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THERAPEUTICS

Corporate Presentation

March 2023

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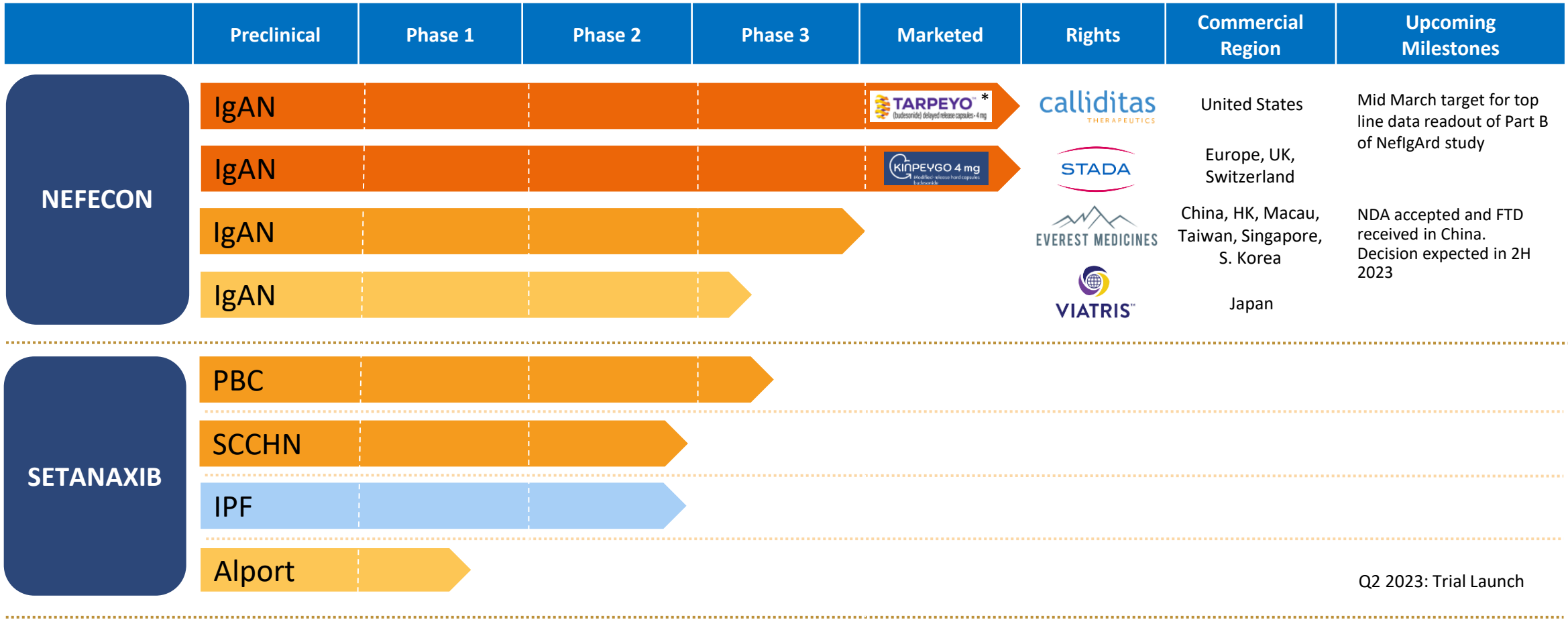
A commercial stage biopharma company focused on novel treatments in orphan indications, with an initial focus on renal and hepatic diseases with significant unmet needs

- **Pioneers in immunoglobulin A nephropathy (IgAN)**
  - Positive Phase 2b results published in The Lancet
  - Successful top-line data read out of the Phase 3 NefIgArd Trial
    - from Part A in November 2020, from Part B in March 2023
  - Both clinical trials met primary and key secondary endpoints
  - **Dec 2021 the first and only FDA approved medication for IgAN**, launched Jan 2022
  - **Jul 2022 the first and only EC approved medication for IgAN**, launched Sept 2022

- **Strong pipeline in orphan liver and kidney indications; platform of first-in-class NOX Inhibitors**
  - Actively expanding through in-licensing and/or acquisition of product candidates

Headquartered: Stockholm	
Exchange	OMX NASDAQ (CALTX)
	NASDAQ (CALT)
Key Institutional Shareholders	
BVF Capital Linc Polar Capital Sofinnova Partners	
Cash (31/12/22): **SEK 1,249m (~$119m)**	

Our Pipeline



Commercial: Ongoing Trial: Planned Trial: Investigator Led Trial:

* Approved under accelerated approval in the USA under the tradename TARPEYO. TARPEYO™ (budesonide) delayed release capsules is a prescription medicine used to reduce levels of protein in the urine (proteinuria) in adults with a kidney disease called primary immunoglobulin A nephropathy (IgAN) who are at high risk of rapid disease progression, generally UPCR ≥ 1.5g/g

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First Approved Medication for IgAN in Europe & USA

TARPEYO®
(budesonide) delayed release capsules • 4 mg

The first FDA approved drug specifically designed for Immunoglobulin A Nephropathy (IgAN)
Approved by FDA in December 2021; Launched in Jan 2022

KINPEYGO 4 mg
Modified-release hard capsules
budesonide

The first and only EMA approved drug specifically designed for IgAN
Positive CHMP Opinion in May 2022, European Commission approval in July 2022
Commercial Partner, STADA, received MA in September 2022 and launched in Germany in October 2022

Launched under an accelerated approval to reduce proteinuria in adults with IgAN at risk of rapid disease progression, generally a urine protein-to-creatinine ratio (UPCR) ≥ 1.5 g/g

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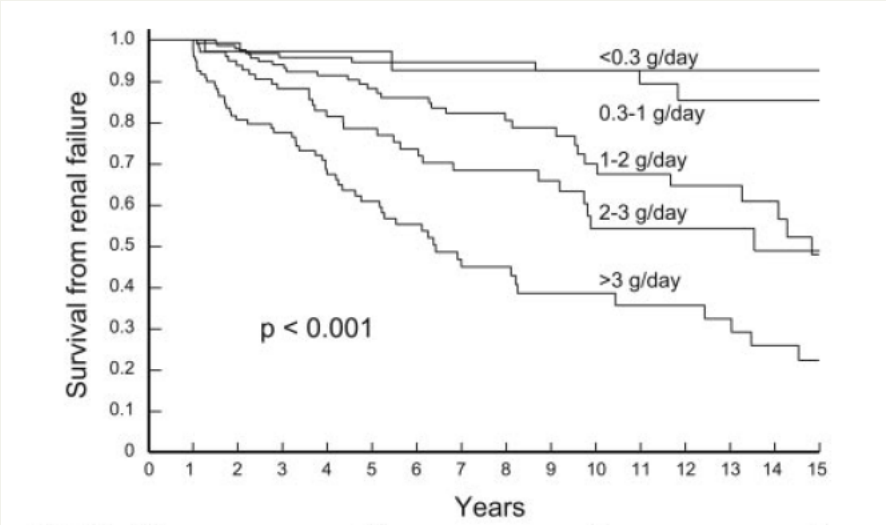
IgA Nephropathy – a significant unmet medical need

Profile

- Genetic predisposition is required but not sufficient; environmental, bacterial, dietary factors may play a role
- More than 50% are at risk of developing ESRD within 10-20 years, which can only be treated via regular haemodialysis or kidney transplant
- High levels of proteinuria is connected to disease progression and worse outcomes for patients



<0.3 g/day	37	22	8	1
0.3-1 g/day	134	79	35	11
1-2 g/day	145	79	28	10
2-3 g/day	105	50	18	4
>3 g/day	120	44	13	6

Figure 1. Renal survival by category of TA-proteinuria.

