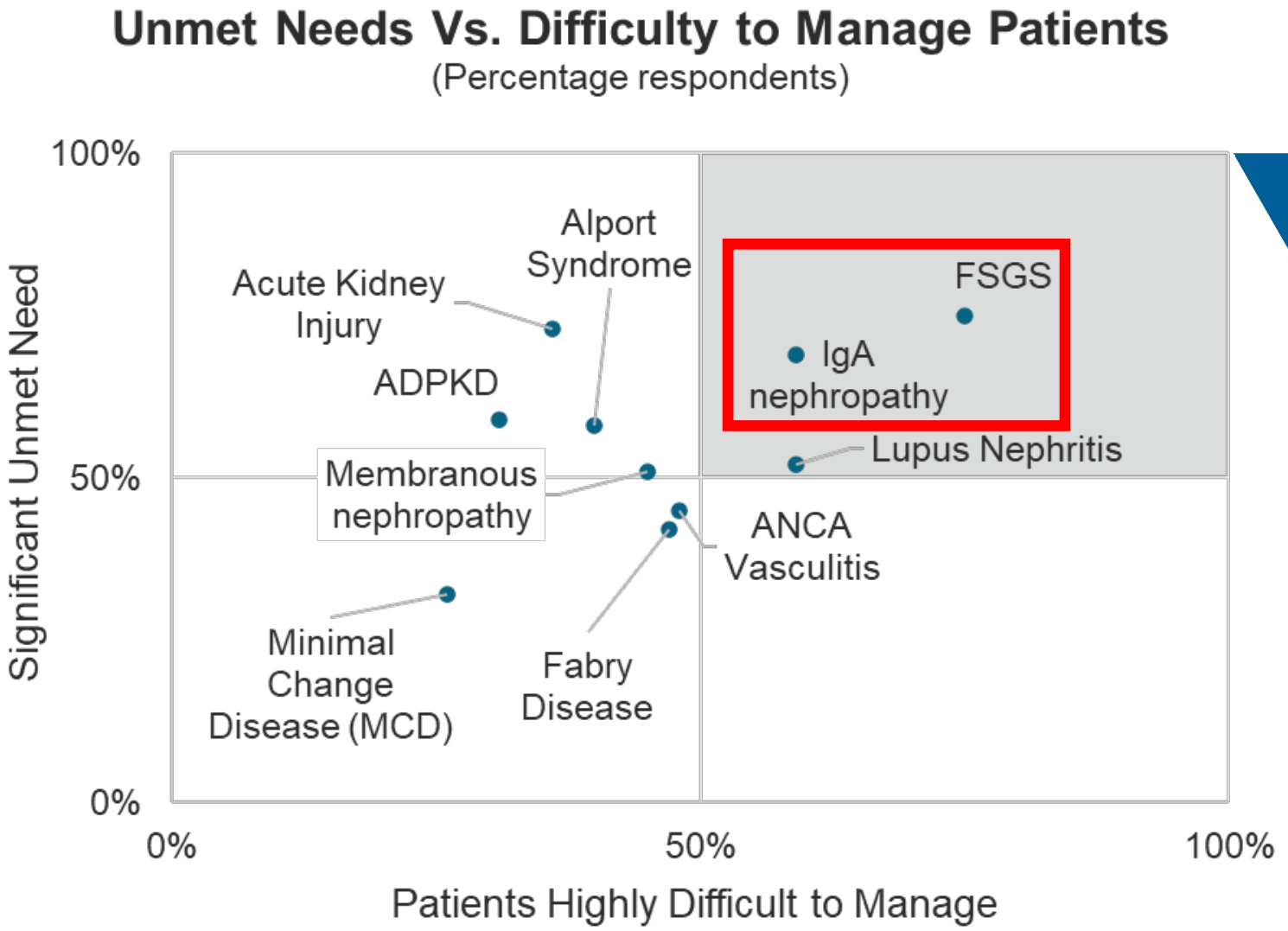
Given no FDA or EMA Approved Medicines Indicated for FSGS or IgAN, Nephrologists Rate FSGS & IgAN as Most Challenging to Manage



Surveyed nephrologists believe *only 8%* of their FSGS patients are "optimally managed"

Surveyed nephrologists: *only 19%* of IgAN patients are "optimally managed"

Source: Independent market research, data on file

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FSGS and IgAN Share Common Renal Injury Pathways

Kidney injury in FSGS

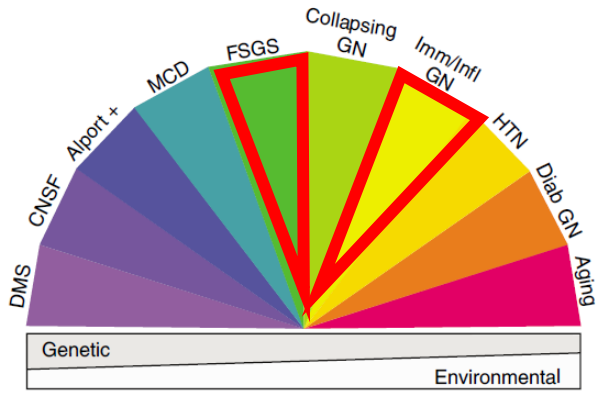
Factors affecting podocyte structural integrity

Podocyte Pathology

Proteinuria

Glomerulosclerosis

Tubulointerstitial Fibrosis



Kidney Failure = Transplant/ Dialysis

Kidney injury in IgAN

Formation of glomerular deposition of immune complexes with hypogalactosylated IgA

Mesangial Cell Activation

Glomerular Inflammation

Proteinuria

Hematuria

Glomerulosclerosis

Tubulointerstitial Fibrosis

Source: Wiggins, Kidney International (2007)

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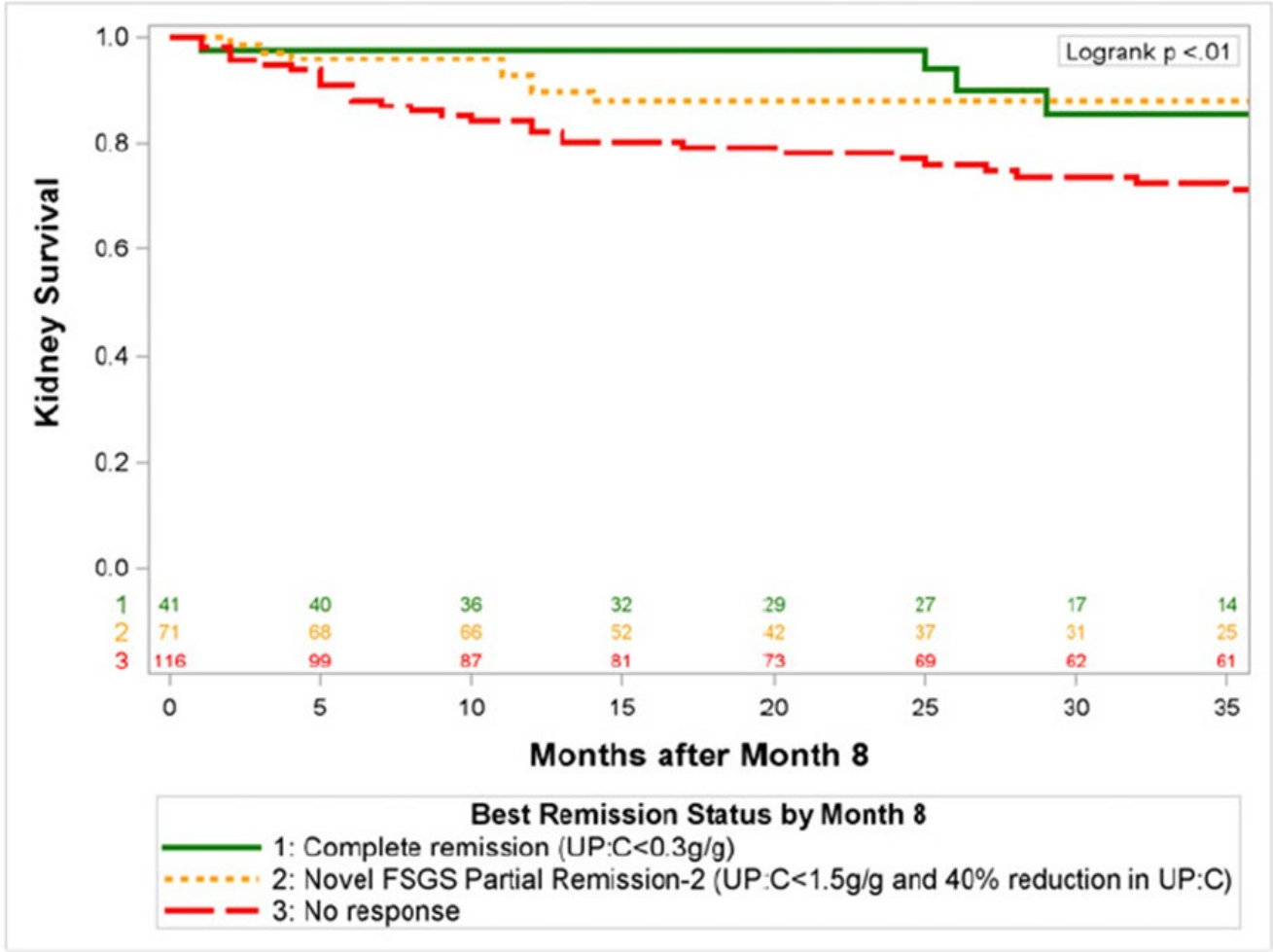
Significantly Reducing Proteinuria in FSGS Has Been Shown to Correlate With Improved Kidney Survival

- Data from five independent cohorts totaling 466 patients with primary FSGS were analyzed and established 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



UP/C = urinary protein-to-creatinine ratio.

Troost JP, *et al*. *Clin J Am Soc Nephrol* 2018; **13:**414–421.

