Pioneering New Treatments for Rare Diseases

JANUARY 2024

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THERAPEUTICS

Disclaimers

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This presentation contains forward-looking statements within the meaning of the Private Securities Litigation Reform Act of 1995, as amended, including, without limitation, statements regarding Calliditas' strategy, commercialization efforts, business plans, regulatory submissions, clinical development plans and focus. The words "may," "will," "could," "would," "should," "expect," "plan," "anticipate," "intend," "believe," "estimate," "predict," "project," "potential," "continue," "target," and similar expressions are intended to identify forward-looking statements, although not all forward-looking statements contain these identifying words. Any forward-looking statements in this presentation are based on management's current expectations and beliefs and are subject to a number of risks, uncertainties, and important factors that may cause actual events or results to differ materially from those expressed or implied by any forward-looking statements contained in this presentation, including, without limitation, any related to Calliditas' business, operations, continued and additional regulatory approvals for TARPEYO® and Kinpeygo®, market acceptance of TARPEYO and Kinpeygo, clinical trials, supply chain, strategy, goals and anticipated timelines, competition from other biopharmaceutical companies, revenue and product sales projections and forecasts and other risks identified in the section entitled "Risk Factors" in Calliditas' reports filed with the Securities and Exchange Commission. Calliditas cautions you not to place undue reliance on any forward-looking statements, which speak only as of the date they are made. Calliditas disclaims any obligation to publicly update or revise any such statements to reflect any change in expectations or in events, conditions, or circumstances on which any such statements may be based, or that may affect the likelihood that actual results will differ from those set forth in the forward-looking statements. Any forward-looking statements contained in this presentation represent Calliditas' views only as of the date hereof and should not be relied upon as representing its views as of any subsequent date. For our product developed under the name NEFECON®, we use "TARPEYO" to describe the product commercialized in the United States, "Kinpeygo" to describe the product commercialized in the UK and EU, and "Nefecon" for all other purposes, including for the global Nefecon product franchise. In the United States, the European Union, and other countries, we and our commercialization partners own trademark registrations and applications, and unregistered trademarks, including TARPEYO, NEFECON, and Kinpeygo. Solely for convenience, the trademarks and trade names in this presentation may be referred to without the ® and ™ symbols, without limiting the assertion of rights thereto by the respective owners.

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Corporate Overview

Commercial biotech focused on building a franchise in rare disease

Headquartered in Sweden with full commercial footprint in United States

Dual listed on OMX NASDAQ (CALTX) and NASDAQ (CALT)

Calliditas commercializes the first and only fully FDA approved therapy for IgAN in the US

Late stage rare disease pipeline with 3 Ph2 data readouts in 2024

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Leadership Team Across US & Europe

OVER 150 YEARS' COMBINED EXPERIENCE IN BIG PHARMA AND BIOTECH, WITH COMPANY HEADCOUNT OF ~225 FTES

Renee Lucander
CEO

Maria Törnsén
President N. America

Richard Philipson
CMO

Fredrik Johansson
CFO

Frank Bringstrup
VP Regulatory Affairs

Lars Stubberud
Head Technical Operations

Sandra Frithiof
Head Human Resources

Brian Gorman
General Counsel

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Key Company Metrics

CONTINUAL GROWTH AND SUCCESSFUL EXECUTION OF THE COMMERCIAL ROLLOUT IN THE UNITED STATES

TARPEYO Net Sales 2023[1]	**$100-102M**	**↑ 170% Growth yoy**
Net Revenues in Q4[1]	**$31-33M**	**↑ 100% Growth yoy**
Enrollments Since Launch Through Q4[1]	**2,791**	**555 enrollments in Q4**
Total 2023 Revenues[1]	**$111-113M**	**Includes Milestone Payments & Royalties ex-US**
Cash Used in Operating Activities Q3 2023	**$6M**	**↓ 50% Reduction yoy**
Cash Position Q3 2023	**$73M**	

Calliditas is the category leader in IgAN - a multi-billion dollar global opportunity

[1]Calliditas' unaudited, estimated results for the quarter and year ended December 31, 2023 included on Slide 4 are preliminary financial information, remain subject to completion, and were prepared by management based upon estimates, a number of assumptions and currently available information, and are subject to revision based upon, among other things, quarter-end closing procedures and/or adjustments, the completion of Calliditas' financial statements and other operational procedures. Calliditas' actual results could be materially different from this preliminary financial information, which should not be regarded as a representation by Calliditas as to Calliditas' actual results for the quarter and year ended December 31, 2023. In addition, Calliditas' independent registered public accounting firm has not audited, reviewed, compiled or performed any procedures with respect to this preliminary financial information and does not express an opinion or any other form of assurance with respect to this preliminary financial information. During the course of the preparation of Calliditas' financial statements and related notes as of and for the quarter and year ended December 31, 2023, Calliditas may identify items that would require Calliditas to make material adjustments to this preliminary financial information.

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Pipeline to Build a Rare Disease Franchise

DEVELOPING SETANAXIB PLATFORM ACROSS 3 RARE DISEASE INDICATIONS WITH 3 PH2 DATA READOUTS IN 2024

		Phase 1	Phase 2	Phase 3	Approved	Status	Notes
NEFECON	Immunoglobulin A Nephropathy (IgAN)					Commercial	US (TARPYEO) Europe (KINPEYGO) China (NEFECON)
						Pre-Commercial	Japan
SETANAXIB	Primary Biliary Cholangitis					Ongoing	
	Idiopathic Pulmonary Fibrosis					Ongoing	Investigator Led Study
	Alport Syndrome					Ongoing	
	Solid Tumors (SCCHN)					Ongoing	Partnering Focus

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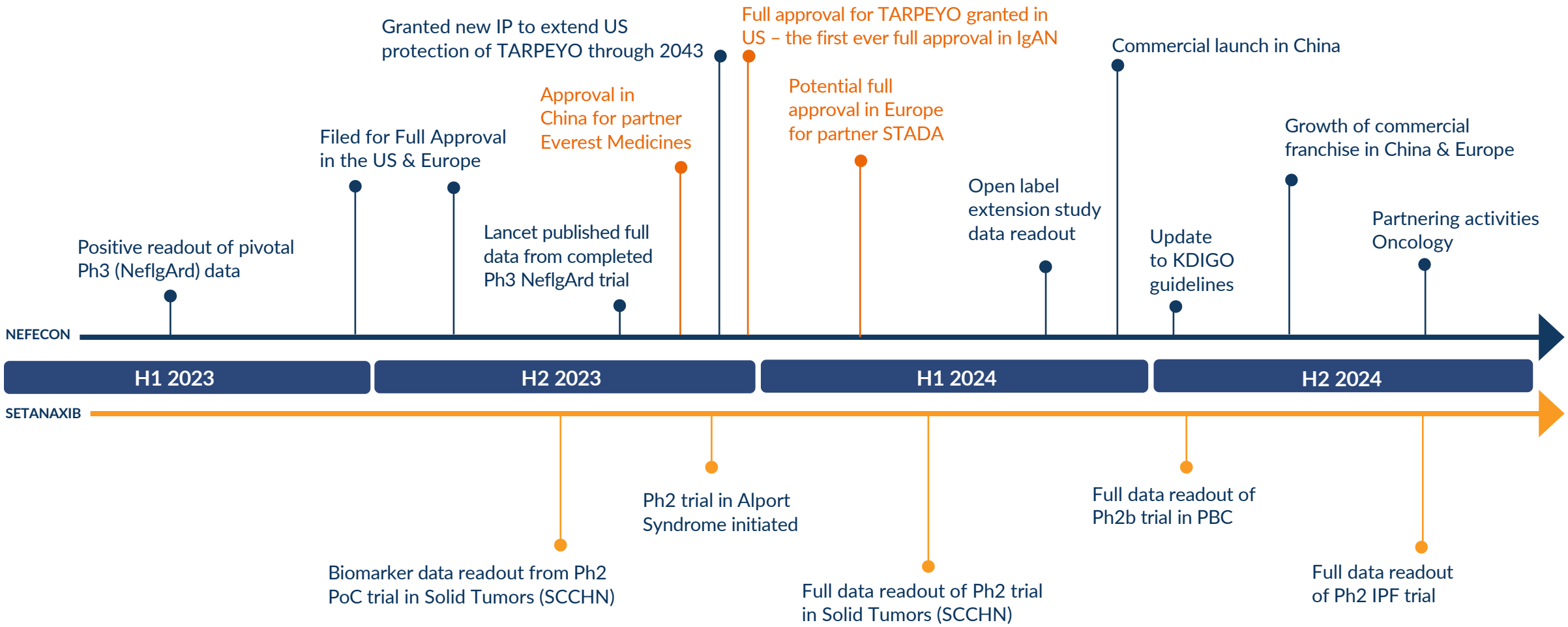
Recent and Upcoming Key Events