Estimated Prevalence

Market Opportunity

130,000 - 150,000

200,000

~5,000,000

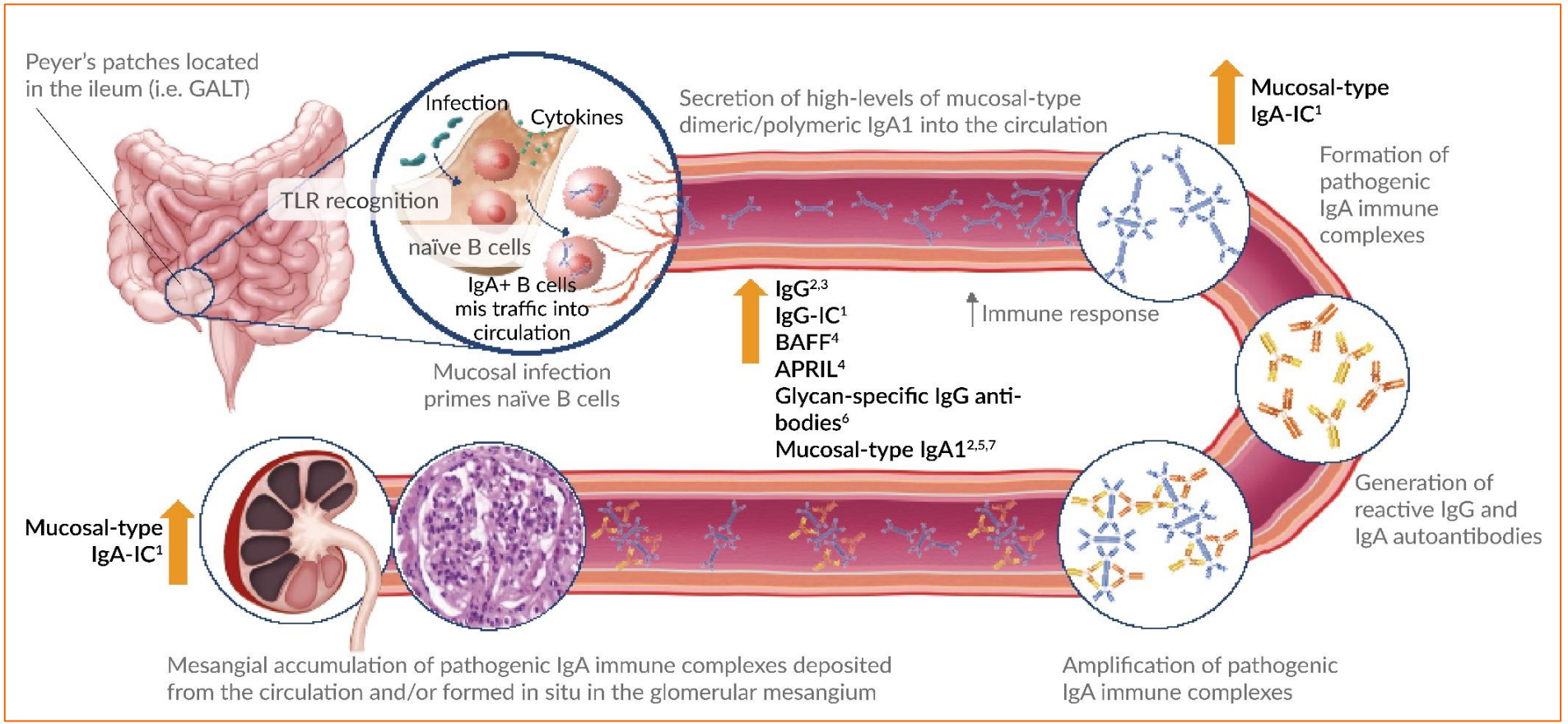
Pathophysiology of IgAN – predominant theory

Patients with IgAN have an increased appearance in the blood of secretory IgA antibodies, which are produced in the Peyer's patches, that lack galactose units in the hinge region

These galactose-deficient IgA antibodies are immunogenic, triggering IgA and IgG autoantibody production directed against the hinge region

The galactose-deficient secretory IgA antibodies form immune complexes with the IgA and IgG autoantibodies

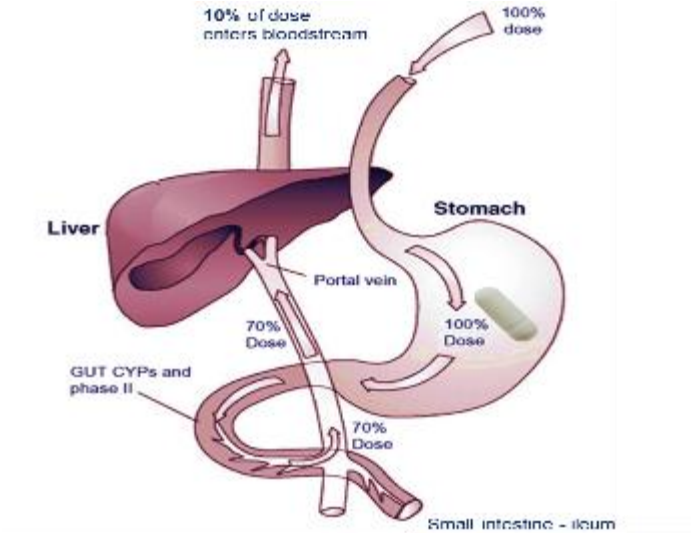
These immune complexes are deposited in the glomeruli of the kidney, causing an inflammatory cascade and destroying the glomeruli. This reduces the kidney's ability to remove waste products from the blood and eventually may result in ESRD



Designed to target the presumed origin of the disease

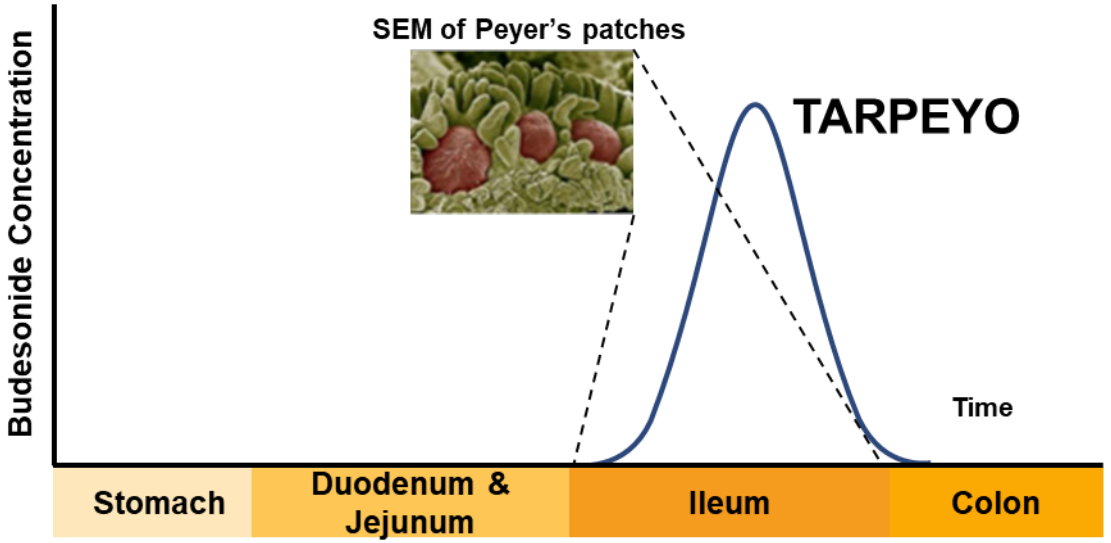
Drug product based on known active ingredient