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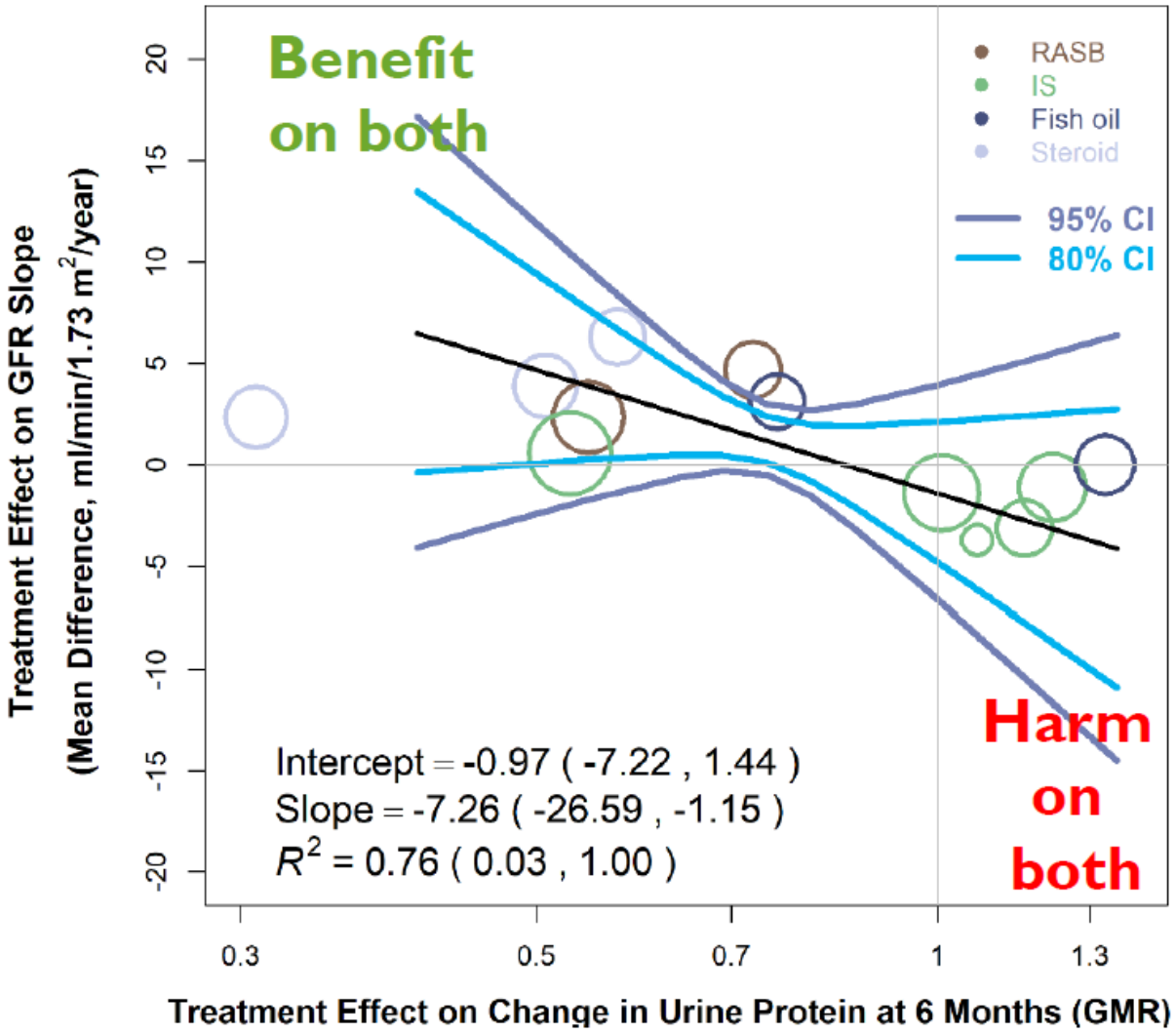
Studies Suggested Greater Reduction in Proteinuria Also Resulted in Slower Progression of IgAN



- **The most widely recognized and well-studied risk factor for progression to ESRD in IgAN is proteinuria[1]**
- **Recent analyses of trial-level data from ~1,000 patients with IgAN show associations between treatment effect on change in proteinuria and rate of change in GFR[2]**
  - Predicted treatment effects on GFR slope were strongest for larger treatment effects on change in UP/C

1. Thompson et al. CJASN 2019, 2. Inker on behalf of CKD-EPI et al. ASN Kidney Week 2019;

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Sparsentan is a Potential First-in-Class Molecule Designed to Selectively Inhibit the Endothelin Receptor and Angiotensin II Receptor

- Sparsentan is an investigational product candidate designed to inhibit both endothelin receptor type A (ET_A) and angiotensin II receptor type 1 (AT_1)[1-3]
- Distinct selectivity profile: high affinity selective antagonist at both the ET_A and AT_1 receptors; highly selective ET_A/ET_B
- Has shown nephroprotective properties across pre-clinical studies in both FSGS and IgAN
- Sparsentan has been granted orphan drug designation for the treatment of FSGS by the FDA and European Commission

Source: 1. Kowala MC, et al. *J Pharmacol Exp Ther*. 2004;309:275–284., 2. Komers R, et al. *Am J Physiol Regul Integr Comp Physiol.* 2016: 310 (10): R877-884., 3. Benigni A, et al. *Pediatr Nephrol* 2020. https://doi.org/10.1007/s00467-020-04518-2

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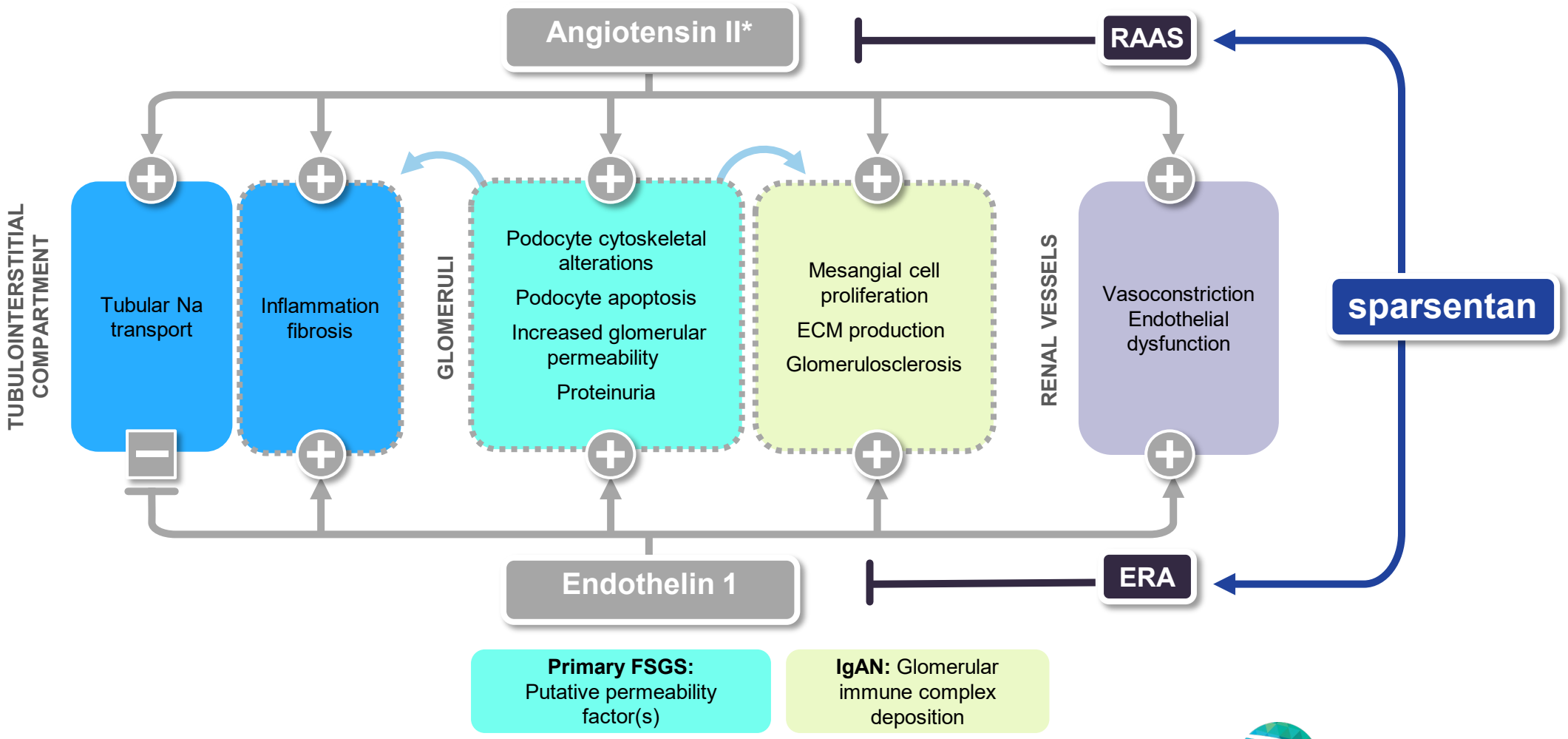
Sparsentan Acts by Inhibiting the Signaling Pathways Present in FSGS and IgAN



*Also applies to aldosterone. Image adapted from Komers R. et al. *Am J Physiol Regul Integr Comp Physiol.* 2016;310:R877–R884.
ECM, extracellular matrix; ERA, endothelin receptor antagonist; RAAS, renin-angiotensin-aldosterone system.

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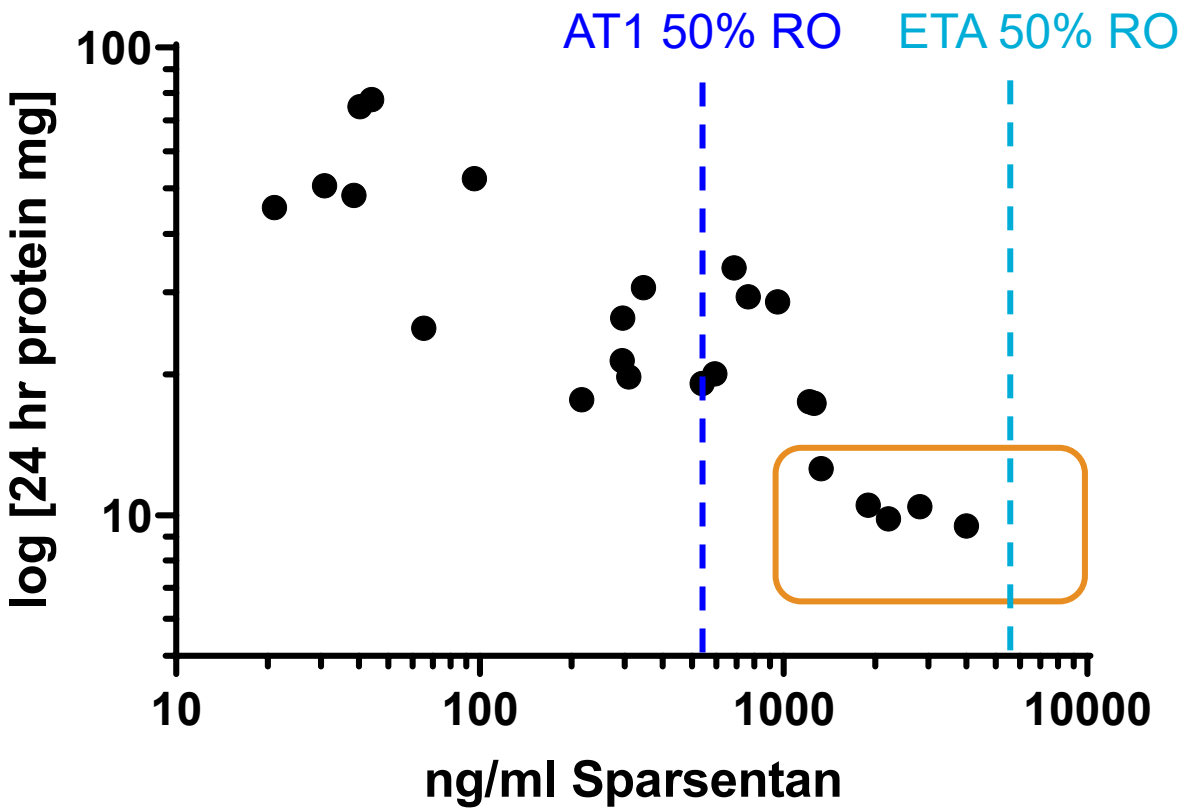
Inhibition of Both AT_1 and ET_a Together with Sparsentan Resulted in Further Proteinuria Reduction in Rat Models