EXCITING YEAR AHEAD WITH GLOBAL COMMERCIAL RAMP AND 3 PH2 DATA READOUTS EXPECTED FROM OUR PIPELINE

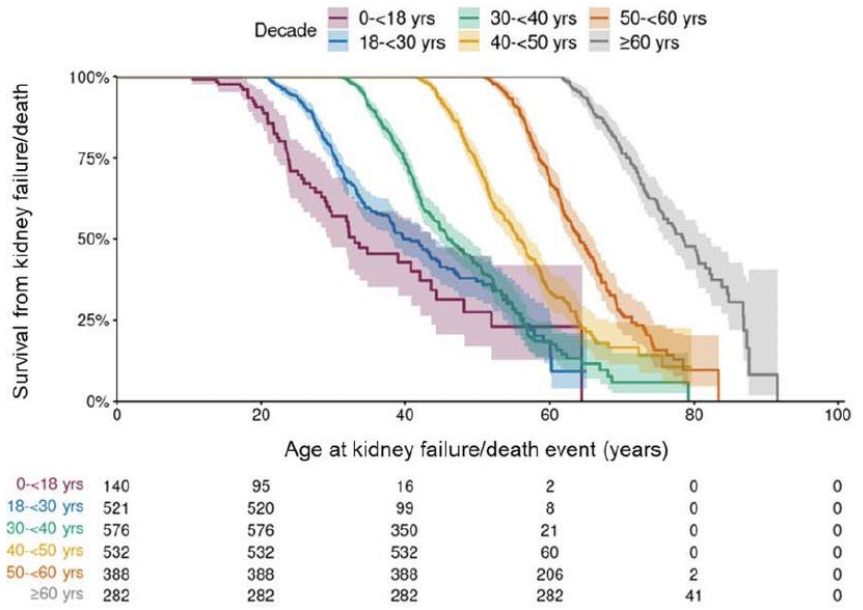


IgA Nephropathy – A Rare Autoimmune Kidney Disease

A PROGRESSIVE DISEASE WITH A HIGH UNMET NEED FOR A SAFE AND EFFECTIVE DISEASE-MODIFYING TREATMENT

- Unknown cause but genetic predisposition
- Majority diagnosed before 40 years old
- Most patients progress to kidney failure within 10–15 years across all age groups
- Mean age of kidney failure is 48 years old (n=2,439)
- Progression to kidney failure leads to hemodialysis or kidney transplant

FEW PATIENTS AVOID KIDNEY FAILURE DURING LIFETIME



Pitcher D. et al. Long-Term Outcomes in IgA Nephropathy. Clin J Am Soc Nephrol. 2023 Jun 1;18(6):727-738. DOI: 10.2215/CJN.0000000000000135

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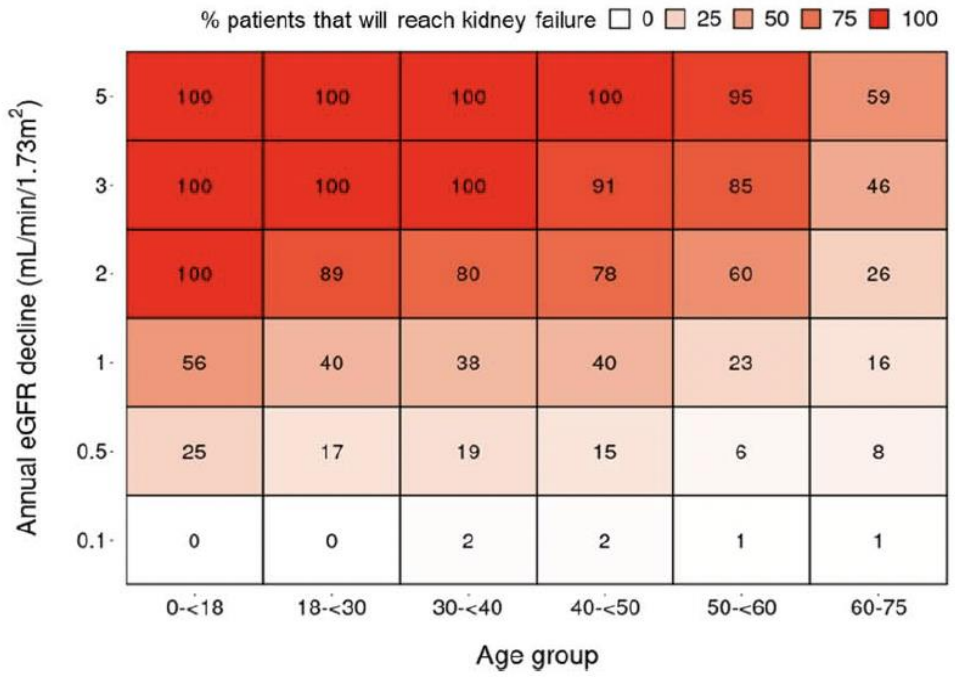
IgAN Population at Risk is Larger Than Anticipated

INCREASING EVIDENCE OF FASTER PROGRESSION IN 'HIGH RISK' PATIENT SEGMENT AND PATIENTS TRADITIONALLY REGARDED AS 'LOW RISK' EXHIBITING HIGH RATES OF KIDNEY FAILURE WITHIN 10 YEARS OF DIAGNOSIS



Almost all patients at risk of kidney failure within their expected lifetime, **regardless of risk profile – preservation of kidney function being key**

Urgency of need to treat earlier to preserve kidney function

High Risk Defined: Patients with baseline proteinuria values ≥0.88g/g, Low Risk Defined: Patients with baseline proteinuria <0.88 g/g

Pitcher D. et al. Long-Term Outcomes in IgA Nephropathy. Clin J Am Soc Nephrol. 2023 Jun 1;18(6):727-738. DOI: 10.2215/CJN.0000000000000135

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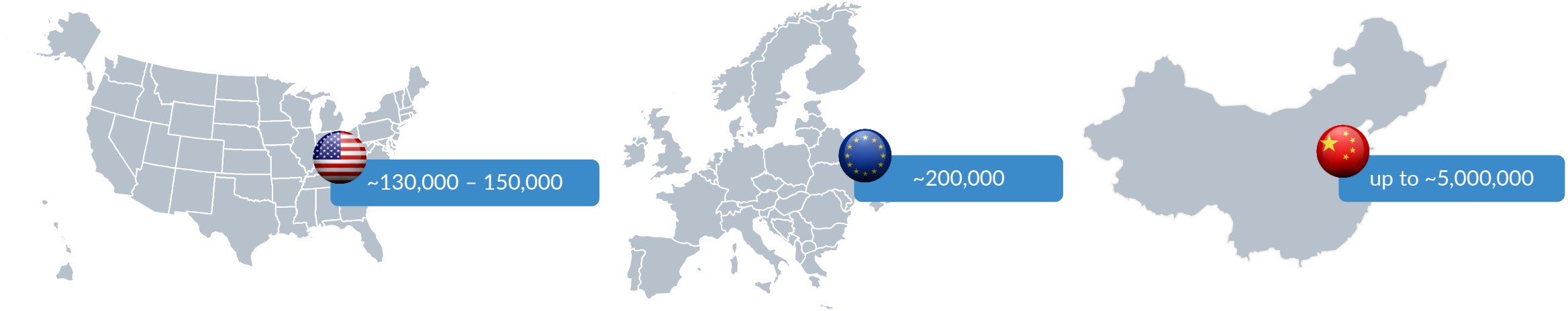
IgA Nephropathy – A Multi-Billion Dollar Global Market Opportunity

RARE KIDNEY DISEASE THAT IS THE MOST COMMON PRIMARY GLOMERULONEPHRITIS GLOBALLY

PREVALENCE & EPIDEMIOLOGY



In the US we estimate 2/3rds of the 12,000 nephrologists treat IgAN patients

Majority of patients are seen by 4,000-5,000 specialists in the US

40% vs. 60% treated in academic and community settings respectively*

We estimate the global IgAN market to grow to ~$5-8B

Pitcher D. et al. Long-Term Outcomes in IgA Nephropathy. Clin J Am Soc Nephrol. 2023 Jun 1;18(6):727-738. DOI: 10.2215/CJN.0000000000000135
*Veeva OpenData for 2023, including all active HCPs where the primary specialty is Nephrology

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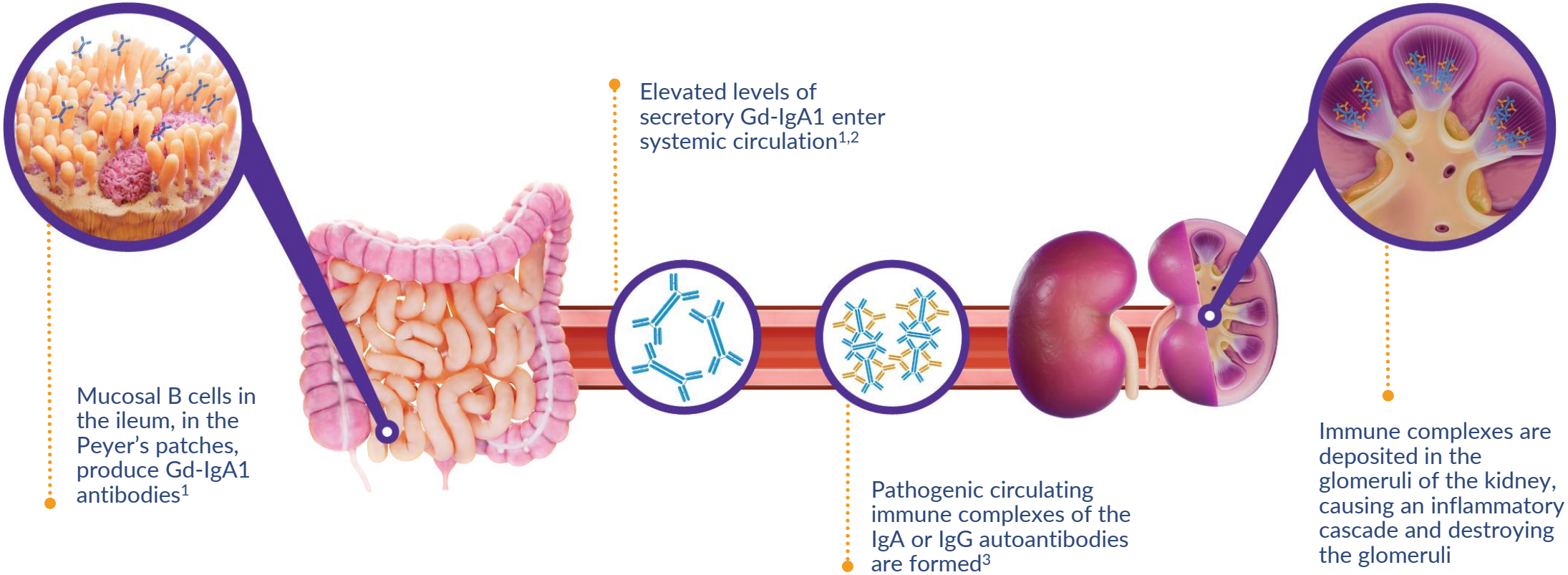
Underlying Pathogenesis of IgAN Informed Targeted Approach

