



Corporate Presentation

July 2022





Forward Looking Statement

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Diversified Pipeline Against Well-Validated Targets in Areas of High Unmet Medical Need

PROGRAM	CLASS (Route of Admin.)	INDICATION						RIGHTS
			RESEARCH	PRE-CLINICAL	PHASE 1	PHASE 2	PHASE 3	
Seralutinib (GB002)	PDGFR, CSF1R, c-KIT Inhibitor (Inhaled)	Pulmonary Arterial Hypertension (PAH)						WW
GB5121	CNS-Penetrant, BTK Inhibitor (Oral)	Primary CNS Lymphoma (PCNSL)						WW
GB7208	CNS-Penetrant, BTK Inhibitor (Oral)	Multiple Sclerosis (MS)						WW
Research Programs	Multiple Programs	Oncology, Immunology						WW

Seralutinib (GB002)

Inhaled PDGFR, CSF1R & c-KIT Inhibitor

Pulmonary Arterial Hypertension (PAH)



Seralutinib (GB002): Potential To Deliver Disease-Modifying Effects to Patients with PAH

Product Candidate Description

- Selective, inhaled PDGF receptor, CSF1R, and c-KIT kinase inhibitor designed to address the disease pathogenesis of PAH
- Kinase inhibition was shown to be clinically significant in Phase 3 PAH trial of imatinib (Gleevec), with systemic toxicities (IMPRES Study)
- Seralutinib formulation and administration via dry powder inhaler (DPI) designed to reach areas of the deep lung; DPI device is small, convenient and currently used in commercial products
- Designed to deposit inhaled seralutinib at site of disease due to proximity of terminal bronchiole and alveolar space to affected pulmonary arteries
- Patent protection to 2039⁽¹⁾; Orphan Drug Designation from FDA and EMA



1) Includes assumption of maximum 5 years of patent term extension. Corresponding patent(s) and patent application(s) with compound-specific claims. Total patent life with patent term extension cannot exceed 14 years from approval.

PAH Has High Unmet Need & Significant Disease Burden

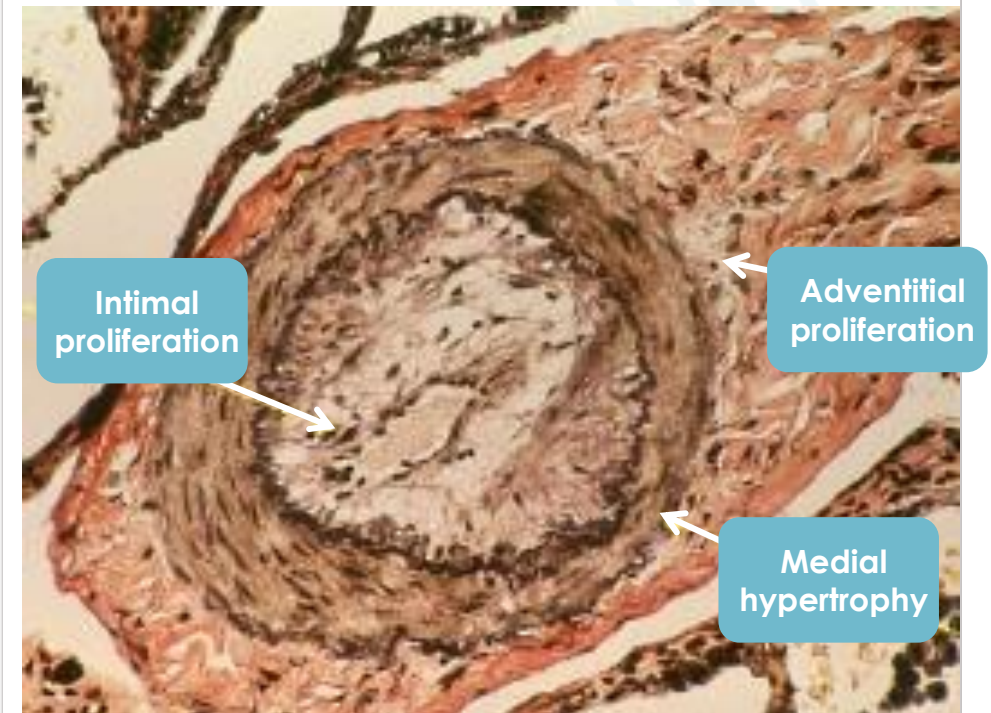
Pulmonary Arterial Hypertension (PAH)