- In vitro receptor binding in Rats
  - AT1 Ki = 11 nM
  - ETA Ki = 110 nM
- Sparsentan circulates bound to protein, hence 10x Ki concentration results in ~50% receptor occupancy
- **Maximum efficacy is observed with significant inhibition of ETA receptors**

Source: RE-021-0045; RE-021-0057, RE-021-Report003-2018-PHARM; RE-021-Report007-2018-BIOA

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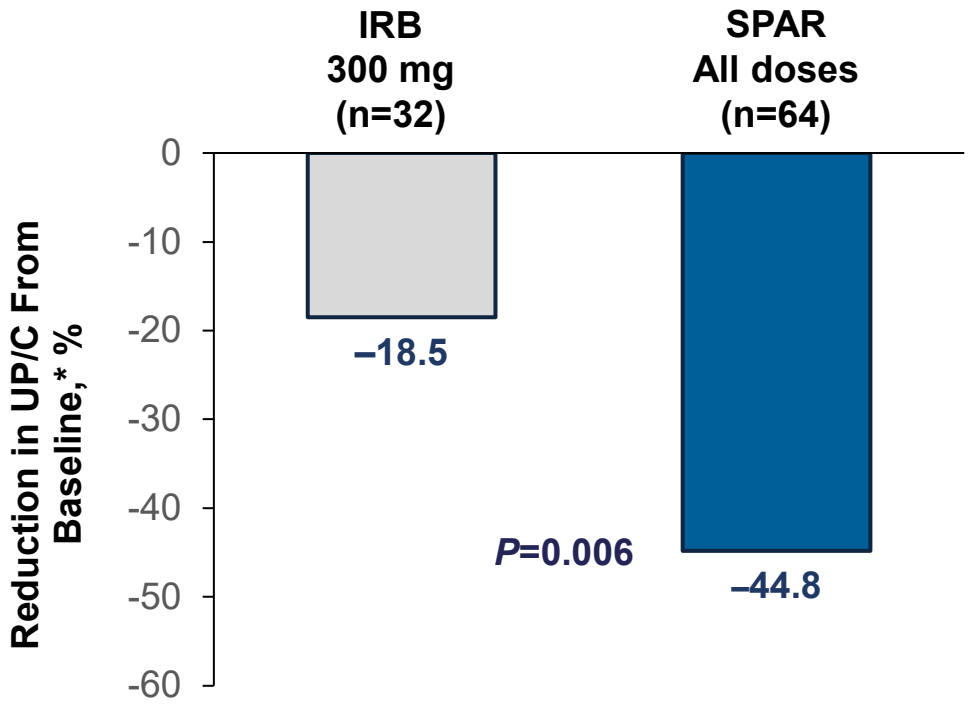
Phase 2 DUET Study: Overall Sparsentan Treatment Group Met Primary Endpoint; More Than Doubled Reduction of Proteinuria vs. Irbesartan



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

TEAE	Patients with TEAEs During the Double-Blind Period, %	
	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

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

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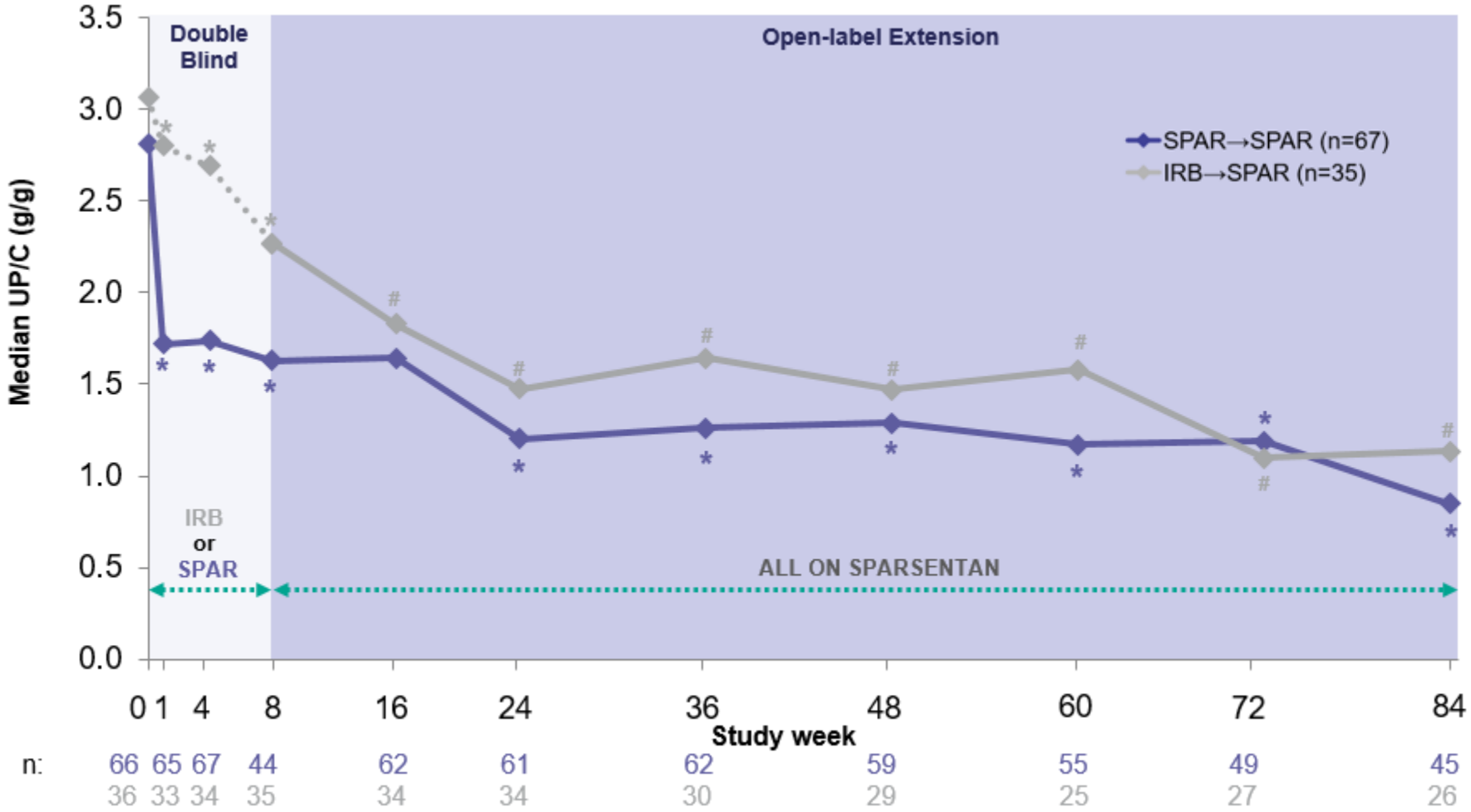
Phase 2 DUET Study: Sustained Long-Term Proteinuria Reduction over 84 Weeks



95% confidence interval of the mean change from baseline (* Week 0; # Week 8) excludes 0.
Based on the full analysis set. UP/C based on first morning void.
UP/C = urinary protein-to-creatinine ratio; OLE = open-label extension

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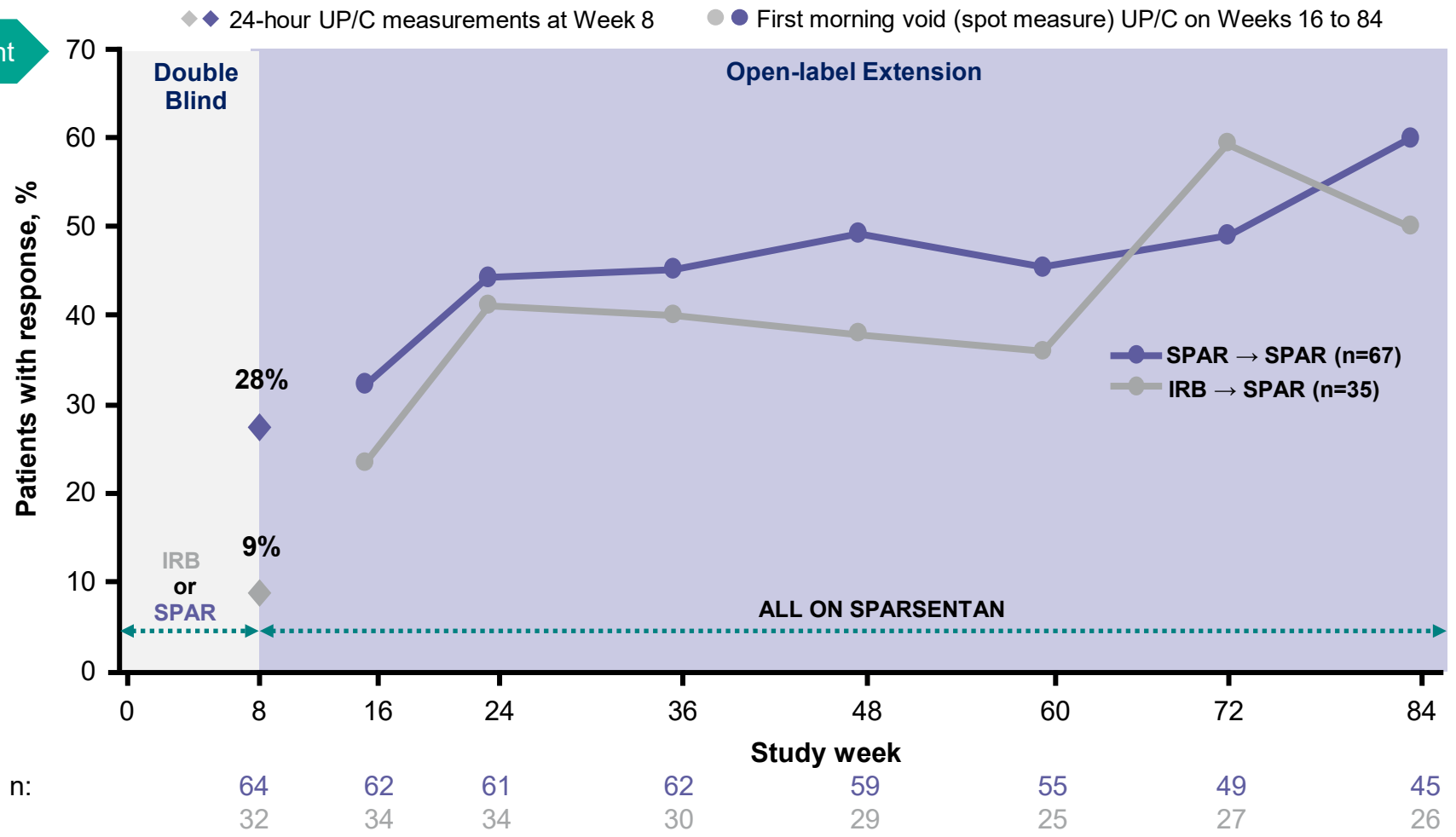
Phase 2 DUET Study: Promising Proportion of Patients Achieved FSGS Partial Remission Endpoint (FPRE)

Secondary Endpoint



p<0.04

An increasing proportion of patients achieved FPRE with ongoing sparsentan treatment in the OLE

FPRE is defined as UP/C ≤1.5 g/g and >40% reduction in UP/C from baseline. Baseline in the double-blind period defined as Week 0; baseline for the open-label period defined as last observation before start of open-label sparsentan treatment (i.e.Week 8). Data for Week 8 are based on the EES. Data for Weeks 16 to 84 are based on the full analysis set. EES = efficacy evaluable set; FPRE = FSGS partial remission endpoint; IRB:SPAR = patients randomized to irbesartan who then transitioned to sparsentan in the OLE; OLE = open-label extension; SPAR:SPAR = patients randomized to sparsentan who also received sparsentan in the OLE; UP/C = urinary protein-to-creatinine ratio. 1. Hogan J, et al. *J Am Soc Nephrol* 2018; 29:61 (Abstract FR-OR087); 2. Trachtman H, et al. *J Am Soc Nephrol* 2018; 29:2745–2754; DUET ClinicalTrials.gov Identifier: NCT01613118.