DISEASE ORIGINATING IN THE ILEUM LEADS TO A DAMAGING DEPOSITION OF IMMUNE-COMPLEXES IN THE KIDNEY



Gd-IgA1: Galactose-deficient Immunoglobulin A1, IgA: Immunoglobulin A, IgG: Immunoglobulin G

[1]Barratt J, et al. Kidney Rep. 2020;5(10):1620-1624, [2]Kiryluk K, et al. J Clin Invest. 2014;124(6):2325-2332, [3]Canetta PA, et al. Clin J Am Soc Nephrol. 2014;9(3):617-625

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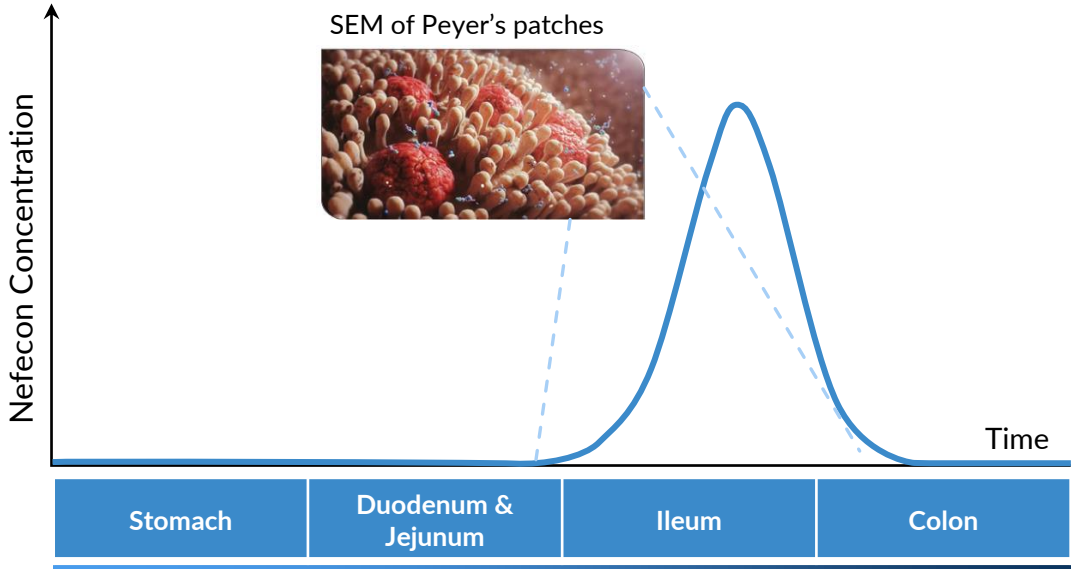
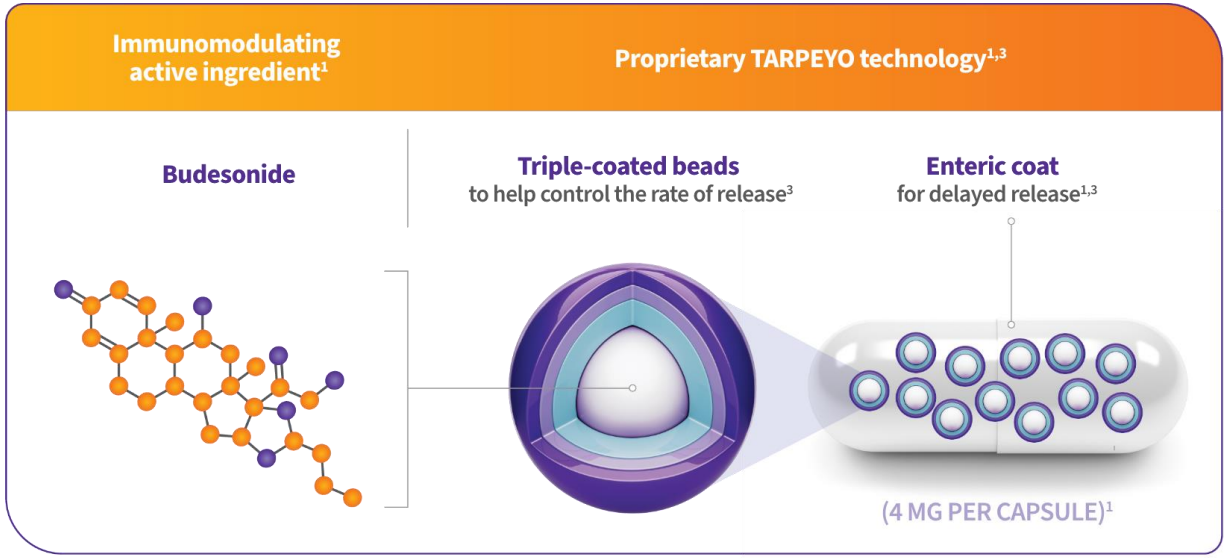
Formulated to Target IgAN at the Disease Origin

NEFECON DELIVERS A HIGHLY TARGETED DOSE DIRECTLY TO B CELLS IN PEYER'S PATCHES OF THE ILEUM





pH-governed release delivers a highly potent sustained release of budesonide to ileum

90% cleared in first-pass metabolism by the liver minimizing systemic side effects

Statistically significant impact on IgA1 and circulating immune complexes

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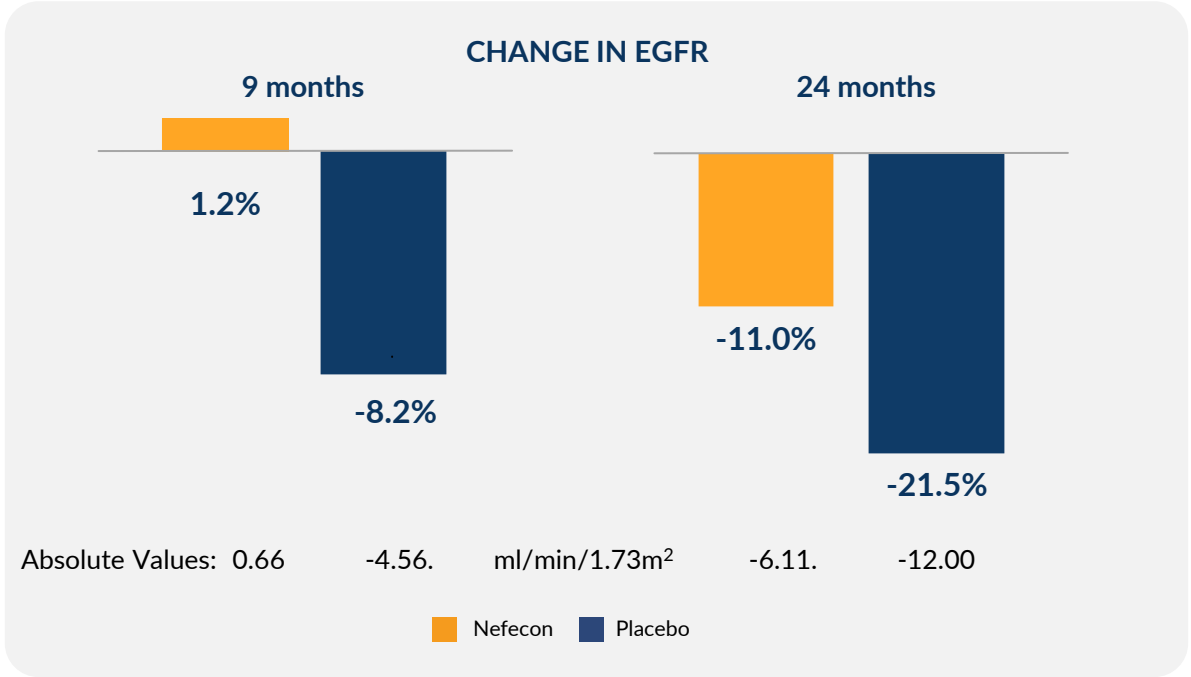
eGFR Stabilization during Treatment – Benefit Sustained Long Term