- Rare, orphan disease
- Characterized by high blood pressure in the blood vessels carrying deoxygenated blood from the right side of the heart to the lungs
- Caused when the arteries in the lungs become narrowed, thickened and / or stiff as a result of pathological remodeling and vasoconstriction
- Heart works harder to pump blood to the lungs, potentially leading to right heart failure
- Progressive disease and often fatal

Symptoms

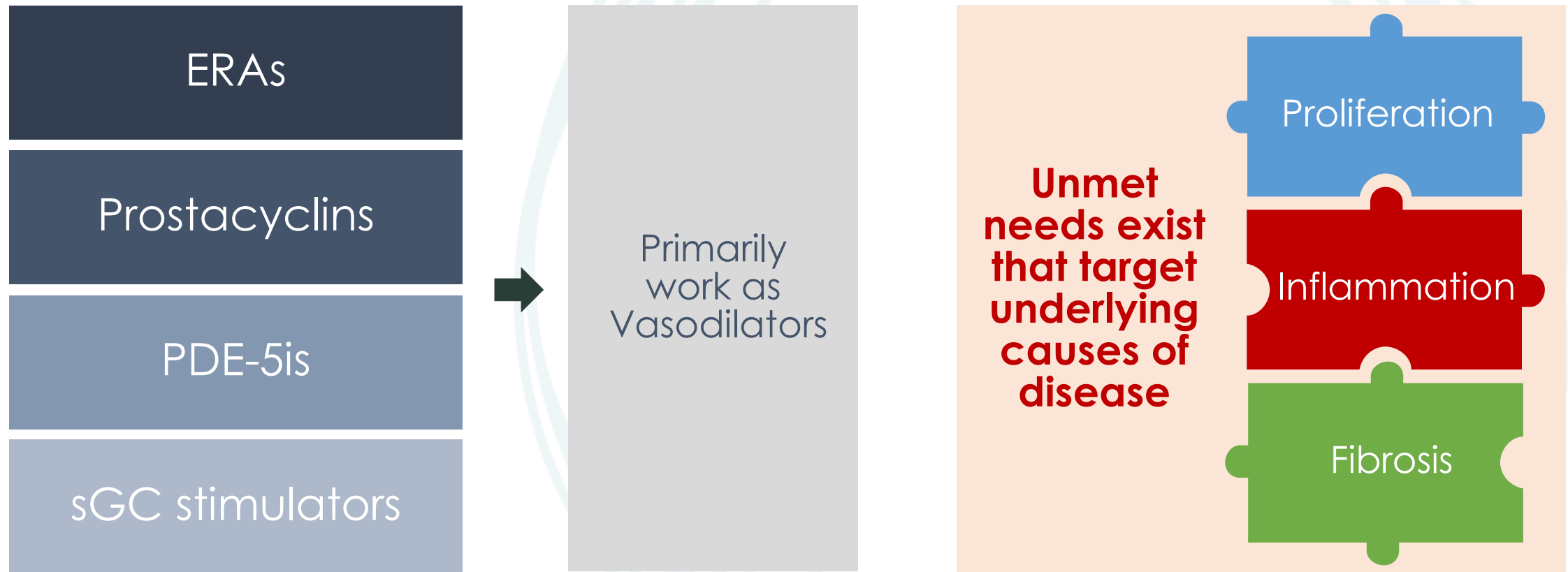
- Dyspnea
- Fatigue
- Dizziness
- Chest pressure / pain
- Edema in ankles, legs, abdomen
- Cyanosis
- Heart palpitations

PAH is Characterized by Vascular Remodeling



Muscular pulmonary artery from iPAH patient¹

What Do Currently Available Therapies Do?