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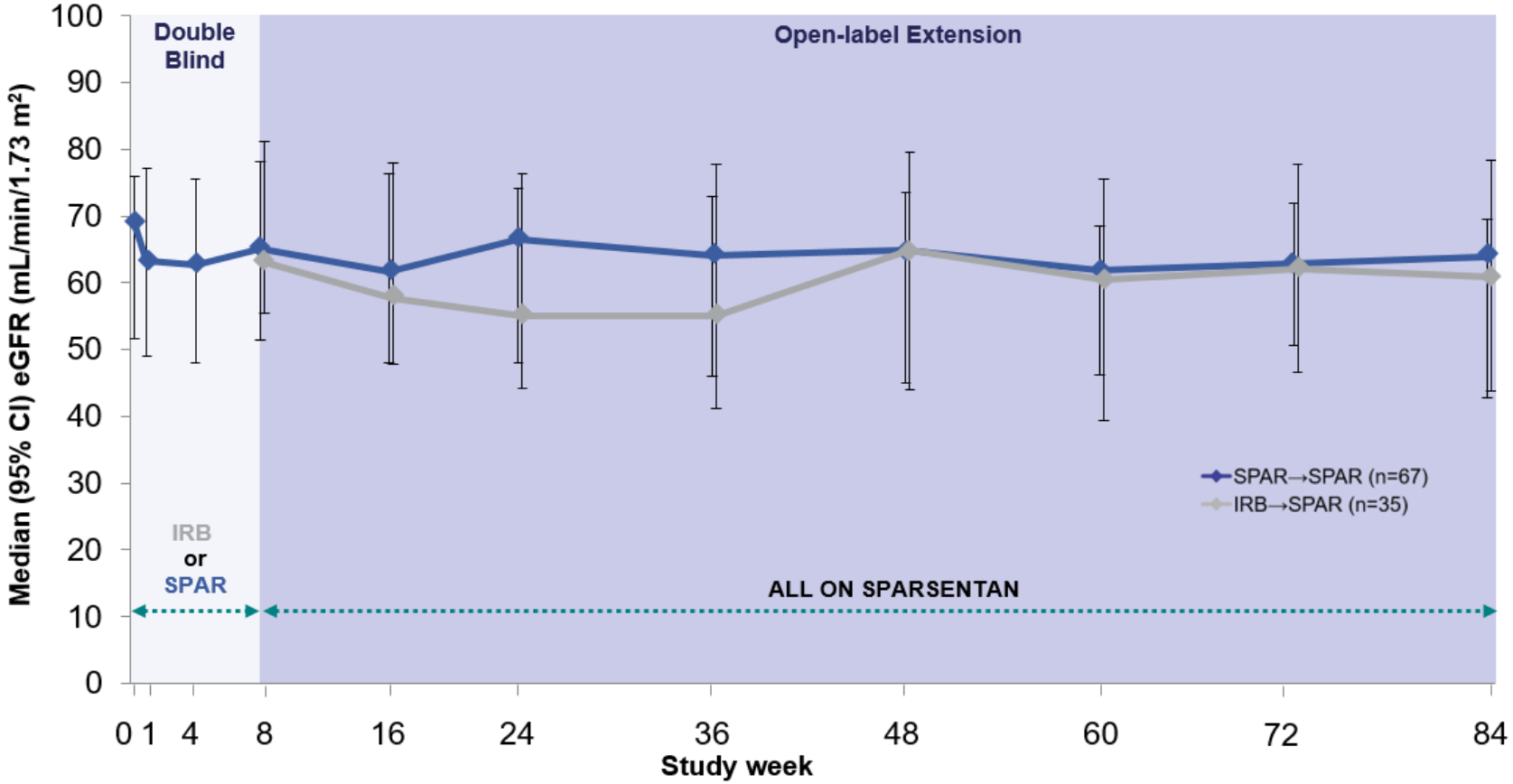
Phase 2 DUET Study: eGFR Remained Stable in Sparsentan-Treated Patients Over 84 Weeks



Based on the full analysis set. 95% CIs were calculated for the median value at each time point. Note: There were 9 study withdrawals due to renal causes during the follow-up period, including 1 case of end-stage renal disease.

CI = confidence interval; eGFR = estimated glomerular filtration rate; IRB:SPAR = patients randomized to irbesartan who then transitioned to sparsentan in the OLE; OLE = open-label extension; SPAR:SPAR = patients randomized to sparsentan who also received sparsentan in the OLE.

Hogan J, et al. Long-Term Effects of Sparsentan, a Dual Angiotensin and Endothelin Receptor Antagonist in Primary FSGS: Interim 84-Week Analysis of the DUET Trial (Abstract FR-OR087). *J Am Soc Nephrol*. 2018;29:61. Accessed at https://www.asn-online.org/education/kidneyweek/archives

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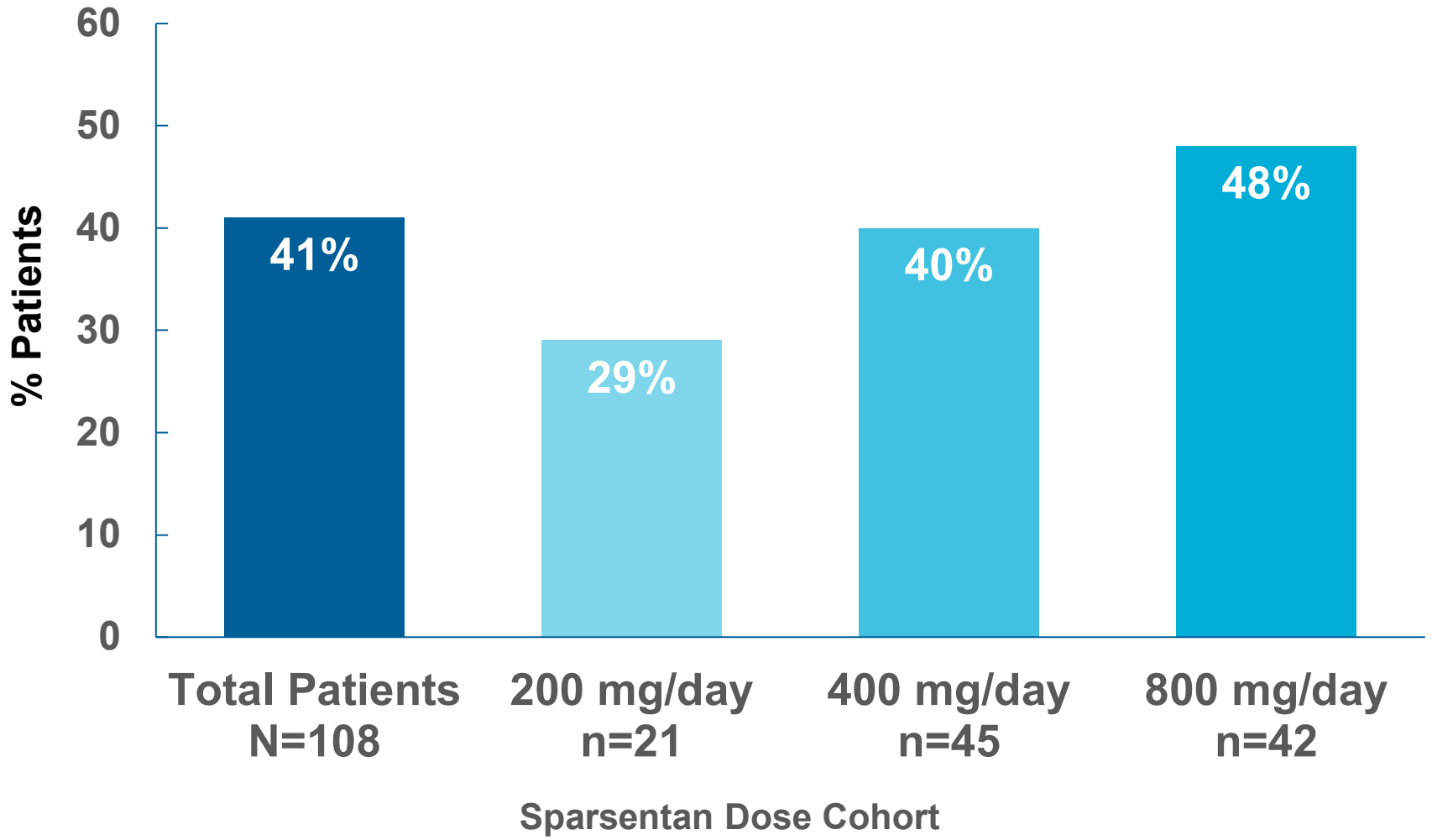
Phase 2 DUET Study: Post hoc analysis – A High Percentage of Patients Achieved UP/C ≤0.3 g/g at Any Visit in a Dose-related Manner



Hogan J, *et al*. ASN 2020 [oral presentation]
109 patients were randomized in DUET; 108 patients received at least one dose of sparsentan and were eligible for evaluation of UP/C while on sparsentan.