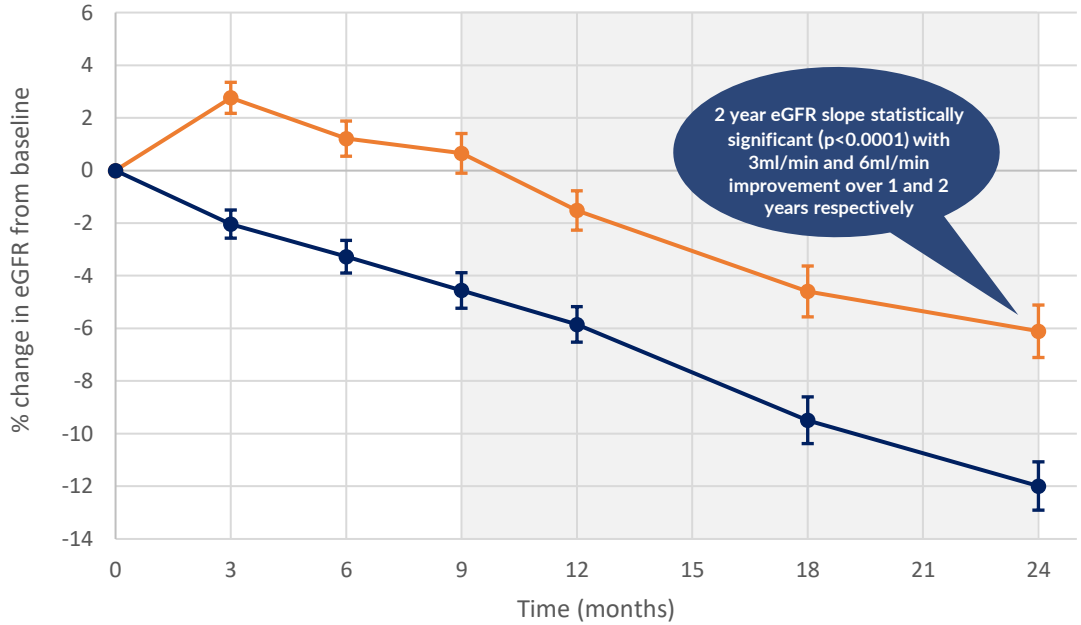
PH3 DATA SHOW SUSTAINED eGFR IMPROVED AT 24 MONTHS FOLLOWING 9 MONTHS OF DOSING WITH 16MG NEFECON (N=364)

Ph3 met primary endpoint demonstrating a highly statistically **significant eGFR benefit vs. placebo (p< 0.0001) over 24 months**

This sustained eGFR benefit was observed following a 9 month treatment period on Nefecon (16mg) and a 15 month follow up period off drug

The positive impact on eGFR was sustained and continued to provide functional benefit to patients beyond the treatment period





50% less loss of kidney function over 24 months

eGFR: estimated Glomerular Filtration Rate

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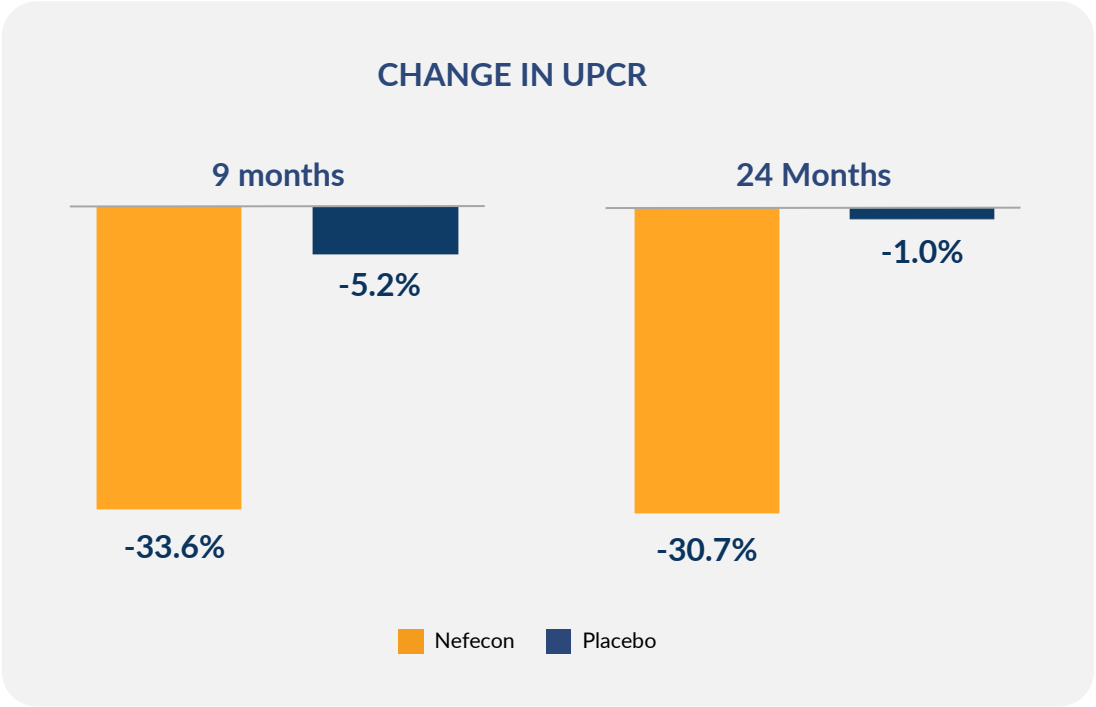
Significant and Sustained Proteinuria Reduction

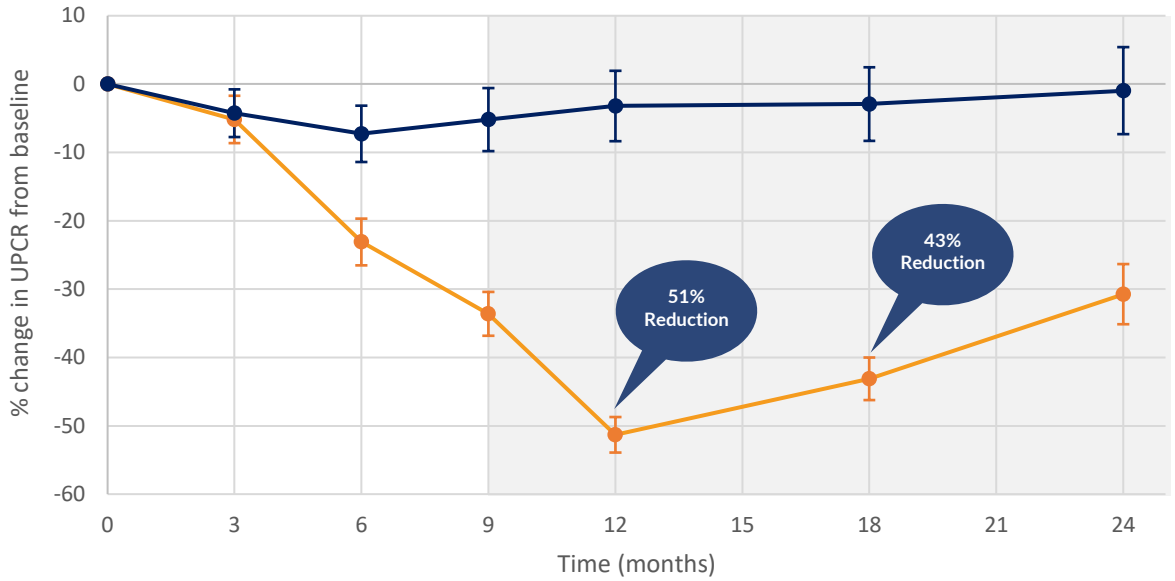
REDUCTION IN UPCR (PROTEINURIA) CONTINUED DURING THE 15 MONTH FOLLOW-UP PERIOD WITH PATIENTS OFF DRUG

Nefecon patients maintained >30% UPCR reduction from the end of the 9 month treatment period through the 15 month follow-up period, with a 51% reduction in UPCR observed at 12 months

Statistically significant difference in UPCR in Nefecon vs. placebo patients between 12 and 24 months ($p<0.0001$)

Ph3 data demonstrate that UPCR reductions gained during treatment were durable beyond treatment