In the Phase 3 IMPRES Study of Imatinib in PAH, Safety Liabilities Outweighed Clinically Meaningful Efficacy



Clinical Efficacy Results

Phase 3 IMPRES Study

- Primary Endpoint:
 - 6-Minute Walk Distance (6MWD)
 - 32-meter improvement (pbo-adj.)*
- Secondary Endpoint:
 - Pulmonary Vascular Resistance (PVR)
 - 32% reduction (pbo-adj.)*

Clinical Safety / Tolerability

Phase 3 IMPRES Study

- Adverse Events:
 - High rate of GI side effects
 - 44% SAE rate for imatinib group
 - 8 subdural hematomas across study and extension
- Discontinuations:
 - 33% for imatinib group, with most occurring in first 8 weeks of trial

Source: Hooper, Marius et al. "Imatinib mesylate as add-on therapy for pulmonary arterial hypertension: results of the randomized IMPRES study." Circulation 127, no. 10 (2013): 1128 – 1138.

*Statistically significant result.

SAE = serious adverse event; pbo = placebo; pbo-adj. = placebo-adjusted; GI = gastrointestinal.

Seralutinib Employs Multiple Strategies to Mitigate Imatinib's Liabilities

Molecule Specifically Designed for PAH

- Imatinib was developed & approved as an anti-cancer therapy
- Seralutinib utilized Phase 3 IMPRES learnings and targets underlying biology of PAH, including PDGFR α/β , CSF1R and c-Kit
- Seralutinib avoids c-ABL inhibition

Improved Selectivity Against Targets of Interest

- Increased potency* across target kinases v. imatinib
 - Increased potency against the PDGFR α isoform
 - Greater than ten-fold higher potency against PDGFR β , c-Kit, and CSF1R