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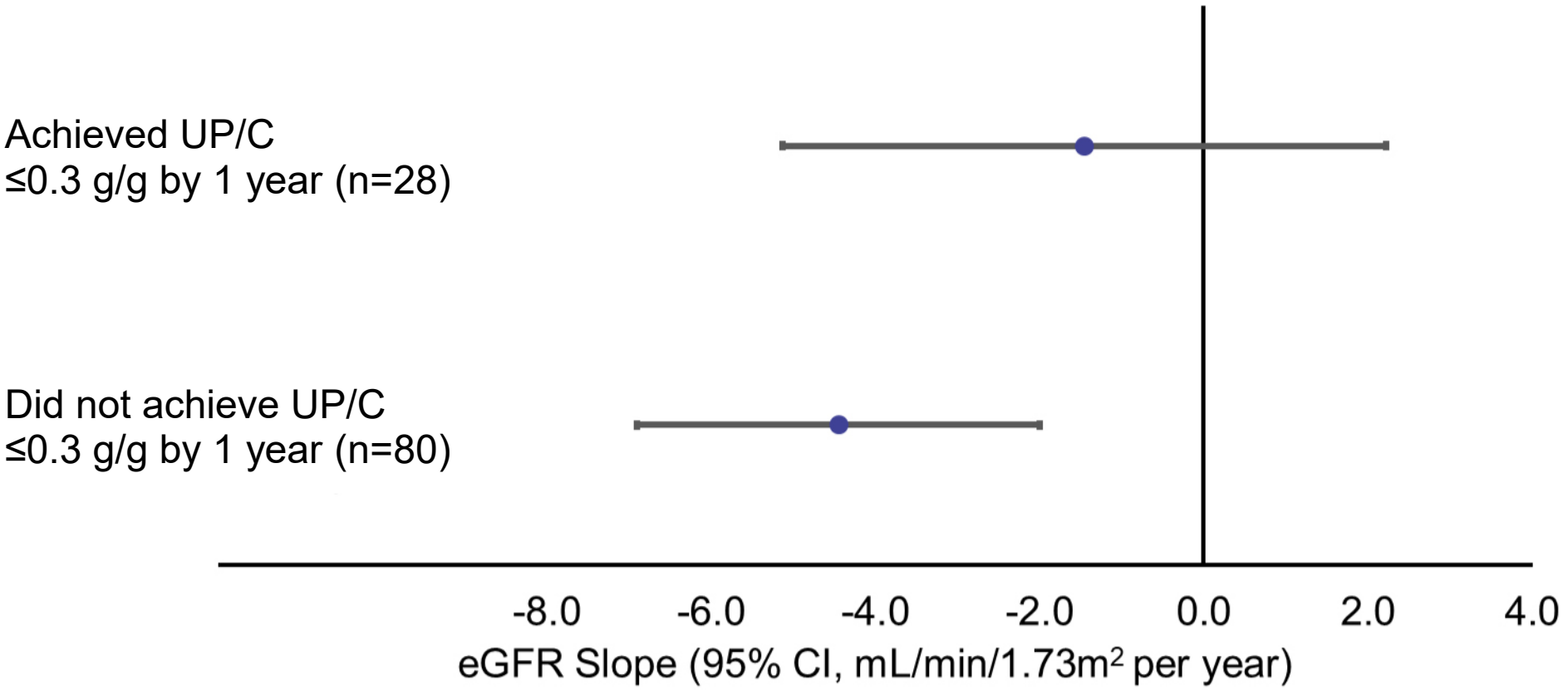
Achieving Complete Remission With Sparsentan in the First Year was Associated With Slower eGFR Decline Over Two Years



Hogan J, *et al*. ASN 2020 [oral presentation]

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A Closer Look at Edema in DUET

TEAEs with Incidence >5%	Patients n (%)			
	Irbesartan		Sparsentan	
	All	Drug Related	All	Drug Related
Headache	7 (19.4)	1 (2.8)	14 (19.2)	**9 (12.3)**
Hypotension/orthostatic hypotension	3 (8.3)	3 (8.3)	12 (16.4)	**11 (15.1)**
Dizziness	4 (11.1)	3 (8.3)	10 (13.7)	**8 (11.0)**
Edema/edema peripheral	1 (2.8)	0	9 (12.3)	**2 (2.7)**
Nausea	3 (8.3)	0	9 (12.3)	**6 (8.2)**
Diarrhea	1 (2.8)	0	6 (8.2)	**2 (2.7)**
Vomiting	1 (2.8)	0	6 (8.2)	**4 (5.5)**
Upper abdominal pain	2 (5.6)	0	4 (5.5)	**2 (2.7)**
Cough	2 (5.6)	0	3 (4.1)	**0**
Fatigue	4 (11.1)	1 (2.8)	3 (4.1)	**2 (2.7)**
Nasal congestion	4 (11.1)	0	2 (2.7)	**0**
Upper respiratory tract infection	2 (5.6)	0	2 (2.7)	**0**
Hyperkalemia	2 (5.6)	2 (5.6)	1 (1.4)	**1 (1.4)**
Muscle spasms	2 (5.6)	0	0	**0**

- Edema is common in people living with FSGS
- Adverse event of interest given potential ERA class effect
- Treatment related edema events in DUET were well managed with diuretics
- OLE findings are consistent with double blind portion of study

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Leveraging Our Learnings in DUET to Design Pivotal DUPLEX Study

UP/C inclusion criteria from 1.0g/g to 1.5g/g to allow all patients to be eligible for FPRE endpoint

Evaluating the FPRE endpoint after 36-weeks of treatment

Target dose of 800mg of sparsentan with two-week dose titration schedule

Confirmatory eGFR endpoint after 108 weeks of treatment

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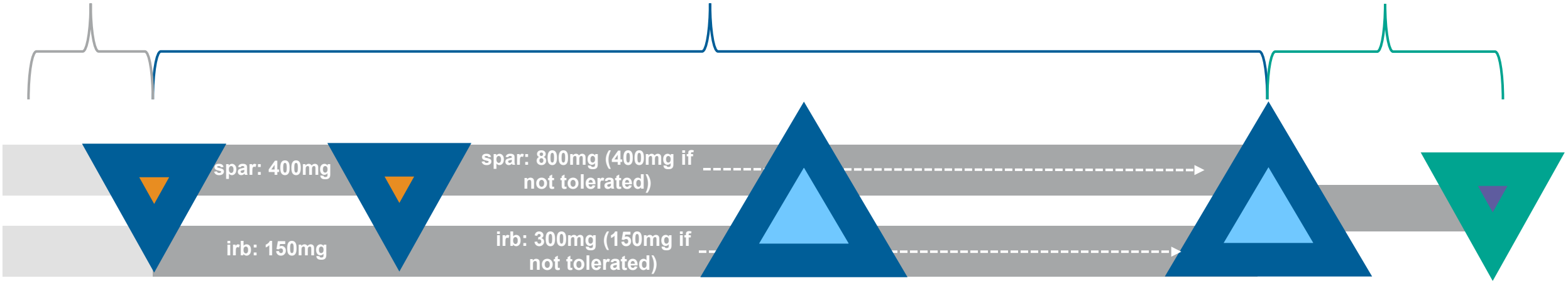
Phase 3 DUPLEX Study Designed to Support NDA & CMA Submissions for FSGS

Washout
2 weeks
no RAAS inhibitors

Double-Blind Treatment Period
108 Weeks

Cessation of Treatment
4 Weeks
No sparsentan or irbesartan



DAY 1
- Baseline
- UP/C, eGFR
- Randomize 1:1
- Initial dose

WEEK 2
Achieve target dose of 800mg sparsentan or 300mg irbesartan

WEEK 36
Interim analysis of FPRE endpoint
~190 patients

WEEK 108
End of treatment, primary endpoint of change in slope of eGFR endpoint from ~300 subjects

WEEK 112
End of study

- **More than 90% powered to detect difference in FPRE response**
- **90% powered to detect low single digit difference in eGFR slope b/t sparsentan and irbesartan arms after 108wks**

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DUPLEX Enrollment Complete; On Track for Topline Interim Data in February 2021

- DUPLEX achieved completion of patient enrollment in November 2020
- DUPLEX has completed four independent data monitoring committee assessments of safety with recommendations for study to proceed as planned
- Sample size reassessment completed - no increase in sample size recommended
- Initial baseline characteristics supporting the interim assessment are in-line with expectations and the DUET/NEPTUNE databases used to design DUPLEX
  - No clinically meaningful difference in the baseline distribution of UP/C compared to DUET
  - Blinded baseline range of eGFR is consistent with DUET
  - Blinded variability in-line with expectations
- Topline data from interim proteinuria analysis are anticipated in February 2021

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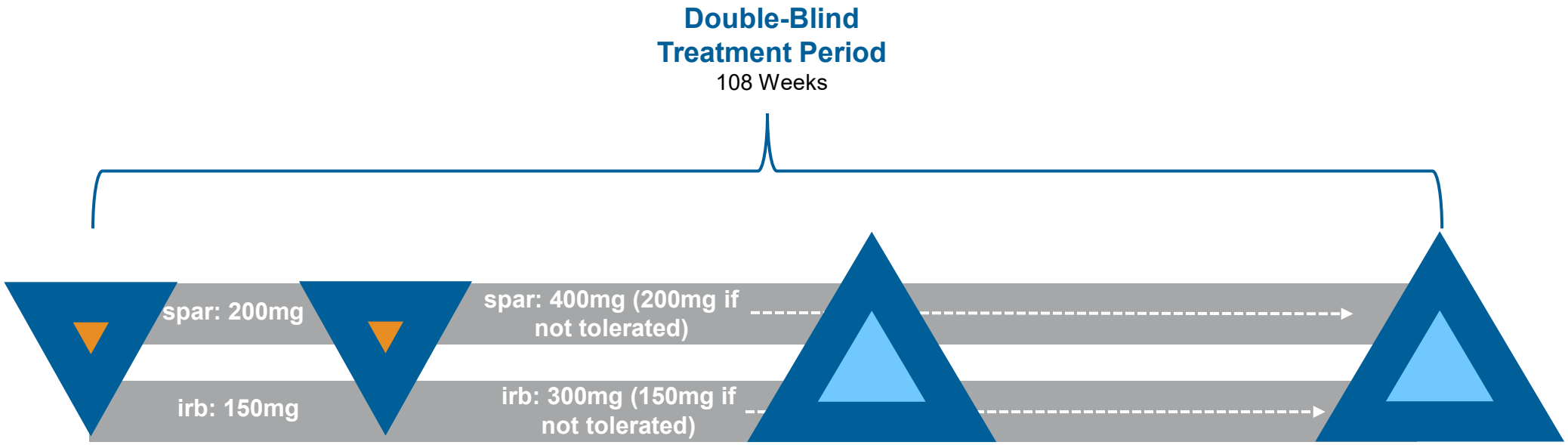
Leveraging Learnings from DUET, Historical ET_A Inhibition and Trial-Level Data to Design PROTECT in IgAN



DAY 1
- Discontinue prior ACE and/or ARB
- Randomize 1:1
- Baseline UP/C
- Initial dose

WEEK 2
Achieve target dose of 400mg sparsentan or 300mg irbesartan

WEEK 36
Primary endpoint of change from baseline UP/C
~280 patients

WEEK 58
Secondary endpoints rate of change in eGFR
~380 subjects

WEEK 110
End of treatment, confirmatory analysis of rate of change in eGFR
~380 subjects

- **90% powered to detect a 30% difference in proteinuria between sparsentan and irbesartan arms**