- Active ingredient is budesonide – a highly potent, locally acting corticosteroid
- 90% cleared in first pass metabolism by liver, with the view of minimizing systemic side effects
- Safety profile as expected for oral administration; predominantly mild to moderate AEs, reversible upon discontinuation



Novel targeted release profile

- Designed to deliver a targeted and highly potent dose directly to Peyer's patches in the ileum
- Differentiated release profile
  - pH-governed delayed disintegration of the capsule until it reaches the ileum
  - Potent, sustained exposure throughout the ileum



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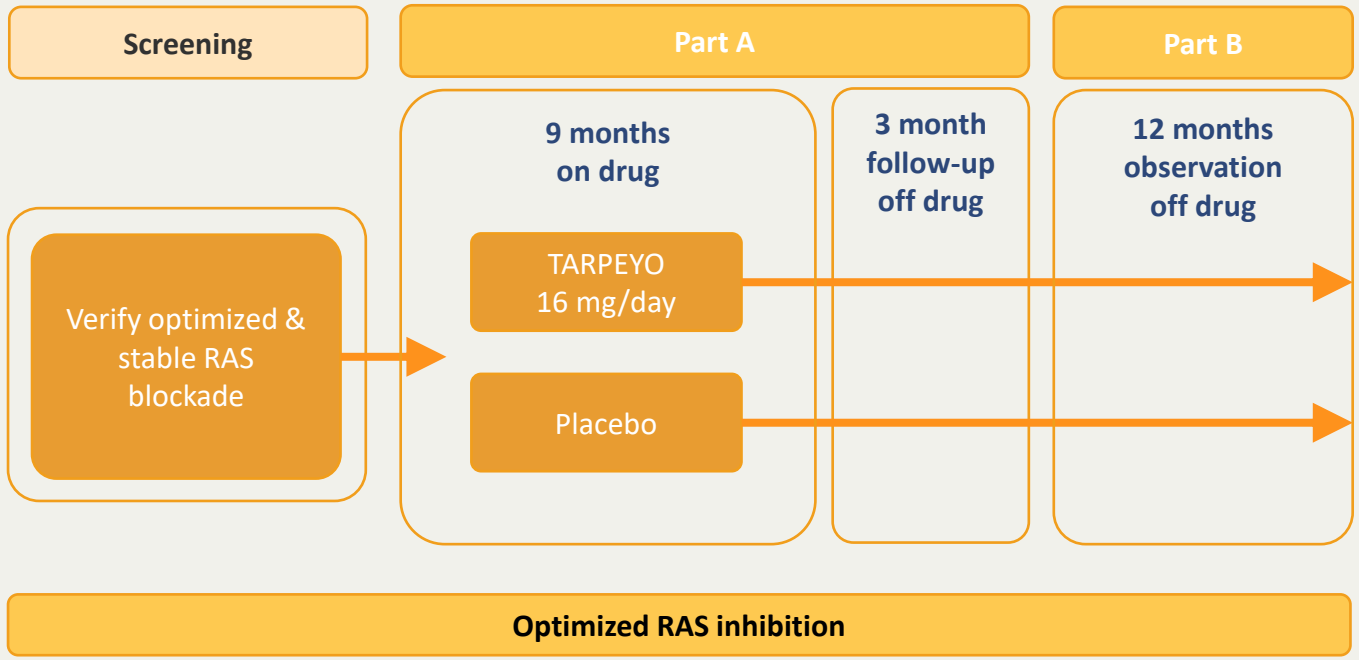
NefIgArd study design



Base inclusion criteria:

- Biopsy proven IgAN; > 1 gram of proteinuria; > 35 eGFR < 90 ml/min 36o patients, including 200 from Part A
- Patients were required to have well-controlled blood pressure of <140/90 mmHg to enter into the study, to ensure no BP confounding effects on proteinuria reduction.
- No immunosuppressive drugs were permitted during the study; changes to anti-hypertensive medications were discouraged.

Part A

- 200 patients in 19 countries with >145 sites
- Primary endpoint: proteinuria
  Key secondary endpoint: eGFR
- Read out positive data in November 2020

Part B

- Post approval follow up trial design
  - confirm the long-term renal benefit of observed proteinuria reduction
- 360 patients, including 200 from Part A
- Primary endpoint: difference in kidney function as measured by eGFR over the 2-year period
- Read out positive data in March 2023

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eGFR
Primary analysis of eGFR $AUC_{(0-2)}$
The Nef-301 Part B Primary Endpoint was met

- Over 2 years, eGFR was on average 5.05 mL/min/1.73 m^2 higher with Nefecon compared to placebo (p<0.0001)
  - Mean change in eGFR over the 2-year period was -2.47 mL/min/1.73 m^2 for Nefecon 16 mg versus -7.52 mL/min/1.73 m^2 for placebo

Nef-301 Primary analysis of eGFR $AUC_{(0-2)}$ (Full Analysis Set N=364)		
	Nefecon 16 mg (N=182)	**Placebo (N=182)**
eGFR $AUC_{(0-2)}$ (95% CI) [a]	-4.4% (-7.0% to -1.8%)	-13.5% (-15.8% to -11.1%)
Absolute change from baseline in eGFR over 2 years (mL/min/1.73 m^2)	-2.47 (-3.88 to -1.02)	-7.52 (-8.83 to -6.18)
Comparison: Nefecon 16 mg versus Placebo		
Percentage change in eGFR $AUC_{(0-2)}$ (95% CI); p-value	10% (6%, 15%); p<0.0001	
Absolute change (mL/min/1.73 m^2)	5.05	

[a] $AUC_{(0-2)}$ is a time-weighted average of eGFR observed at each time point over 2 years, with the treatment effect interpreted as the average effect of Nefecon over 2 years.