Treatment Period | **Off Drug Follow Up Period**

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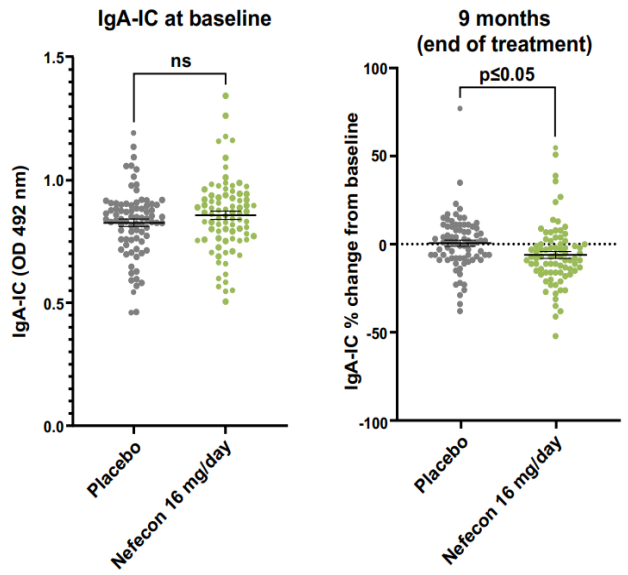
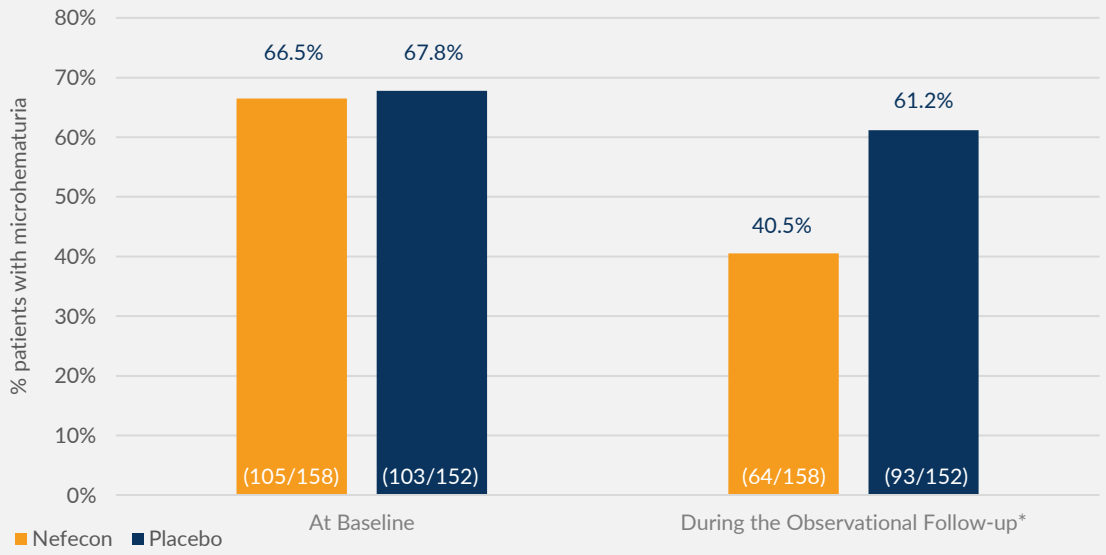
Statistically Significant Improvement in IgAN Biomarkers

DATA SUPPORT NEFECON'S B-CELL MODULATORY EFFECT AND DISEASE-MODIFYING EFFECT ON IGAN



Nefecon resulted in a statistically significantly reduction in IgA-IC levels over the treatment period vs. placebo (p≤0.05)



Nefecon resulted in a statistically significant improvement in the proportion of patients exhibiting microhematuria vs. placebo (p=0.0001)

IgA-IC: IgA-containing Immune Complex *Patients with a positive urine dipstick result in at least 2 of the following time points: 12, 18 and 24 months following the first dose of Nefecon

Zand et al. Microscopic hematuria as a risk factor for IgAN progression: considering this biomarker in selecting and monitoring patients, Clin Kidney Journal, 2023 Dec; 16(2): ii19–ii27. https://doi.org/10.1093/ckj/sfad232

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FDA Granted Full Approval in United States

TARPEYO IS THE FIRST FULL APPROVAL IN IGAN AS OF DEC 20, 2023

TARPEYO is indicated to reduce the loss of kidney function in adults with primary IgAN who are at risk for disease progression

The recommended dosage is 16mg administered orally once daily

Please refer to package insert for full prescribing information including safety information

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TARPEYO™ (budesonide) delayed release capsules

US Commercial Rollout Gaining Momentum

NEPHROLOGIST AWARENESS NOW >90 PERCENT WITH PEER-TO-PEER RECOMMENDATIONS BUILDING

555
New Patients Enrolled in Q4
YTD patients enrolled: ~1,753

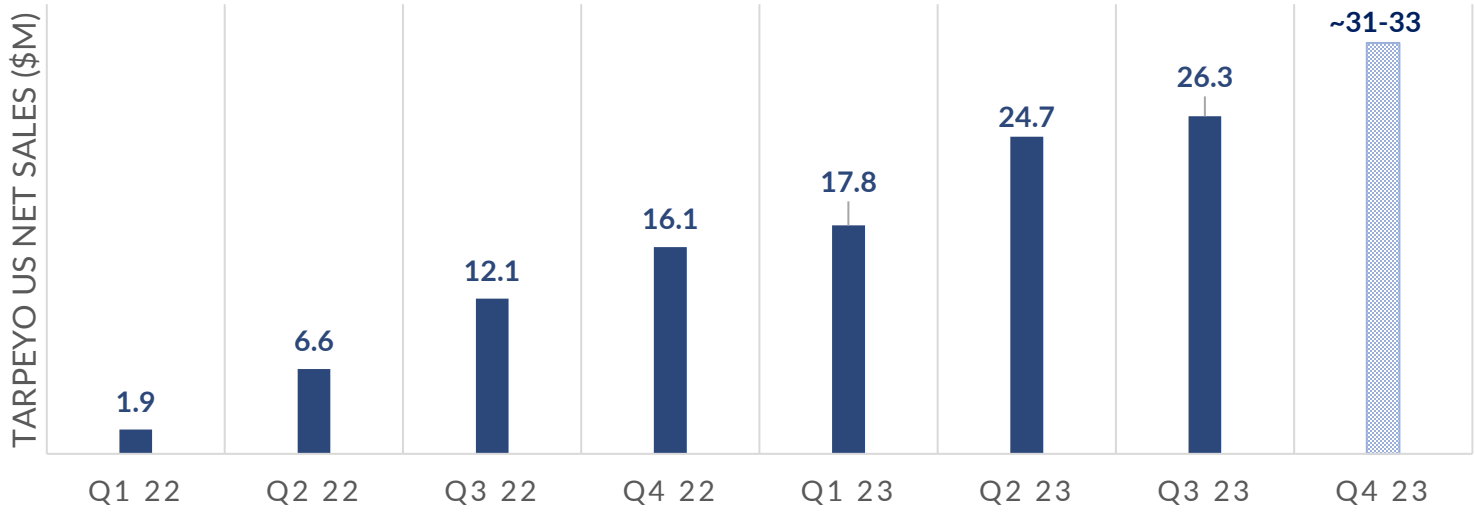
197
New Prescribers in Q3
LTD Prescribers: 1,338

86%
Of patients enrolled in TARPEYO Touchpoints got TARPEYO YTD*

Majority of patients that receive full 9 months of TARPEYO treatment remain on therapy beyond 9 months

>90% payor coverage of US lives
~65% Commercial
~35% Medicare/Medicaid

~$137M in Cumulative Net Sales Since Launch



YTD: Year to Date, LTD: Launch to Date
*Does not include patients that are still waiting final insurance decision

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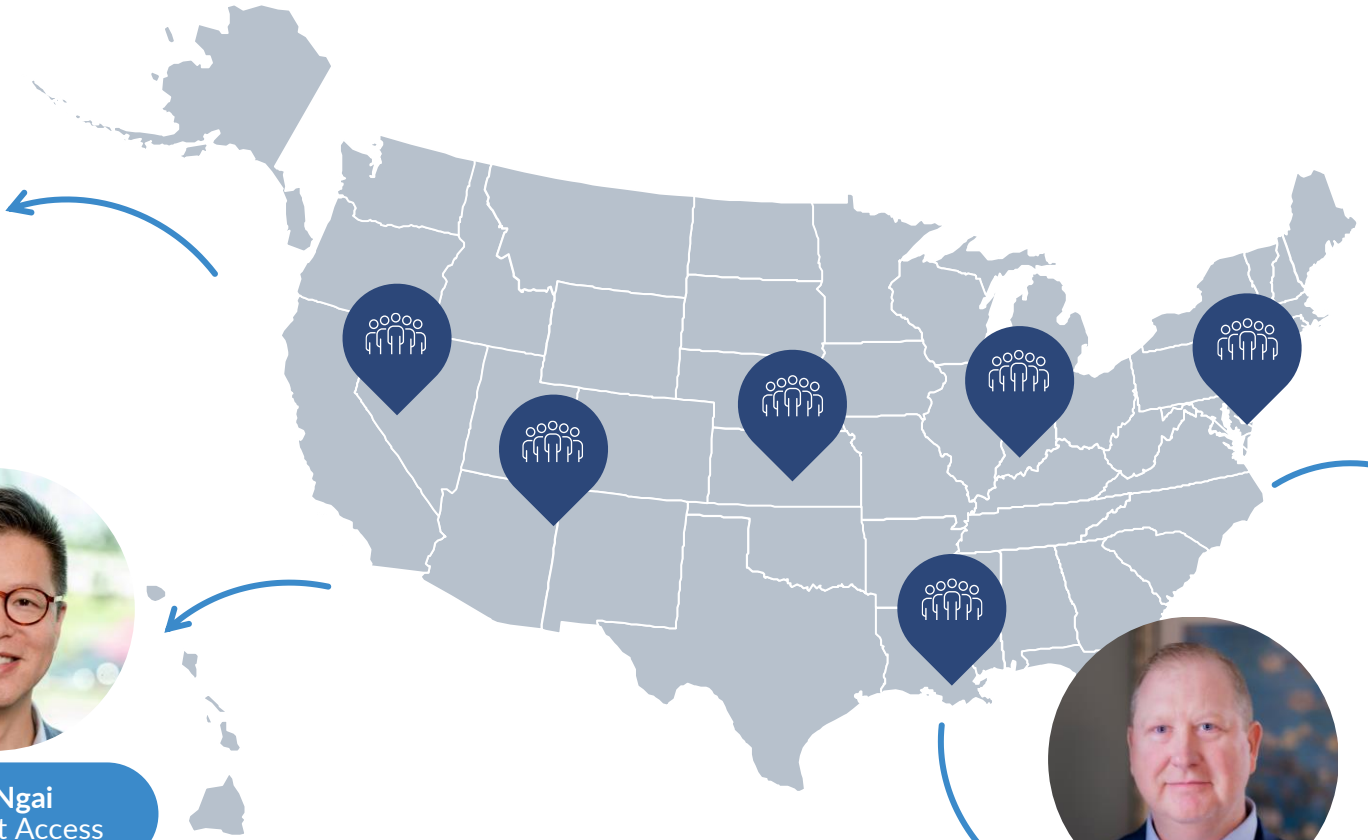
US Commercial Leadership & Infrastructure