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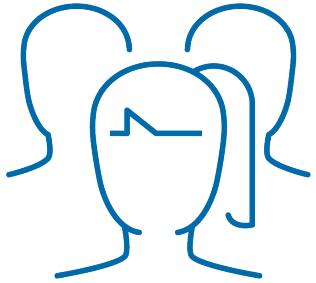
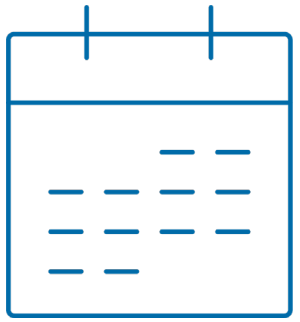
PROTECT On Course for Topline Proteinuria Data in 3Q21

PROTECT achieved enrollment of the first 280 patients to support the interim assessment of UP/C reduction after 36 weeks



PROTECT continues to enroll towards a total sample size of ~380 patients with IgAN – completion of enrollment is expected in 2021



Topline data from the 36-week interim proteinuria analyses are expected in 3Q21; if successful, expected to support accelerated approval submissions in the US and EU

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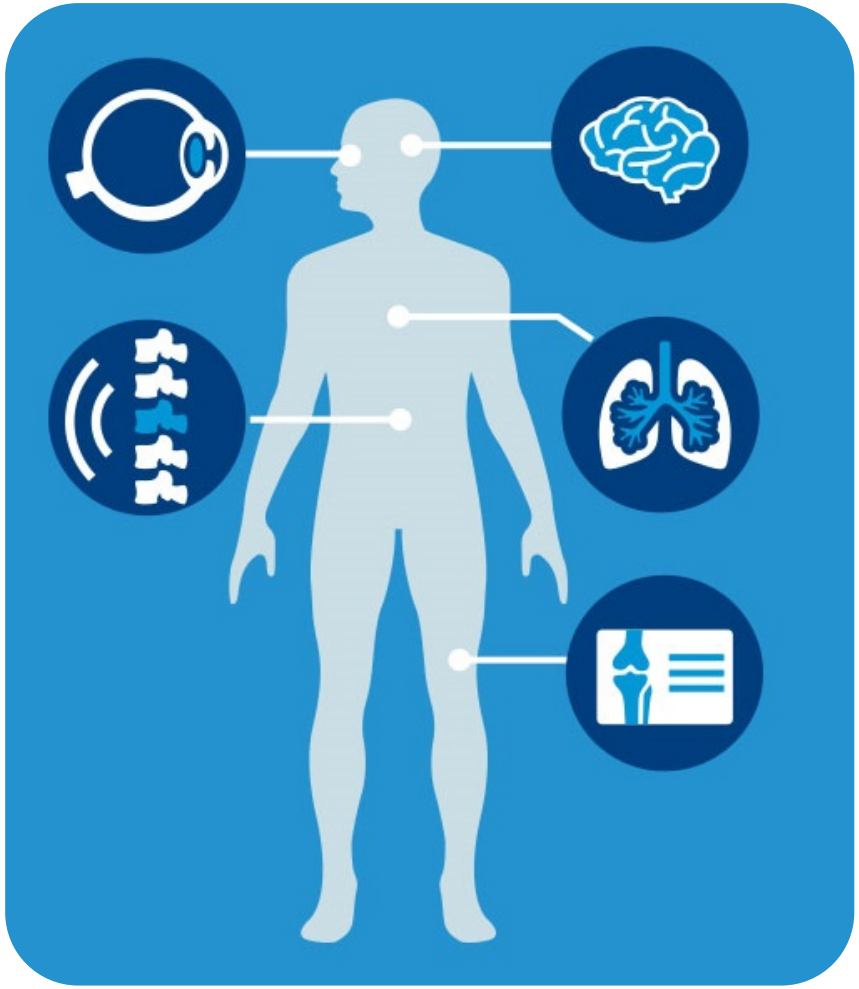
TVT-058 – The First Potential Disease Modifying Therapy for Classical Homocystinuria (HCU)

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**

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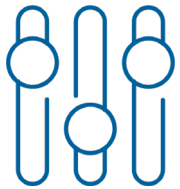
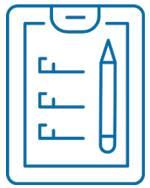
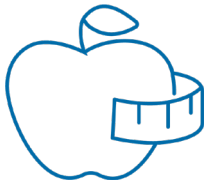
With Largely Ineffective Treatment Options, a Significant Unmet Need Remains for People Living with HCU



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



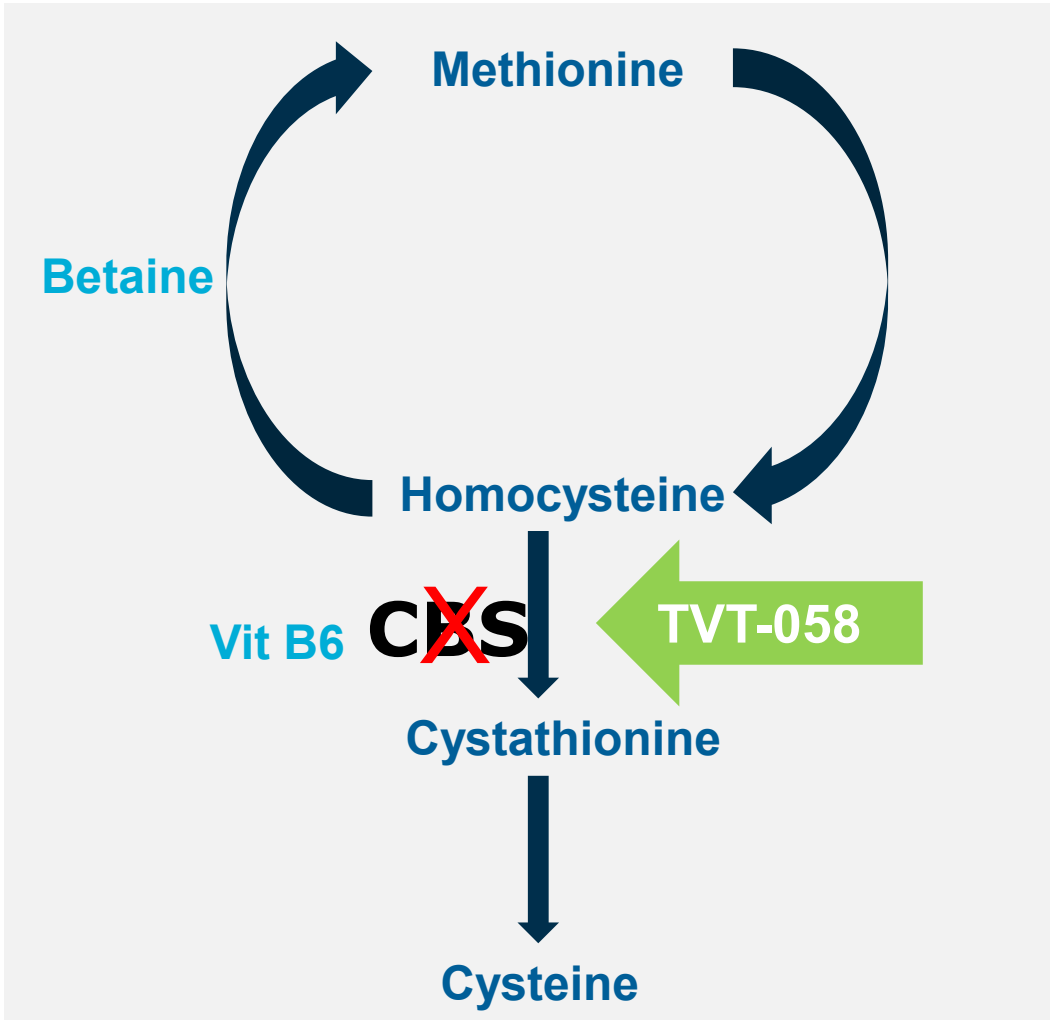
Patients struggle with severe dietary protein restrictions as they age; liberalized diet is amongst top needs

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

TRAVERE™ THERAPEUTICS

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

- **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
- **TVT-058 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**
- **TVT-058 has been granted multiple regulatory designations:**
  - FDA Rare Pediatric Disease designation
  - FDA Fast Track designation
  - Orphan Drug designation in the US and Europe.



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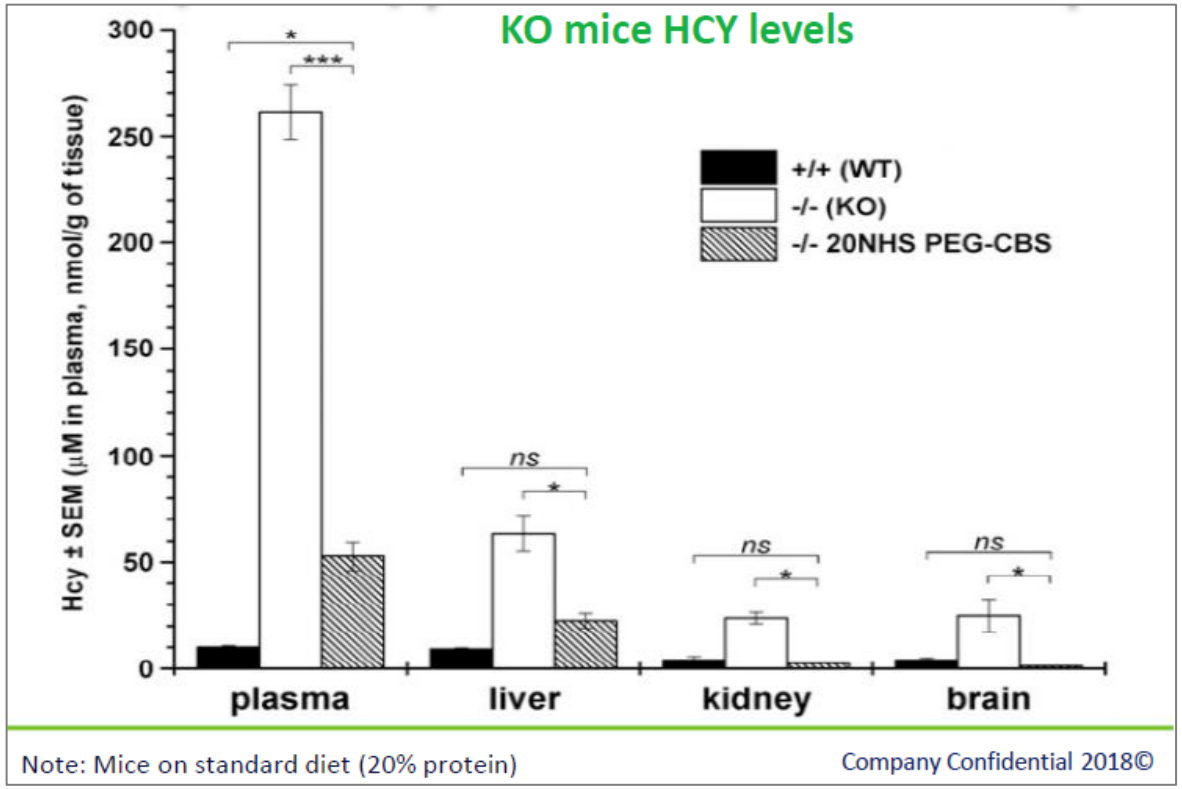
Administration of TVT-058 Resulted in Up To 70-90% Reduction of Plasma and Tissue Hcy Levels in Mouse Models