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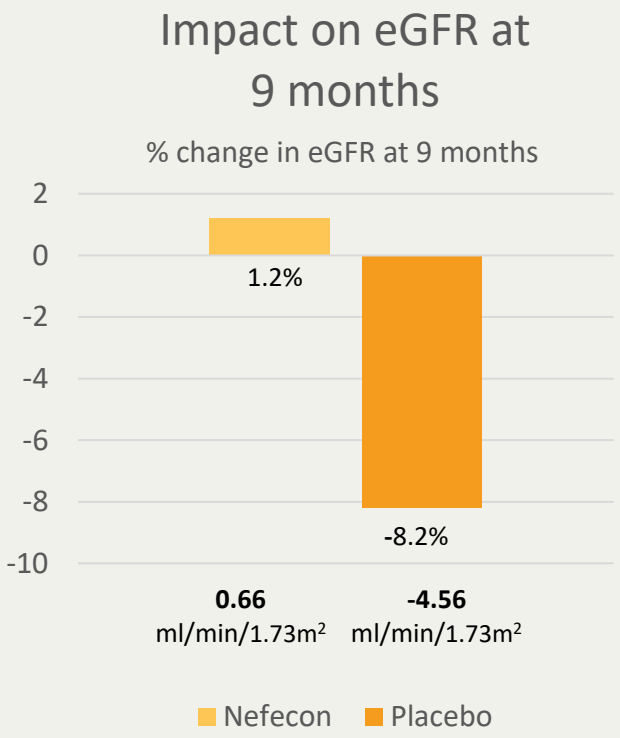
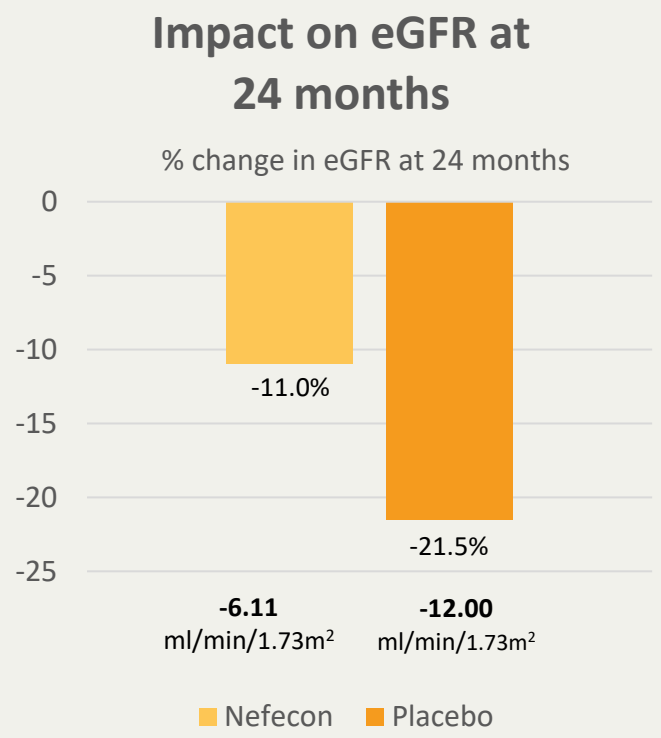
eGFR Phase 3 Data

Sustained eGFR effect observed at 24 months

9 months of dosing with 16mg Nefecon in 364 patients resulted in 50% less loss of kidney function vs placebo at 24 months.





Efficacy Findings

- Statistically significant eGFR stabilization with Nefecon (16 mg) compared to placebo following 9 months treatment ($p < 0.0001$)
- After 9 months:
  - eGFR increase for Nefecon treated patients: 0.66 ml/min/1.73m^2
  - eGFR decline for placebo: 4.56ml/min/1.73m^2
- After 24 months:
  - eGFR decline for Nefecon treated patients: 6ml/min/1.73m^2
  - eGFR decline for placebo: 12ml/min/1.73m^2

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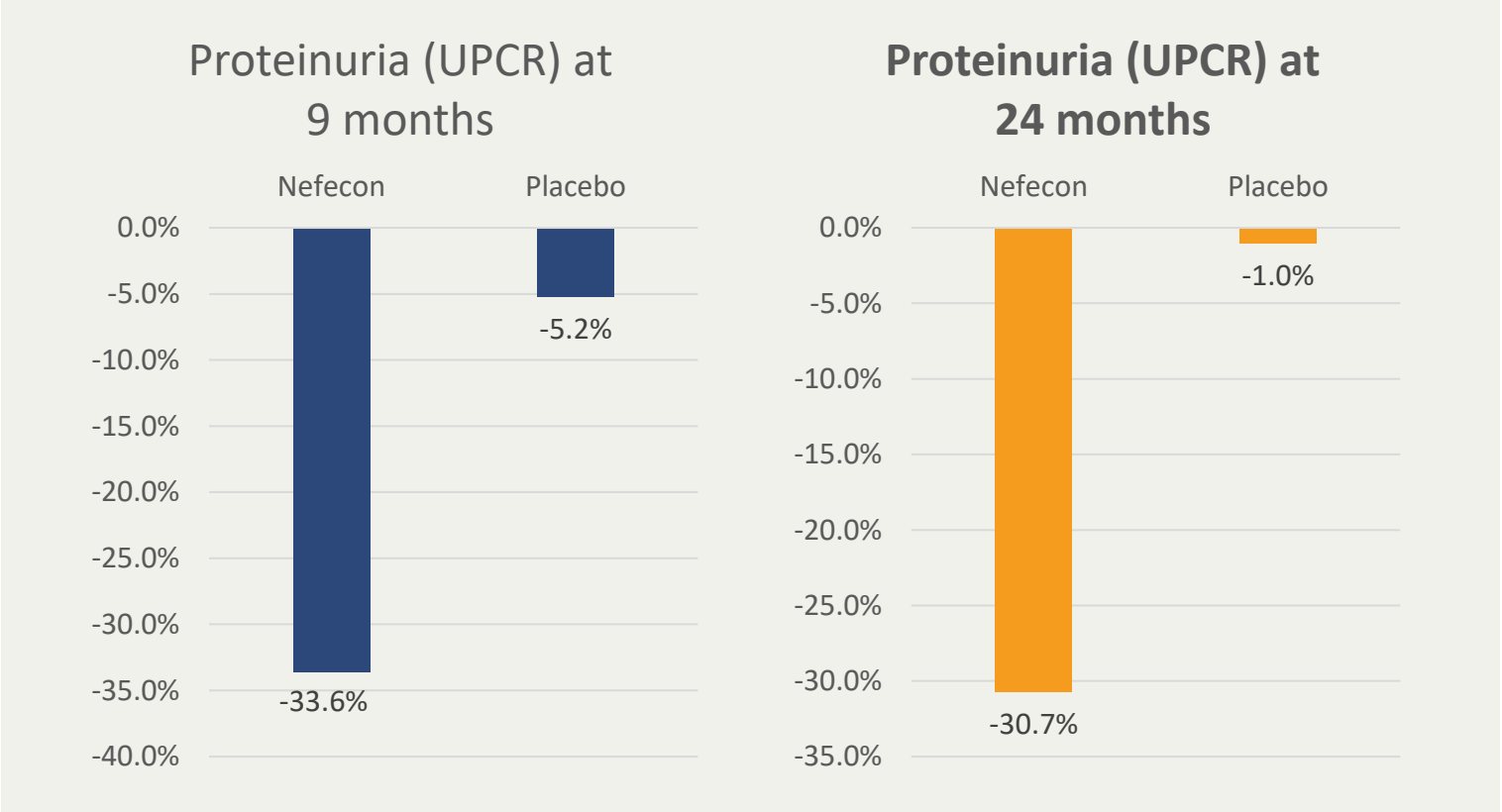
UPCR Phase 3 Data

Effect on UPCR maintained at 9 month level, or lower, from the end of treatment through 24 months

- The percent reduction in UPCR for Nefecon 16 mg versus placebo increased over time from 3 to 12 months, and thereafter returned to end of treatment (9 month) levels at the end of the follow-up period (15 months).



TARPEYO: A differentiated and targeted approach for IgAN

Targeted immunomodulator down regulating IgA1 at the source

- Proteinuria reduction vs SOC of physicians choice significant continued reduction off drug
- Designed to locally target origin of disease with the potential of being disease modifying
- Local action complemented by limited systemic exposure due to first pass metabolism of 90%

Patient centric

- Well characterized active ingredient and safety profile
- Option of intermittent, rather than chronic treatment
- Demonstrated impact not just on proteinuria but on eGFR

TARPEYO™ (budesonide) delayed release capsules

Established highly successful support service for frictionless access

- TARPEYO Touchpoints™: full-service patient and provider support program. Fully operational on day 1 of TARPEYO approval
- Utilizes Biologics by McKesson's PharmacyElite™ model; integrated HUB* and exclusive Specialty Pharmacy
- Staffed by Care Navigators: dedicated case managers + designated Rare Pod Team (nurses, pharmacists, fulfillment and distribution team)
- Integrated with a financial assistance (commercial co-pay) program provided by CoverMyMeds® from McKesson

*HUB: Allows a manufacturer to have a singular point of contact with patients. Services generally entail benefits investigation, prior authorization processing, drug delivery and administration support, financial and co-pay assistance, education, compliance with risk evaluation and mitigation strategies (REMS), data reporting, bridge supplies, and prescription triaging.