TARPEYO FRANCHISE IS SUPPORTED BY 100 FIELD-BASED PROFESSIONALS WHICH INCLUDES 70 SALES REPS

David Ferraro
Head US Sales



Maria Törnsén
President N. America

Chris Ngai
VP Market Access

Warren Brooks
VP Medical Affairs

Teona Johnson
VP Marketing

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US Commercial Strategic Approach

REDUCING MARKET ACCESS FRICTION BY INCREASING PHYSICIANS' EDUCATION & AWARENESS WITH FIELD SUPPORT

MEDICAL AFFAIRS

- Educate HCPs on IgAN & TARPEYO
- Advisory boards
- Local, regional, national conferences
- Peer-to-Peer Engagements

MARKETING

- F2F & digital outreach by salesforce
- Advocacy groups & associations
- Patient summits & conferences
- Social media

MARKET ACCESS

- Integrated hub for physicians, payors & patients
- Dedicated case managers
- Financial assistance program
- Single specialty pharmacy

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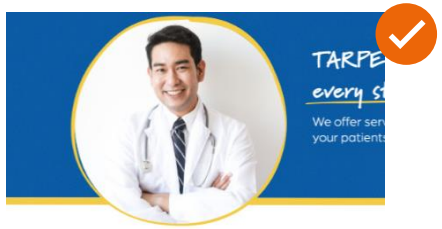
Anticipated Positive Impact of Full Approval

PH3 MET PRIMARY ENDPOINT WITH HIGH STATISTICAL SIGNIFICANCE AND SHOWED SUSTAINED eGFR EFFECT BENEFIT OFF DRUG

FULLY APPROVED LABEL REFLECTS PRESERVATION OF KIDNEY FUNCTION

TARPEYO is indicated to reduce the loss of kidney function in adults with primary IgAN who are at risk for disease progression, irrespective of proteinuria levels



REDUCED MARKET ACCESS FRICTION

Now able to promote on full dataset with recently enlarged salesforce

Open label extension study data from patients with a second 9 month course of treatment due 1H 2024

RaDaR
National Registry of Rare Kidney Diseases

SIGNIFICANTLY LARGER ADDRESSABLE MARKET

Increasing evidence of faster progression than originally assumed in 'high risk' patient segment

Patients traditionally regarded as 'low risk' exhibiting high rates of kidney failure within 10 years of diagnosis

KDIGO to update treatment practice guidelines for IgAN expected in mid-2024 - potential for these to support use of approved drugs and broadening definition of at risk population

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Commercial Partnerships

SUCCESSFUL EXECUTION OF GLOBAL PARTNERSHIP STRATEGY EX-US TO OPTIMIZE VALUE OF NEFECON FRANCHISE

		STADA	EVEREST MEDICINES	VIATRIS
When		**2021**	**2019**	**2022**
Where		**Europe, UK, Switzerland**	**Greater China, Singapore, S. Korea**	**Japan**
Total Deal Value		**€97.5M**	**$121M**	**$100M**
	Upfront	€20M	$15M	$20M
	Milestones	€77.5M	$106M	$80M
	Royalties	Low 20s - Low 30s	Low – Mid Teens	Mid Teens
Stage		**Commercial**	**Commercial**	**Bridging Study (36 pts)**
Status		**Launched in Germany & UK Full approval filed 2H 2023 anticipated 1H 2024**	**Approval granted in Mainland China & Macau Nov 2023**	**Agreement with regulator for approval in conjunction with Global data set**

Commercialized by Tanner Pharma Group via a Named Patient Program for unpartnered RoW territories

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Unencumbered Anti-Fibrotic Rare Disease Platform

CALLIDITAS HOLDS GLOBAL RIGHTS TO SETANAXIB IN ALL INDICATIONS – POTENTIAL TO PARTNER ONCOLOGY RIGHTS

SETANAXIB

	Phase 1	Phase 2	Phase 3	Status	Notes
Primary Biliary Cholangitis				Ongoing	Full data readout 2H 2024 ODD by FDA & EMA granted FTD by FDA granted
Idiopathic Pulmonary Fibrosis				Ongoing / Invest Led	Full data readout 2H 2024 ODD by FDA & EMA granted Investigator led study
Alport Syndrome				Ongoing	Ph2 initiated Nov 2023 ODD by FDA & EMA granted
Solid Tumors (SCCHN)				Ongoing	Full data readout 1H 2024 Available for out-licensing

Extensive safety dataset with >320 subjects exposed to setanaxib in completed Ph1 and Ph2 clinical trials

Favorable safety profile – could be used as an add-on therapy across a broad set of fibrotic indications

2 composition of matter patent families through 2028/29 (ex. extensions) - further patent families issued/in prosecution - medical uses, compounds

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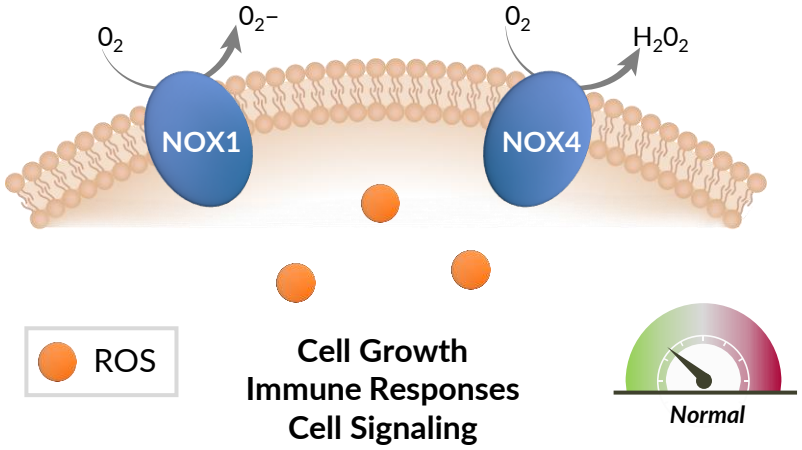
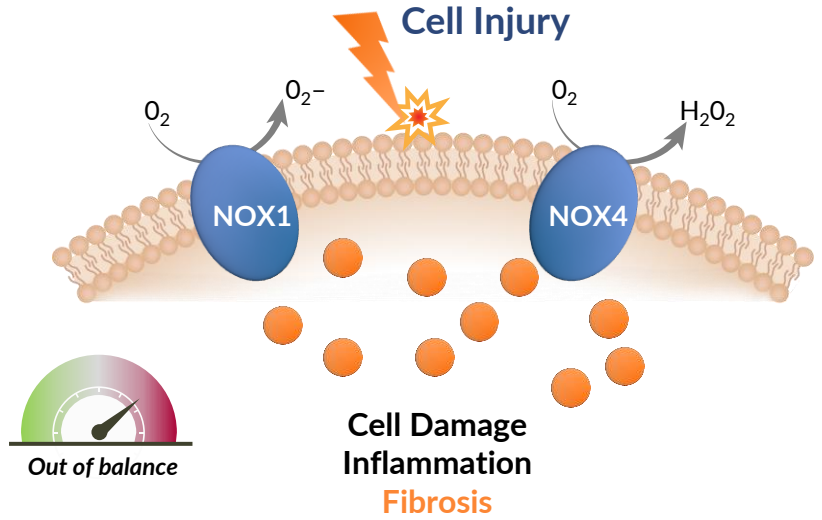
NOX1 and NOX4 are Important Targets for Fibrotic Disease

NOX (NADPH OXIDASE) ENZYMES CATALYZE THE CONVERSION OF OXYGEN INTO REACTIVE OXYGEN SPECIES (ROS)



The generation of ROS by NOX enzymes is an essential part of biochemical cascades which regulate normal cell growth, immune responses, cell signalling and autophagy



Excessive NOX1/4 expression is induced when a cell is injured, leading to unbalanced redox homeostasis and eventually fibrogenesis