Dosing with TVT-058 resulted in a decrease of extracellular Hcy

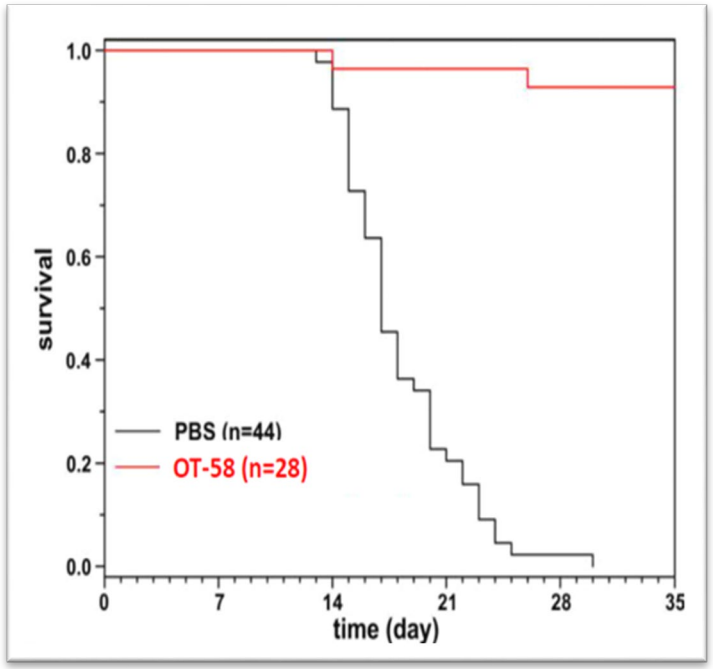


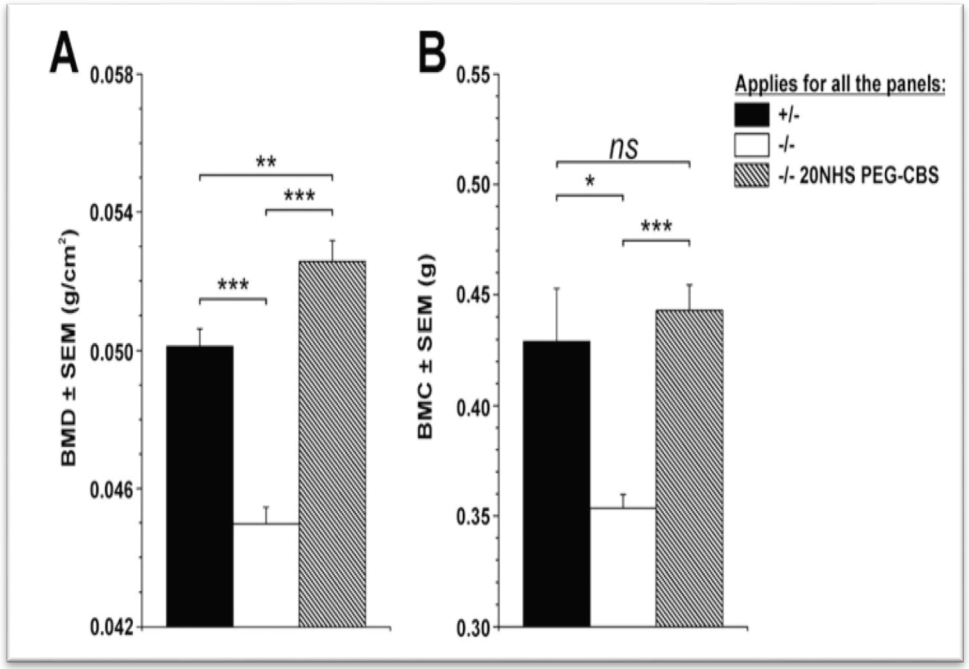
"Metabolic Sink"

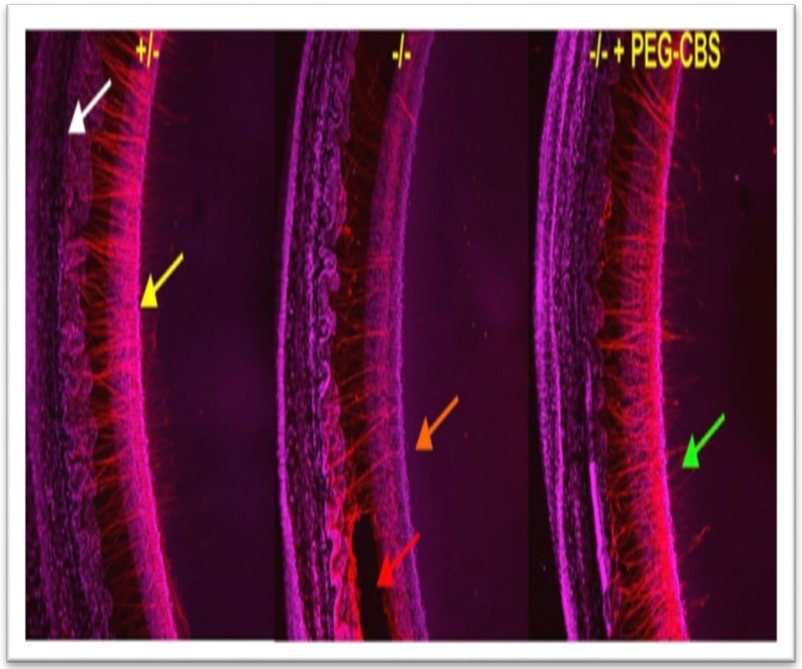
By lowering the Hcy levels in the blood, TVT-058 has been shown to create a concentration gradient that causes excess Hcy in tissues to move to plasma, where it is metabolized

Source: Majtan T. et al., *FASEB J.* 2017; 31(12):5495-5506

Treatment with TVT-058 Appeared to Prolong Survival, Prevent Osteoporosis and Rescue Ocular Structure in Mouse Models







Negative Control | Untreated | TVT-058 treated

Treatment with TVT-058 appeared to prolong survival in KO mouse models[1]

Untreated KO mouse models resulted in significant liver damage and death within 20-30 days

Early treatment with TVT-058 appeared to prevent loss of bone mineralization and fat content in KO mice[1]

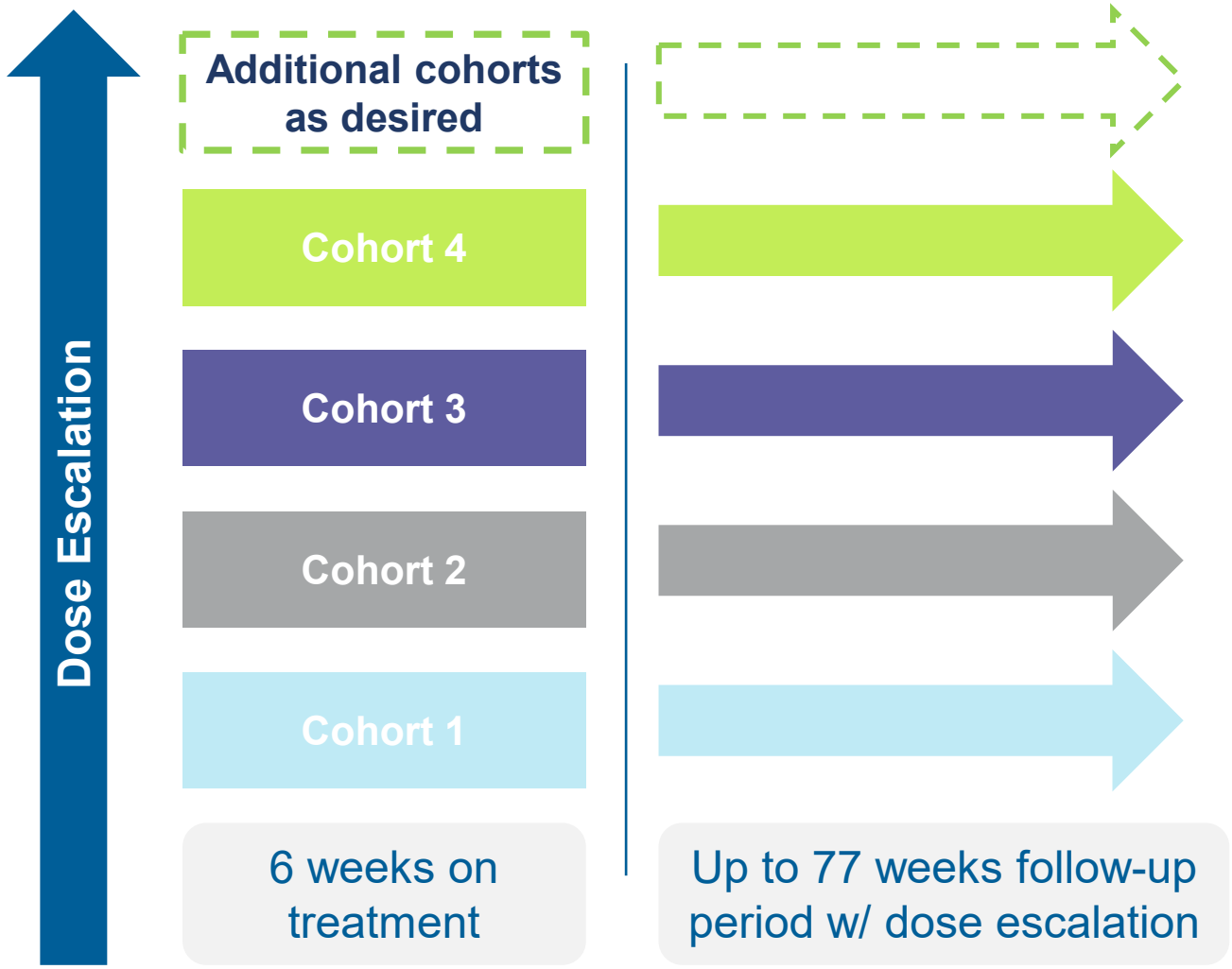
Treatment with TVT-058 appeared to preserve fiber integrity and prevent the degradation of the structure that secures the lens in the eye[2]

Source: 1. Majtan T. et al., *FASEB J.* 2017;31(12):5495-5506; 2. Majtan,T et al., *Mol Ther*. 2018;26(3):834-844

TRAVERE™ THERAPEUTICS

TVT-058 is Advancing in Ongoing Phase 1/2 Clinical Proof-of-Concept Study in HCU

- TVT-058 is advancing in a Phase 1/2 double blind, randomized, placebo-controlled study
- Cohorts are 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