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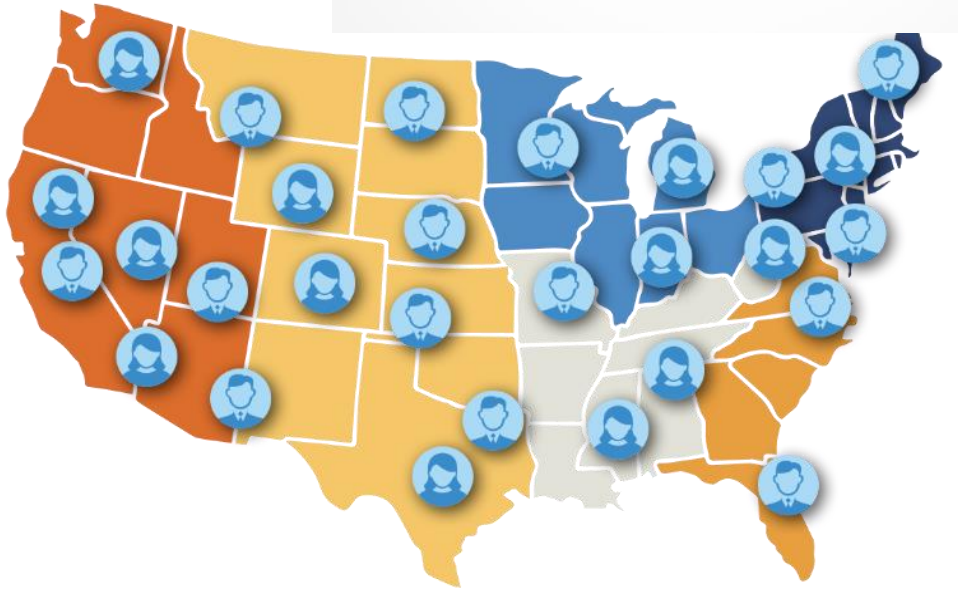
Strong US Commercial leadership team generating results

- Nephrologist awareness, now over 90%, peer to peer recommendations building
- Experienced sales force: 60 sales reps with core background in rare disease, specialty products and nephrology market
- Market access: Over 90% coverage of US lives achieved – over 70% already after 7 months
- Increasing clinical evidence available and positive outcomes for a growing number of patients taking TARPEYO



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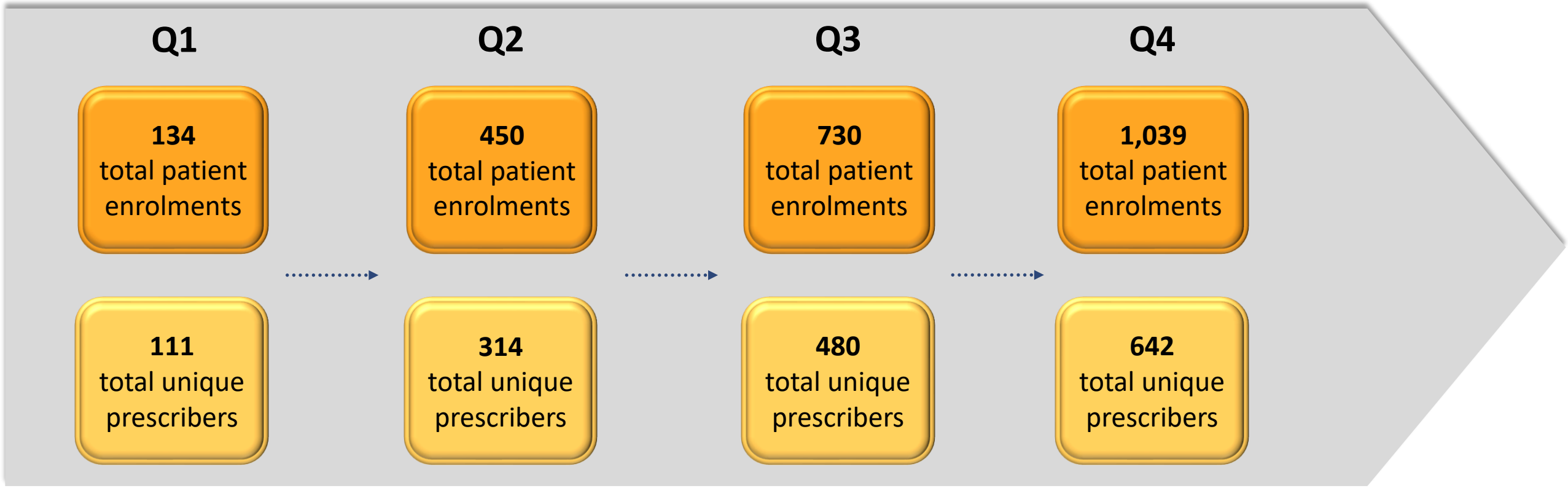
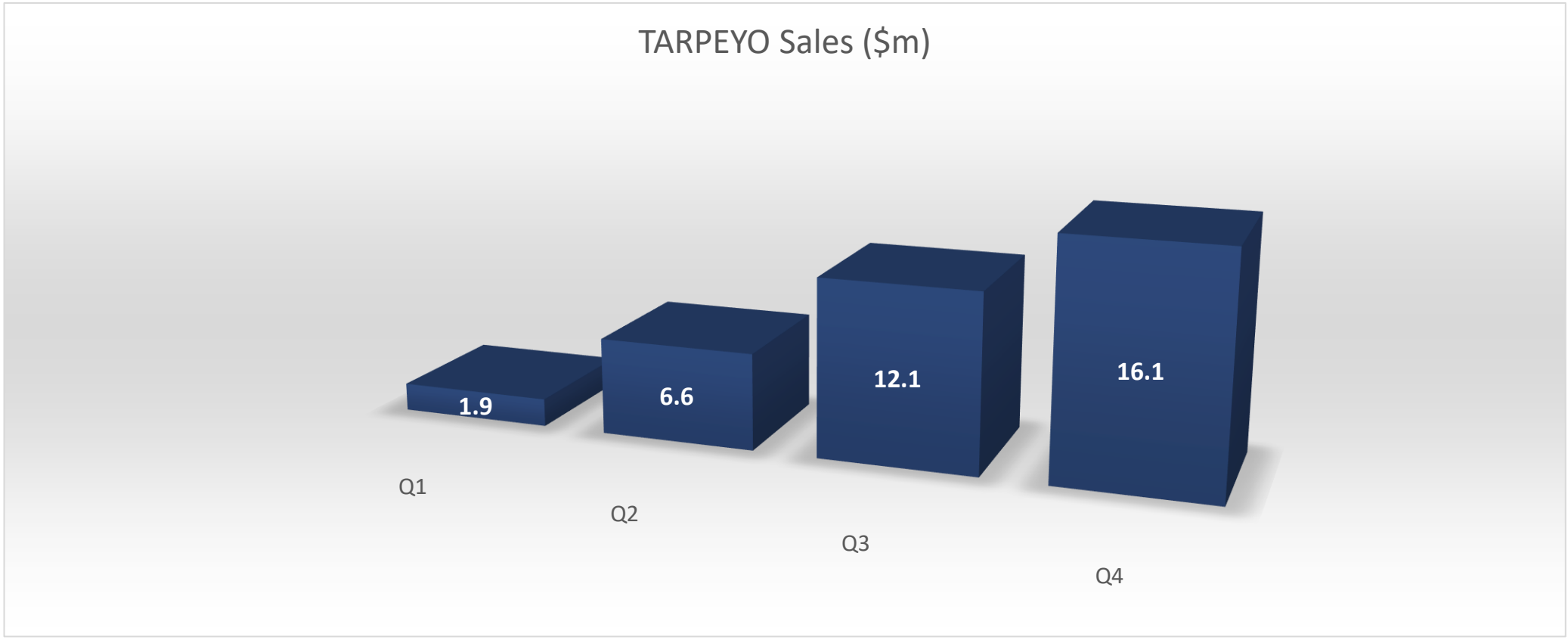
Successful launch reflecting receptivity and growth