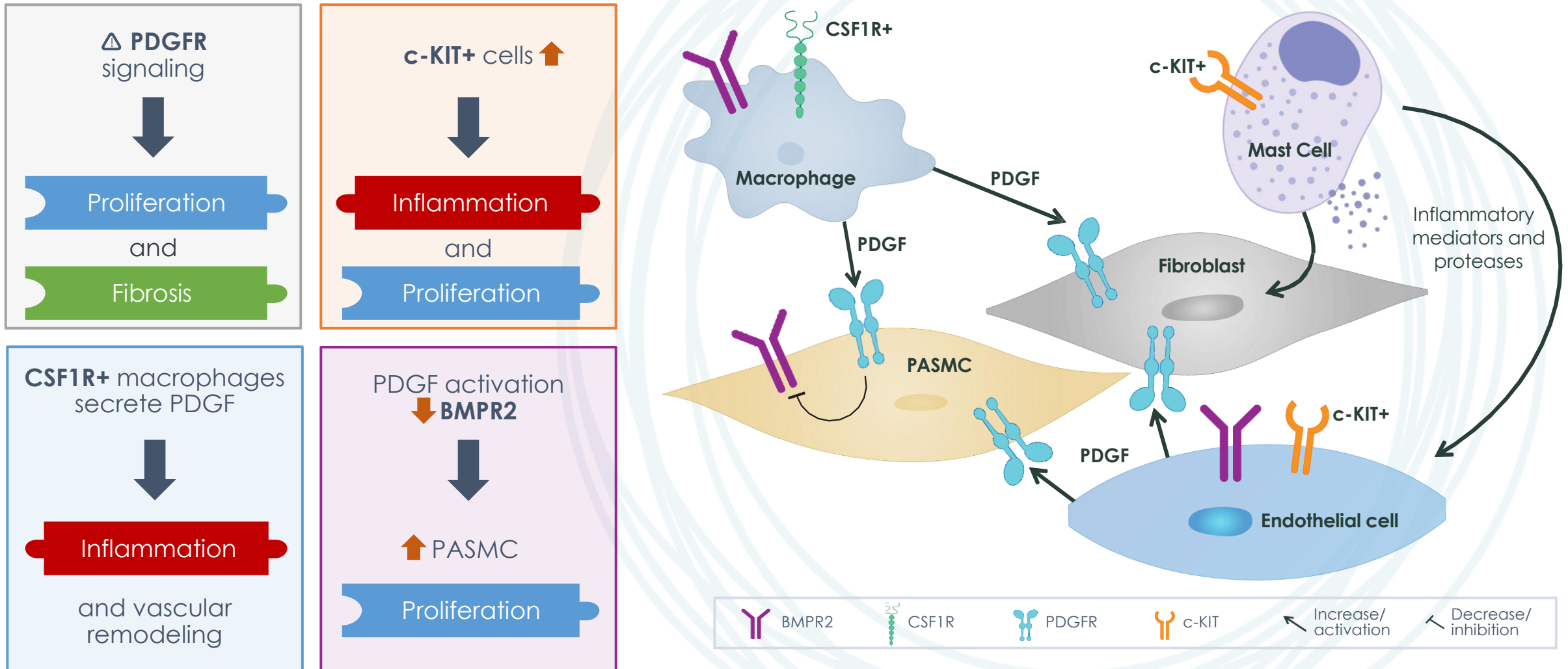
Designed for Inhalation

- Inhalation limits systemic exposure to mitigate systemic AEs, while directly getting drug to site of disease
- As part of inhalation process, some drug product is inevitably swallowed
 - Swallowed / ingested drug can enter systemic concentration
 - Seralutinib designed to have limited oral bioavailability (~5%)

*Based upon cell based IC50 assays. Data on hand.
AE = adverse event.

Contributing Factors to Vascular Remodeling

Role of PDGFR, CSF1R, c-KIT and Interactions with BMPR2

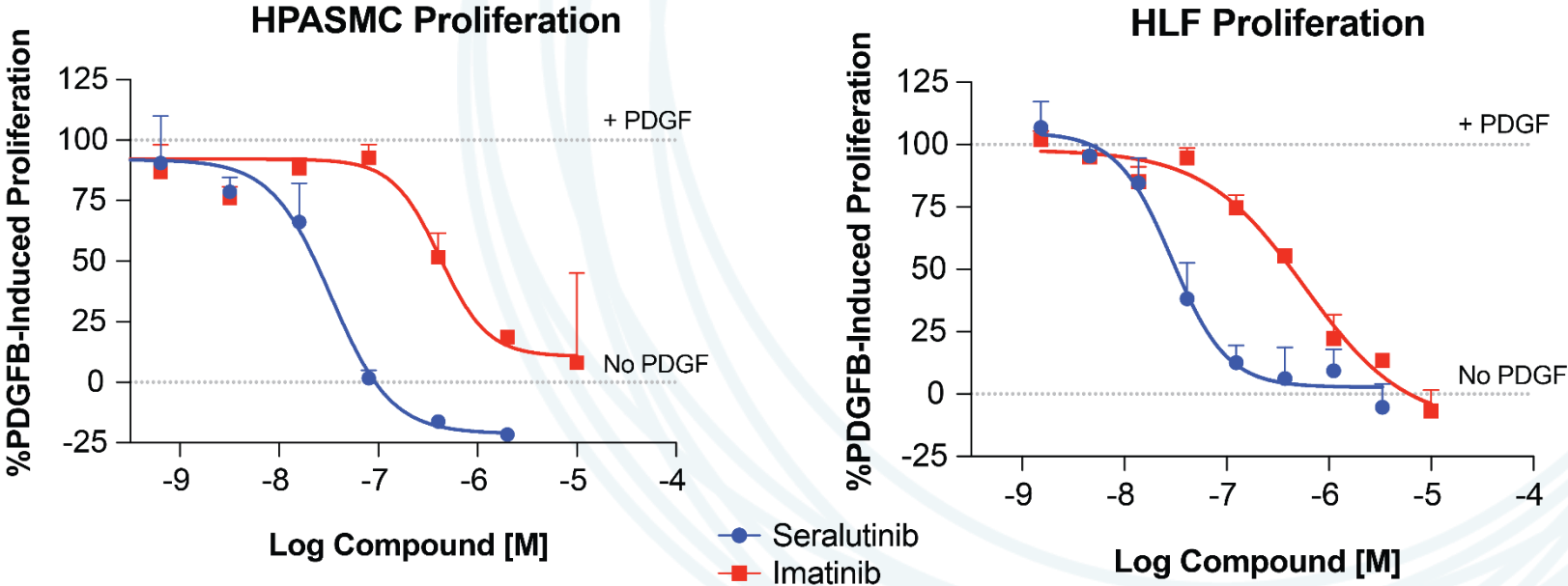


Seralutinib In Vitro Profile

Seralutinib is a potent PDGFR, CSF1R and c-KIT inhibitor

Compound	Cell Based IC50 (nM)				
	H1703 PDGFR α	HLF PDGFR $\beta > \alpha$	PASMC PDGFR $\alpha = \beta$	CSF1R	c-KIT
Seralutinib	32	29	33	8	14
Imatinib	62	579	419	1032	230