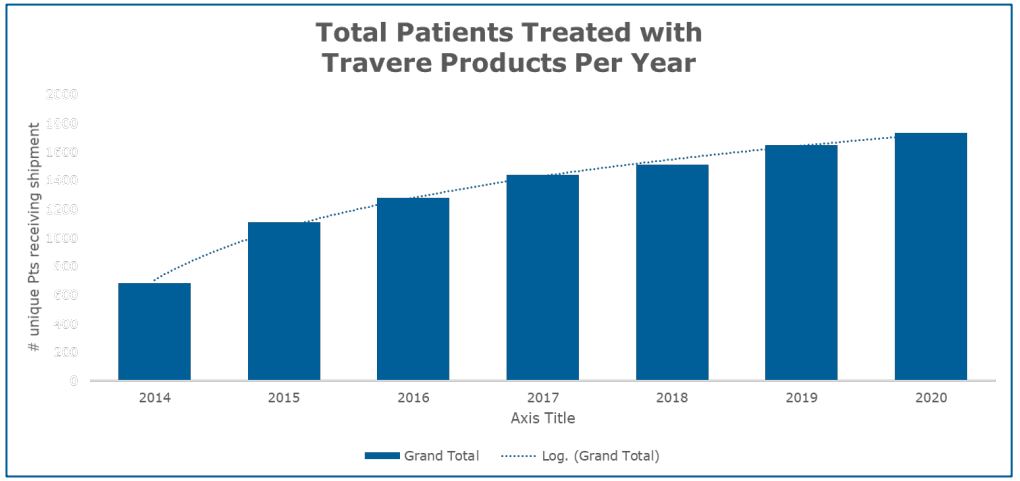
TRAVERE™ THERAPEUTICS

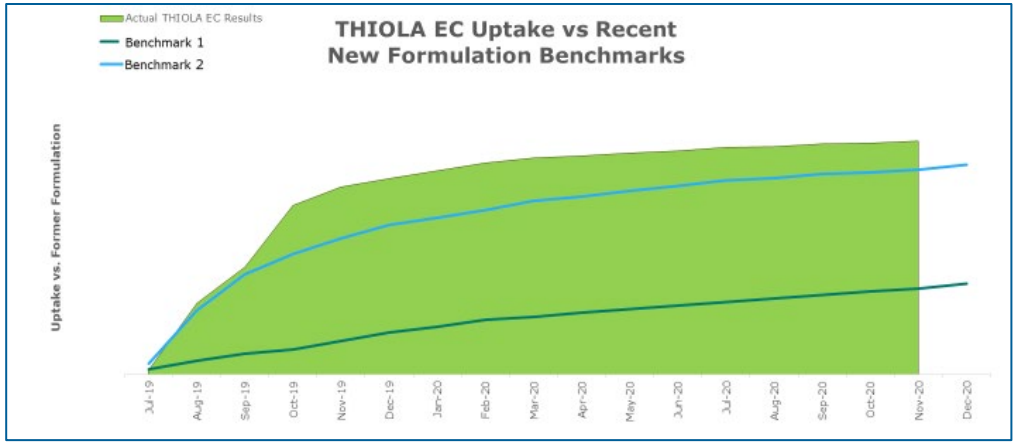
Demonstrated Commercial Capabilities to Deliver Our Pipeline to Patients Upon Approval

TRAVERE™ THERAPEUTICS

Utilizing Established Commercial Rare Disease Capabilities and Nephrology Footprint to Deliver Approved Products*

- **Proven commercial capabilities and infrastructure**
  - Organic year-over-year growth for last five years
  - Preliminary FY 2020 revenue of $199 million, 13% growth over 2019**
- **Experience planning and executing new product launches in rare disease**
  - Recent Thiola EC launch outperformed benchmarks
- **Established nephrology network, Patient Hub and expertise**
  - Field-force currently calling on ~2,000 nephrologists in US
  - Ability to build upon established customer support services
  - Experienced central distribution capabilities to meet the needs of rare patients





ThiolaEC (tiopronin) 300 Enteric-Coated, Delayed-Release Tablets

Thiola (tiopronin)

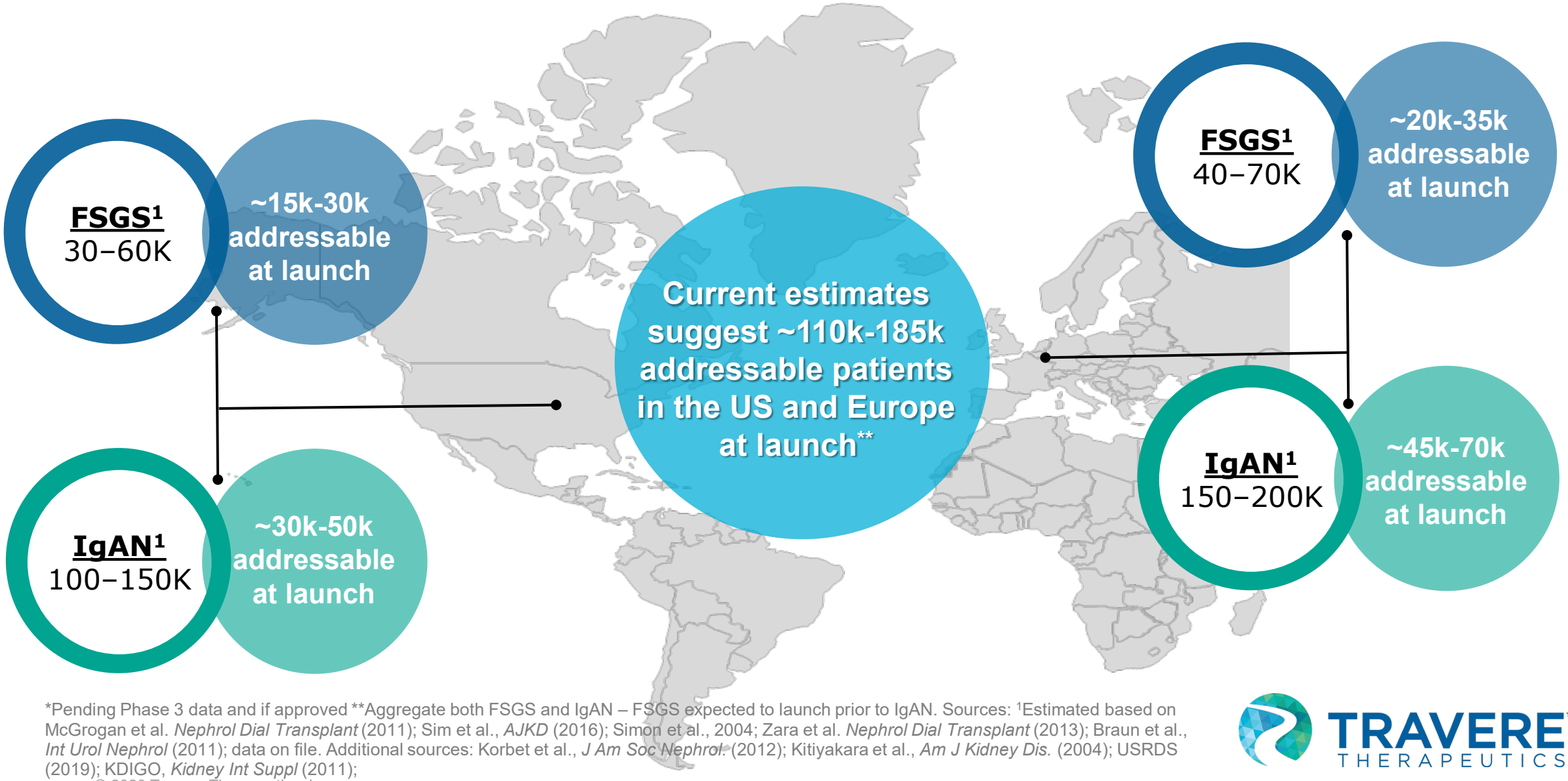
Cholbam (cholic acid) capsules

CHENODAL (Chenodiol Tablets 250 mg)

TRAVERE THERAPEUTICS

*Pending Phase 3 data and if approved, **based upon preliminary, unaudited financial data

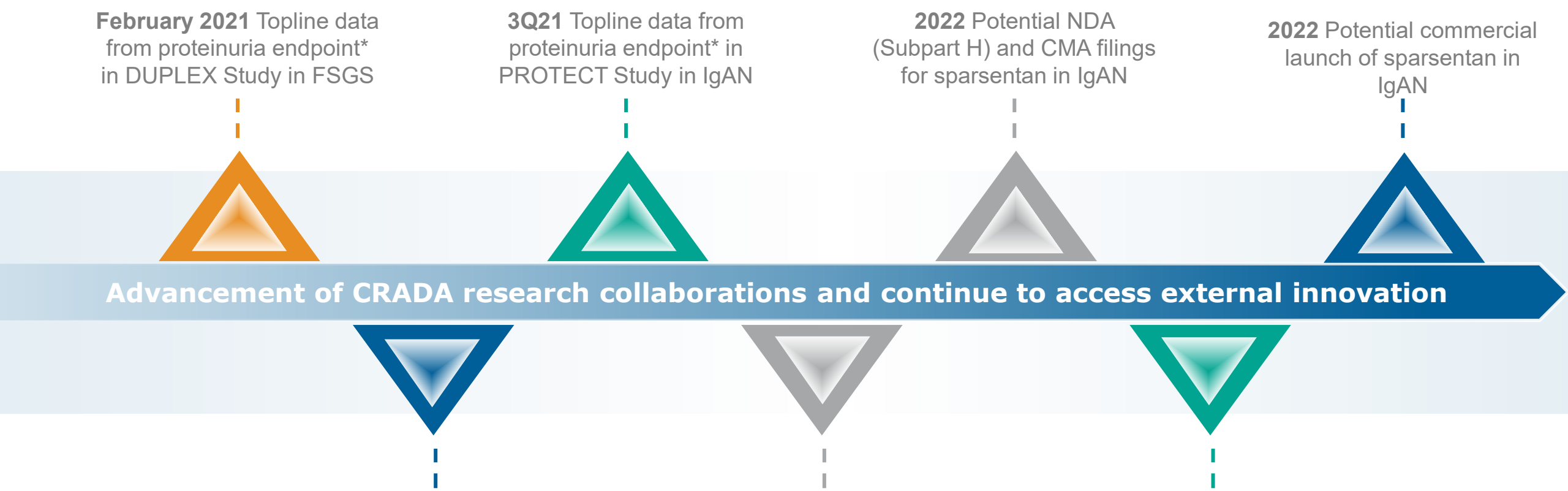
Significant Opportunity to Increase the Number of Patients Treated in the Coming Years if Sparsentan is Approved*



*Pending Phase 3 data and if approved **Aggregate both FSGS and IgAN – FSGS expected to launch prior to IgAN. Sources: [1]Estimated 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);

TRAVERE™ THERAPEUTICS

Path to Potential Breakthrough Growth for Travere Therapeutics



*Interim endpoint; confirmatory endpoint is slope of eGFR

TRAVERE™
THERAPEUTICS

In Rare For Life.