- First sale and shipment of product took place January 28th, 2022



TARPEYO Sales

Total revenues from net sales of TARPEYO in 2022**: $36.8m**

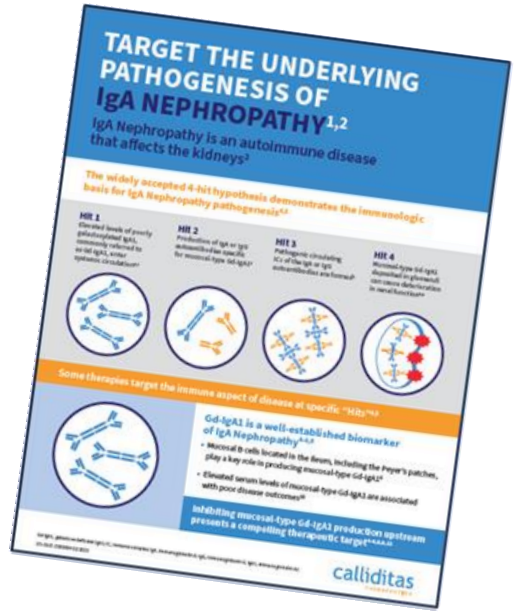


Financial Overview – Key Takeaways from 2022; significant growth!

- Total revenues of SEK 802.9 million ($79.3m) for the year of 2022 – 250% growth year over year
- TARPEYO net sales for 2022 amounted to $36.8m (SEK 372.2 million) for initial 11 months of commercialization
- Operating expense for the year amounted to SEK 1,210 M
- Positive Q4 cash flow from operations of SEK 230 M driven by partner payments and TARPEYO sales.
- Calliditas is well funded with a cash position of SEK 1,249 M as of end of December.
  - we believe that we are, based on our guidance for TARPEYO, funded to profitability and well prepared to capitalize on growth and opportunities in 2023.

What to look forward to in 2023 and beyond

- Increasing patient success stories from TARPEYO & Kinpeygo
- Full impact of expanded reach and growth of the US field team
- Release of additional NefIgArd related data; sustained impact on eGFR with TARPEYO® providing additional support for disease modification
- Decision regarding China regulatory approval of Nefecon
- Setanaxib data from Head and Neck cancer trial
- Initiation of clinical trial in Alport syndrome
- Continued commercial uptake of TARPEYO & Kinpeygo



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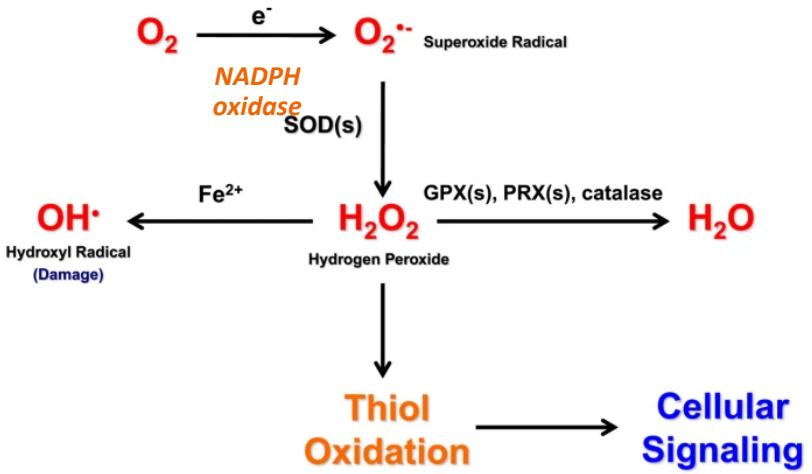
NOX Inhibitor Development Program

A first-in-class platform

Novel First-in-Class NOX1/4 Inhibitor Platform

- Setanaxib is the lead compound in the platform, and the **first NOX inhibitor to reach the clinical trial stage**
- NOX (NADPH oxidase) enzymes are dedicated to producing reactive oxygen species (ROS) which have a central role in cell signalling at appropriate concentrations



- When cell environmental stimuli change, **excess NOX activity** is triggered as part of the cell injury response. Redox homeostasis becomes unbalanced, triggering **fibrogenesis**
- **Cancer cells also induce NOX enzymes** in the microtumor environment, to create a favourable tumor growth and metastases

Sullivan, L.B., Chandel, N.S. Mitochondrial reactive oxygen species and cancer. Cancer Metab. 2014; 2 (17) https://doi.org/10.1186/2049-3002-2-17

Meitzler J.L., Makhlouf H. R., Antony S., Wu Y., Butcher D., Jiang G., Juhasz A., Lu J., Dahan I., Jansen-Dürr P., Pircher H., Shah A. M., Roy K., Doroshow J.H. Decoding NADPH oxidase 4 expression in human tumors. Redox Biology, 2017 (13): 182-195. https://doi.org/10.1016/j.redox.2017.05.016

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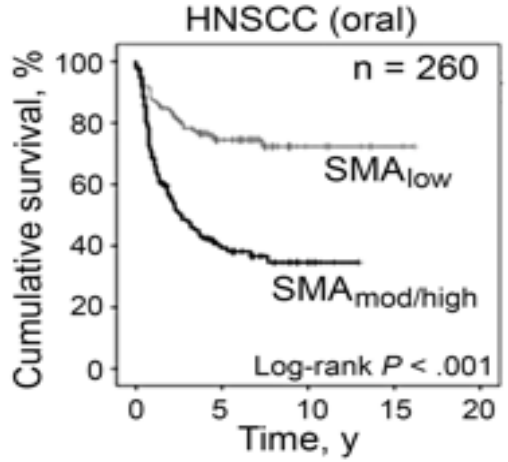
Scientific Rationale for Setanaxib in SCCHN

- The response to immuno-oncology therapies can be affected by the tumor microenvironment, in particular by the numbers of Tumor-Infiltrating Lymphocytes (TILs) and Cancer-Associated Fibroblasts (CAFs) present in the tumor
- A relationship between CAFs and prognosis in SCCHN has been established
- NOX4 is highly over-expressed in CAFs and drives myofibroblastic activation within tumors, shielding them from CD8+ TILs
- Inhibiting NOX1/4 expressed on with setanaxib could improve patients' responses to immunotherapies, and function as an adjunctive therapy
- There is increasing use of pembrolizumab as 1L monotherapy in patients with relapsed/metastatic SCCHN, although response rates are low (ORR approx. 20%)