Sullivan L.B. & Chandel N.S. Mitochondrial reactive oxygen species and cancer. Cancer Metab. 2014 Nov 28;2:17. https://doi.org/10.1186/2049-3002-2-17
Meitzler J.L. et al. Decoding NADPH oxidase 4 expression in human tumors. Redox Biol. 2017 Oct;13:182-195. https://doi.org/10.1016/j.redox.2017.05.016
Lin W. et al. Reactive Oxygen Species in Autoimmune Cells: Function, Differentiation, and Metabolism. Front Immunol. 2021 Feb 25;12:635021. https://doi.org/10.3389/fimmu.2021.635021
Vermot A. et al. NADPH Oxidases (NOX): An Overview from Discovery, Molecular Mechanisms to Physiology and Pathology. Antioxidants (Basel). 2021 Jun 1;10(6):890. https://doi.org/10.3390/antiox10060890
Levy C, Manns M, Hirschfield G. New Treatment Paradigms in Primary Biliary Cholangitis. Clin Gastroenterol Hepatol. 2023 Jul;21(8):2076-2087. https://doi.org/10.1016/j.cgh.2023.02.005

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The Opportunity for Setanaxib in PBC

SETANAXIB ADDRESSES ELEMENTS OF PBC THAT NO APPROVED, OR PHASE 3 DRUGS TARGET

No approved therapies for PBC that specifically target fibrosis of the liver

Disabling, clinically meaningful, symptoms such as fatigue are not addressed by current therapies

Up to 80% of PBC patients suffer from chronic fatigue which meaningfully impacts QoL

A novel anti-fibrotic agent like setanaxib could delay disease progression and obviate transplant

Setanaxib differentiated in its positive impact on important QoL measures such as fatigue

Combination therapy targeting several pathways may present the best path forward for the treatment of PBC

PBC: Primary Biliary Cholangitis, QoL: Quality of Life

Shahini E, Ahmed F. Chronic fatigue should not be overlooked in primary biliary cholangitis. J Hepatol. 2021 Sep;75(3):744-745. https://doi.org/10.1016/j.jhep.2021.02.020
Goldblatt J. et al. The true impact of fatigue in primary biliary cirrhosis: a population study. Gastroenterology. 2002 May;122(5):1235-41. https://doi.org/10.1053/gast.2002.32993
Witt-Sullivan H. et al. The demography of primary biliary cirrhosis in Ontario, Canada. Hepatology. 1990 Jul;12(1):98-105. https://doi.org/10.1002/hep.1840120116

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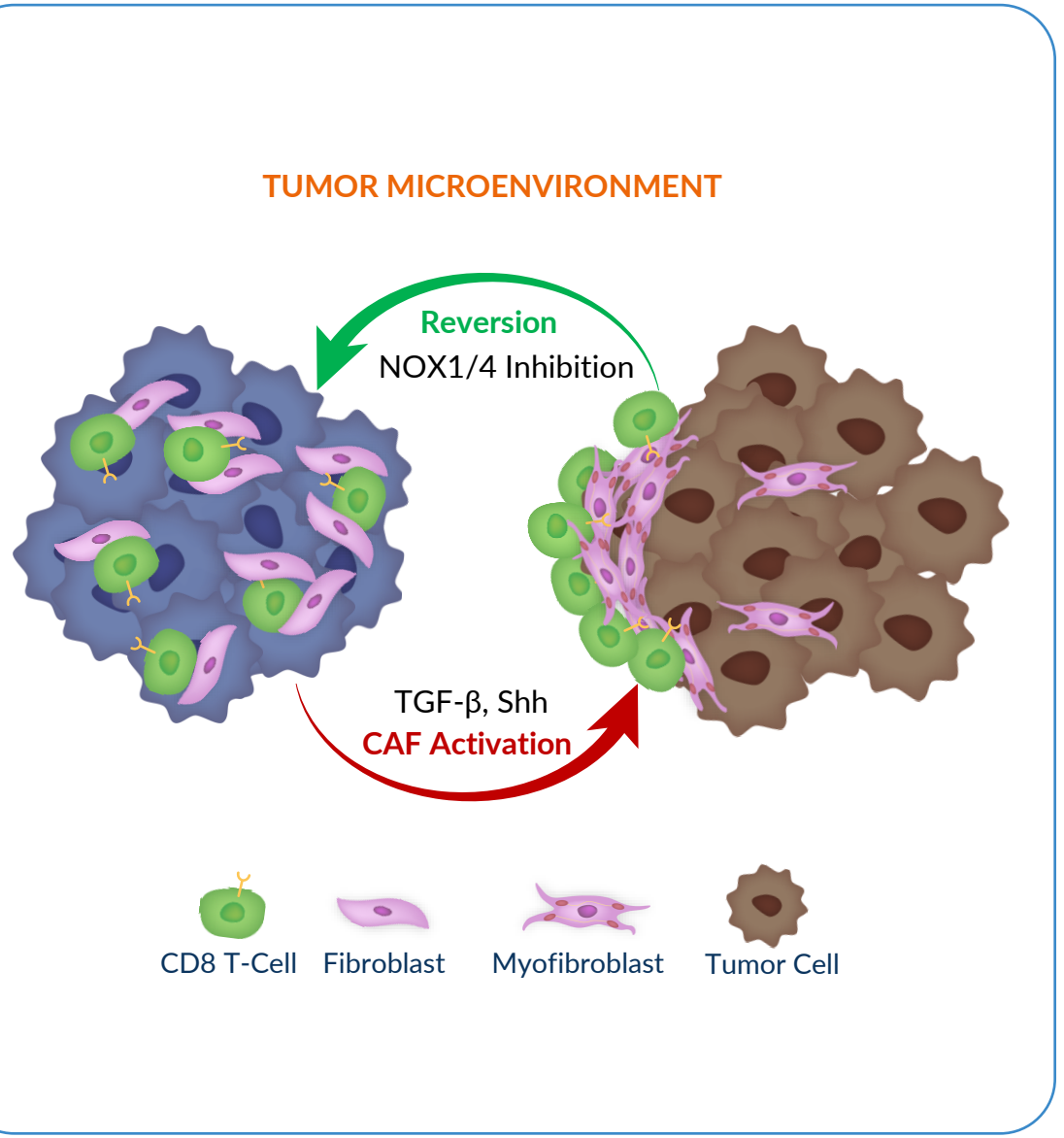
CAF-Rich Tumors are Sitting Targets for NOX Inhibition

SETANAXIB IS THE FIRST DUAL NOX1/4 INHIBITOR TO REACH THE CLINICAL TRIAL STAGE

Solid tumors rich in CAFs are more resistant to IO therapies, with low response rates (ORR ~18%) for 1L pembrolizumab monotherapy in recurrent/metastatic SCCHN

Inhibiting NOX1 and NOX4 expressed by CAFs is thought to trigger the reversion of CAFs (myofibroblasts) back to quiescent fibroblasts

This means NOX1/4 inhibition with setanaxib could prevent the immuno-exclusion by CAFs, re-sensitizing the tumor microenvironment by improving the penetration of immune cells, augmenting IO efficacy



CAFs: Cancer Associated Fibroblasts, IO: Immuno-oncology, NOX: NADPH Oxidase, ORR: Overall Response Rate, TGF-β: Transforming Growth Factor-β, Shh: Sonic Hedgehog

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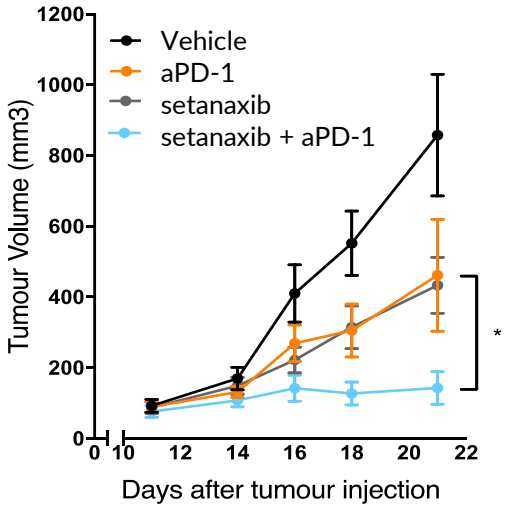
Preclinical Efficacy of Setanaxib + Anti-PD1 in CAF-Rich Solid Tumors

STATISTICALLY SIGNIFICANT REDUCTION IN TUMOR VOLUME, IMPROVED SURVIVAL AND TIL PENETRATION IN CAF-RICH MODELS

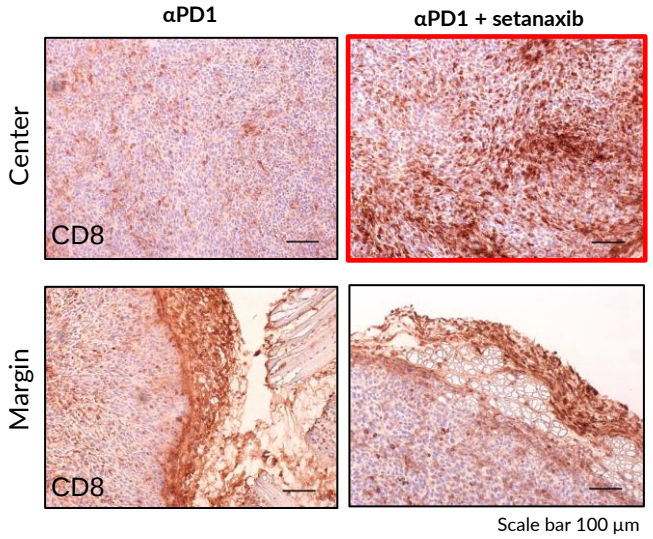
Legacy preclinical data in murine CAF-rich solid tumor models treated with setanaxib + anti-PD1 combination resulted in in statistically significant reduction in tumor volume, improvement in survival and immune cell penetration (TILs) to the tumor center