Relationship between CAF Presence and Patient Prognosis



SCCHN: squamous cell carcinoma of the head and neck, SMA: smooth muscle actin (biomarker of activated CAFs), GKT: reference to inherited Genkyotex code for setanaxib (GKT137831)

Hanley C.J., Mellone M., Ford K., Thirdborough S. M., Mellows T., Frampton S. J., Smith D. M., Harden E., Szyndralewiez C., Bullock M., Noble F., Moutasim K.A., King E. V., Vijayanand P., Mirnezami A. H., Underwood T. J., Ottensmeier C. H., Thomas G. J. Targeting the Myofibroblastic Cancer-Associated Fibroblast Phenotype Through Inhibition of NOX4. J. Natl. Cancer Inst. 2018; 110 (1): 109-120 https://doi.org/10.1093/jnci/djx121

Mir S., Golden B.D.O., Griess B.J.,Vengoji R., Tom E., Kosmacek E.A., Oberley-Deegan R.E., Talmon G. A., Band V., Teoh-Fitzgerald M.L.T. Upregulation of Nox4 induces a pro-survival Nrf2 response in cancer-associated fibroblasts that promotes tumorigenesis and metastasis in part via Birc5 induction. Breast Cancer Res 2022 (24), 48. https://doi.org/10.1186/s13058-022-01548-6

Lin X.L., Yang L., Fu S.W., Lin W.F., Gao Y.J., Chen H.Y., Ge Z.Z. Overexpression of NOX4 predicts poor prognosis and promotes tumor progression in human colorectal cancer. Oncotarget. 2017;8(20): 33586-33600. doi: 10.18632/oncotarget.16829

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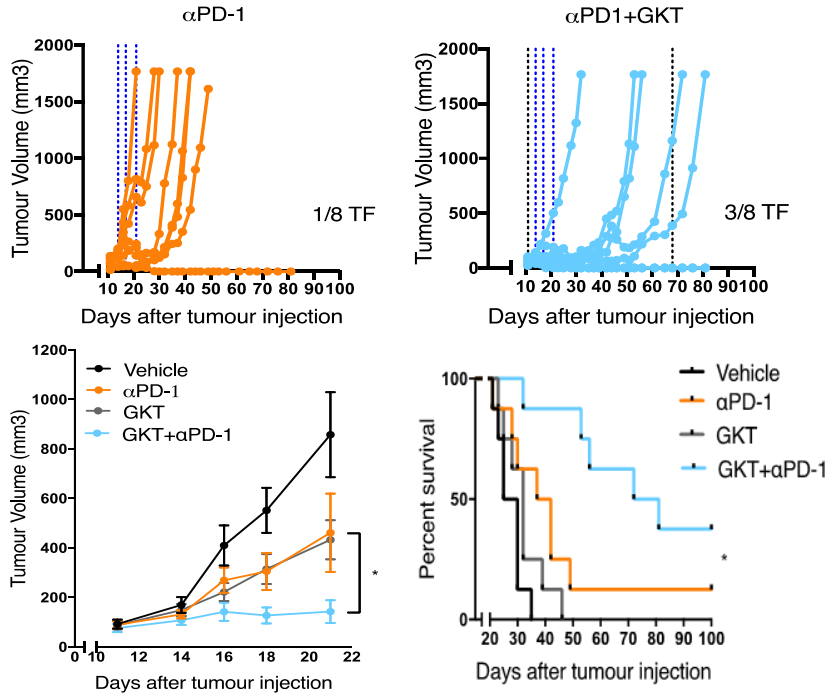
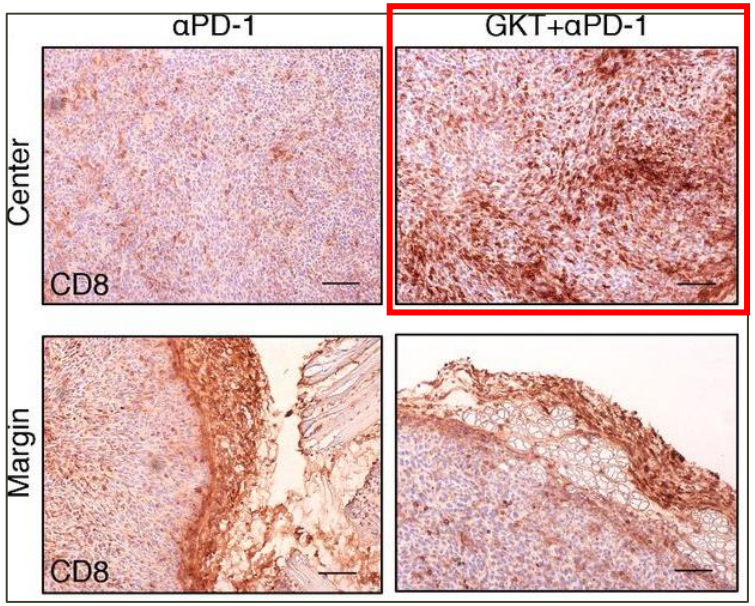
Preclinical Work: Combining Setanaxib with aPD-1 in CAF-Rich Tumors

- Using a CAF-rich tumor mouse model* administration of setanaxib + pembrolizumab vs. either treatment alone resulted in:
  - Improved penetration of TILs into the centre of the tumor
  - Slowing of tumor growth
  - Improved survival

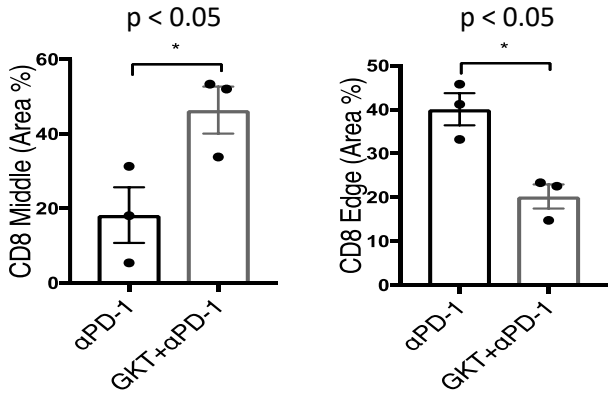
Tumor Growth & Survival



TIL Density Center vs. Margin



TIL Tumor Penetration



*Murine lung cancer cell line (TC1), murine colorectal cancer cell line (MC38), murine breast cancer cell line (4T1). GKT: reference to inherited Genkyotex code for setanaxib (GKT137831), αPD-1: anti-PD-1 antibodies, Bioxcell; RMP1-14, TF: treatment failures

Ford K., Hanley C.J., Mellone M., Szyndralewiez C., Heitz F., Wiesel P., Wood O., Machado M., Lopez M.A., Ganesan A.P., Wang C., Chakravarthy A., Fenton T.R., King E.V., Vijayanand P., Ottensmeier C.H., Al-Shamkhani A., Savelyeva N., Thomas G.J. NOX4 Inhibition Potentiates Immunotherapy by Overcoming Cancer-Associated Fibroblast-Mediated CD8 T-cell Exclusion from Tumors. Cancer Res. 2020; 80(9): 1846-1860. http://doi.10.1158/0008-5472.CAN-19-3158

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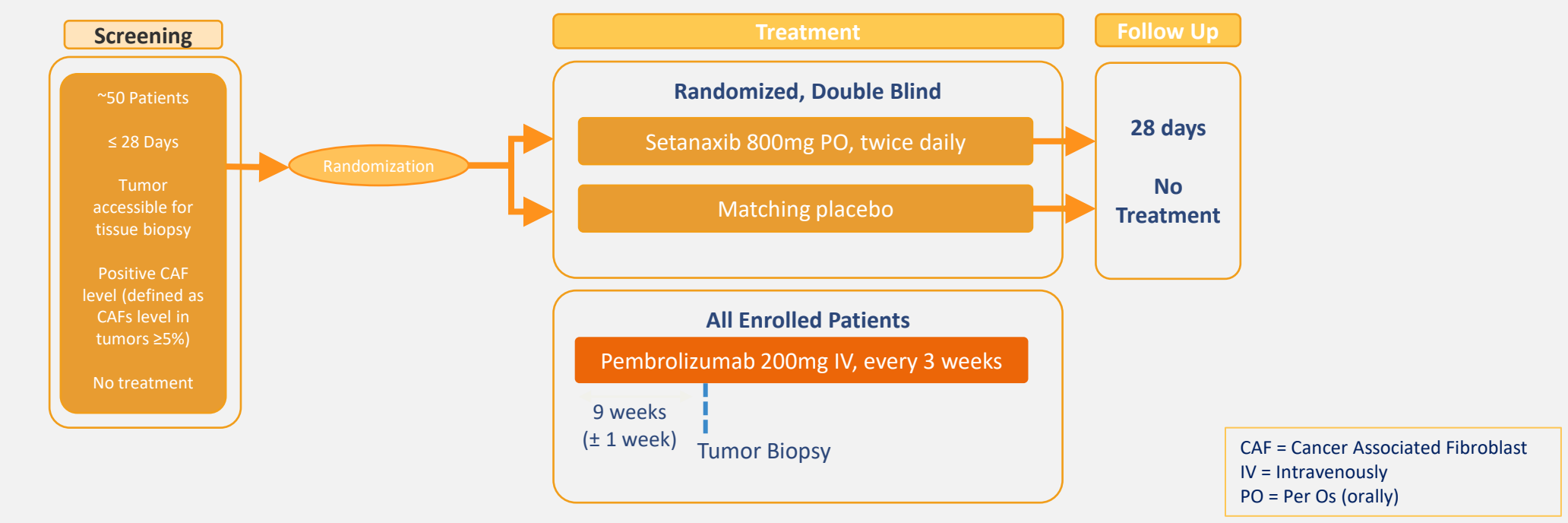
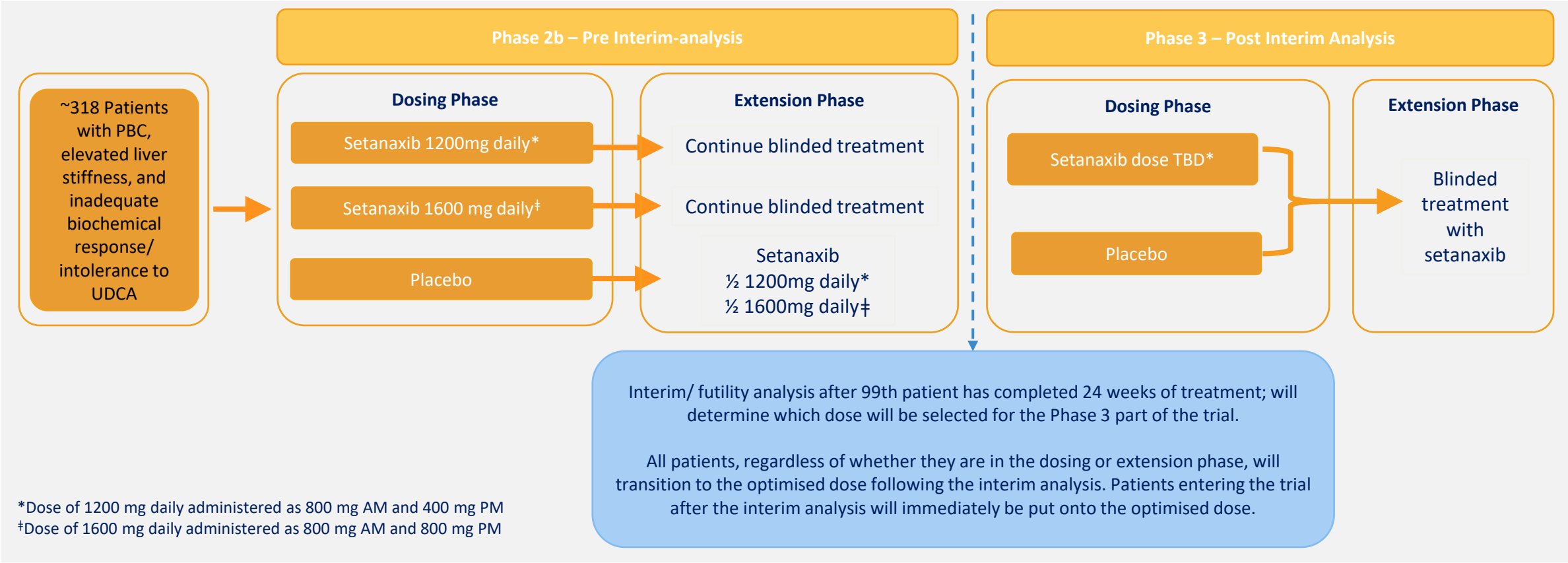
Phase 2 Proof-of-Concept Study in SCCHN

- Investigate the administration of setanaxib in conjunction with immunotherapy targeting CAFs
- The study will involve ~50 patients and investigate the change in tumor size in recurrent/metastatic SCCHN patients treated with either setanaxib + pembrolizumab, or placebo + pembrolizumab (NCT05323656)
  - Biomarker readout expected around middle of 2023



Setanaxib Ph2b/3 (TRANSFORM) in Primary Biliary Cholangitis (PBC)

Double-blind, randomized, placebo-controlled, adaptive study (NCT05014672) design. Calliditas received FDA fast track designation for setanaxib in PBC in August 2021



*Dose of 1200 mg daily administered as 800 mg AM and 400 mg PM
‡Dose of 1600 mg daily administered as 800 mg AM and 800 mg PM

Primary endpoint: ALP <1.67x ULN, and ALP reduction >15%, and total bilirubin <ULN
Interim analysis expected 1H 2024 (subject to recruitment)

Future Milestones

2022

- ☑ Commercial launch of TARPEYO in the US
- ☑ Initiate proof-of-concept Ph2 trial in SCCHN
- ☑ Positive EMA opinion for conditional approval for Kinpeygo and European Commission Approval
- ☑ Commercial launch in Europe
- ☑ Commercial ramp in US
- ☑ Regulatory filing in China

2023

- ☑ Positive readout of topline data from NeflgArd Part B
- ☐ Launch of setanaxib trial in Alport syndrome
- ☐ Readout of biomarker data from Ph2 POC trial with setanaxib in SCCHN
- ☐ Filing for full approval in Primary IgAN in the US and Europe
- ☐ Potential approval in China
- ☐ Commercial ramp in the US & Europe of TARPEYO & Kinpeygo respectively

2024

- ☐ Interim analysis of Ph2b/3 trial in PBC
- ☐ Final data readout of Ph2 POC trial with setanaxib in SCCHN
- ☐ Commercial ramp in China by Everest Medicines
- ☐ Potential full approval in IgAN in US and Europe

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