These data drove the rationale to develop this combination in the Ph2 proof-of-concept study

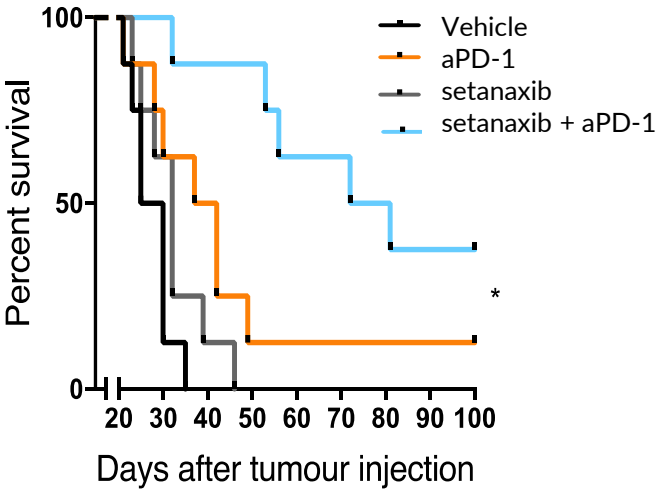
TUMOR GROWTH



TIL TUMOR PENETRATION



SURVIVAL



*p<0.05, CAF: Cancer-Associated Fibroblast, TIL: Tumor Infiltrating Lymphocytes, αPD-1: Anti-Programmed Death-Ligand 1 antibody

Ford K. et al. NOX4 Inhibition Potentiates Immunotherapy by Overcoming Cancer-Associated Fibroblast-Mediated CD8 T-cell Exclusion from Tumors. Cancer Res. 2020 May 1;80(9):1846-1860. https://doi.org/10.1158/0008-5472.CAN-19-3158

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Ph2 Positive Interim Data at 9 Weeks in SCCHN

DATA SHOWS ENCOURAGING EARLY CLINICAL PFS AND SUPPORTS ANTI-FIBROTIC MOA

July 2023 readout from 16 evaluable patients after 9 weeks of treatment

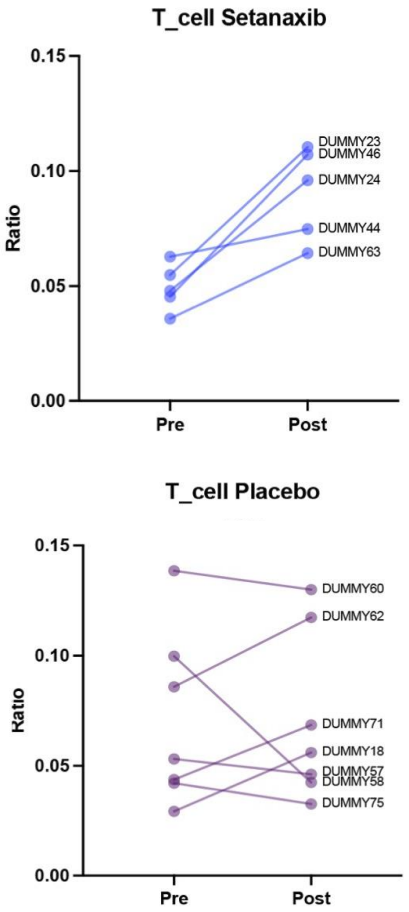
PFS: 7 of the 16 patients were progression-free with either stable disease or partial response (6 setanaxib arm: 1 placebo arm)

Biomarker Analysis: In a subset of 12 evaluable patients (tumor biopsies pre and post treatment) the transcriptomic analysis showed that the 2 top pathways impacted by the treatment were fibrosis-related signaling pathways (the IPF signaling pathway and hepatic fibrosis/stellate cell activation pathway), supporting setanaxib's modulatory effect on activated myofibroblasts

Pathology Analysis: Increased immunological activity within tumors of patients treated with setanaxib, with favorable changes in specific pathology markers Foxp3 and PDL-1 CPS

Full data readout expected 1H 2024 – basis for partnering discussions

Increased Number of T-Cells Penetrating Tumor in Patients on Setanaxib Treatment



PFS: Progression Free Survival, MoA: Mechanism of Action, IPF: Idiopathic Pulmonary Fibrosis, Foxp3: Forkhead Box Protein 3, PDL-1: Programmed Death-Ligand 1, CPS: Combined Positive Score

Press Release: Calliditas announces supportive interim data from Phase 2 head and neck cancer trial with lead NOX inhibitor candidate, setanaxib / Proprietary clinical data

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Preclinical Data in Alport Syndrome Supports Anti-Fibrotic MoA

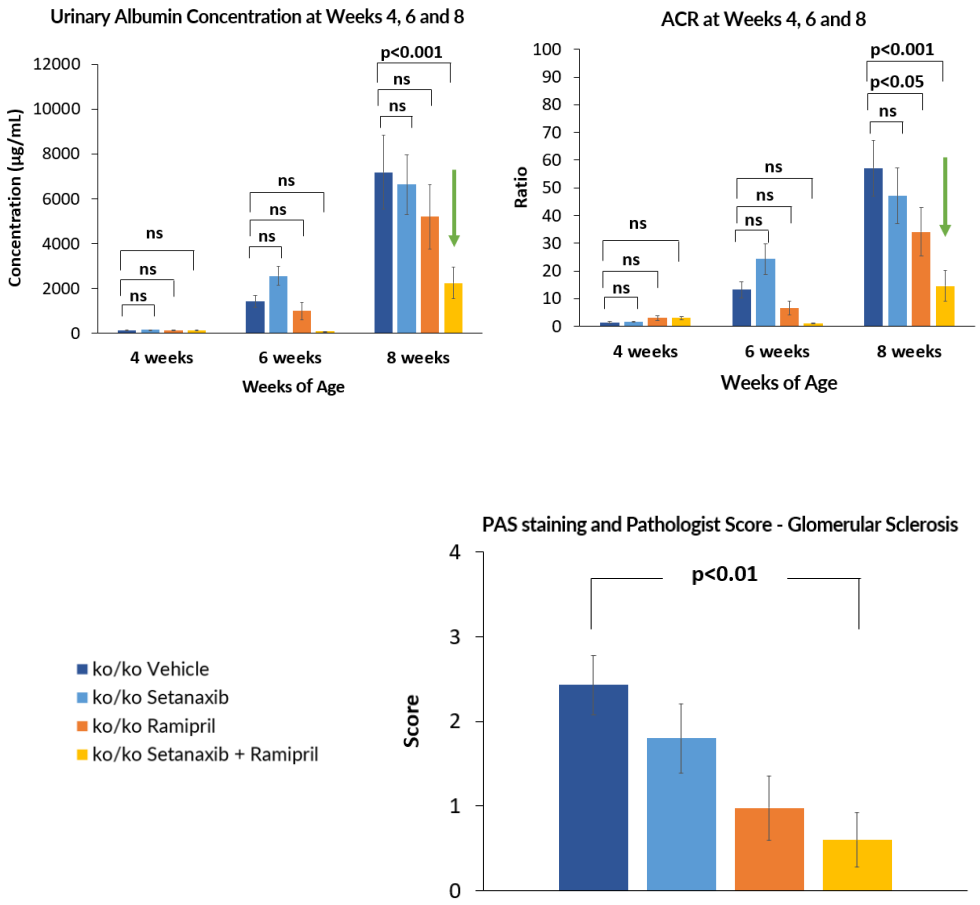
SETANAXIB + RAMIPRIL COMBINATION SIGNIFICANTLY REDUCED FIBROSIS AND THE RATE OF DECLINE IN GLOMERULAR FUNCTION

Alport Syndrome is a genetic rare disease where mutant type IV collagen proteins affect the integrity of basement membranes causing progressive kidney damage, hearing loss and visual problems

The Col4a3 KO mouse model (n=40) was used to assess the potential of setanaxib alone and in combination with ramipril (SoC)

Setanaxib + ramipril combination vs. placebo 8 weeks:

- Highly statistically **significant reduction in albuminuria and urine ACR**
- Statistically significant **reduction in glomerular sclerosis**
- *In silico* analyses showed increased glomerular basement membrane and collagen proteins



Note: Pathologist conducting scoring was blinded to treatment.
KO: Knock Out, SoC: Standard of Care, ACR: Albumin to Creatinine Ratio, PAS: Periodic acid-Schiff
Proprietary preclinical data extracted from ASN 20230 poster (TH-PO481). Christophe T. et al. The NOX inhibitor setanaxib combined with ramipril reduces glomerular function decline and fibrosis in a mouse model of Alport syndrome

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Summary of Setanaxib Platform Development

EXCITING YEAR AHEAD FOR DEVELOPMENT PROGRAM WITH DATA READOUTS AND PARTNERING POTENTIAL

Unencumbered first-in-class NOX1 and NOX4 inhibitor (NCE)

Pipeline in a product – in development for fibrotic rare diseases and solid tumors

Extensive safety dataset with >320 subjects exposed to setanaxib in completed Ph1 and Ph2 studies

Follow-on compounds available and in preclinical development

3x Ph2 data readouts in 2024 – Solid Tumors (SCCHN), PBC and IPF

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Thank You

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