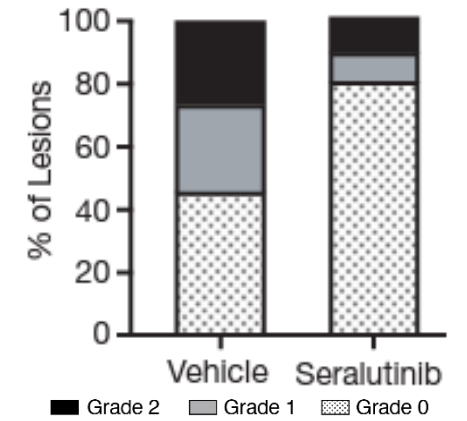
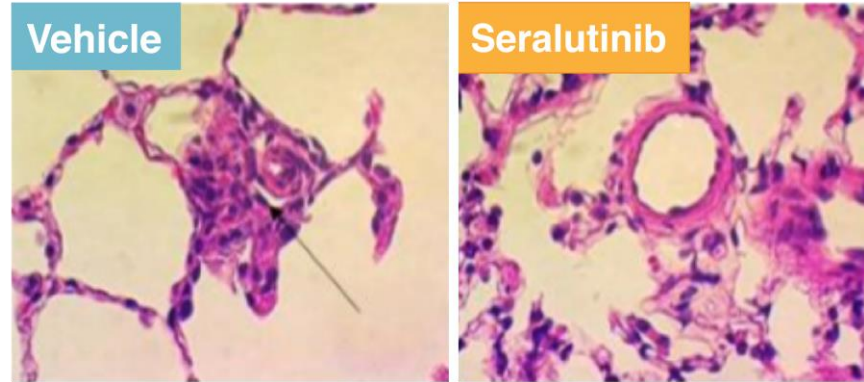
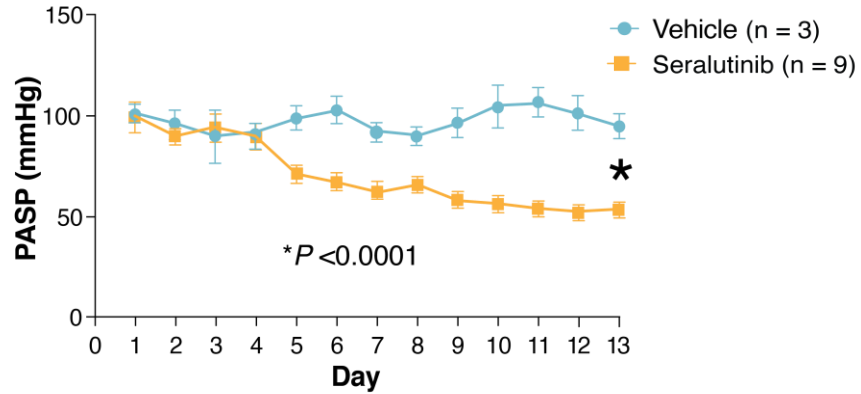
Seralutinib is highly potent in PASMC and HLF proliferation assays



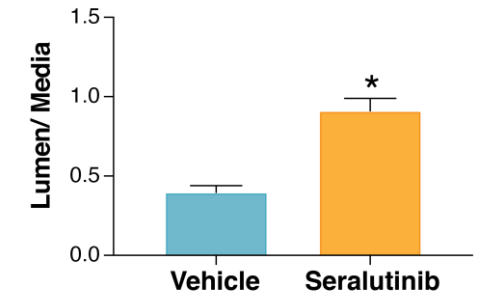
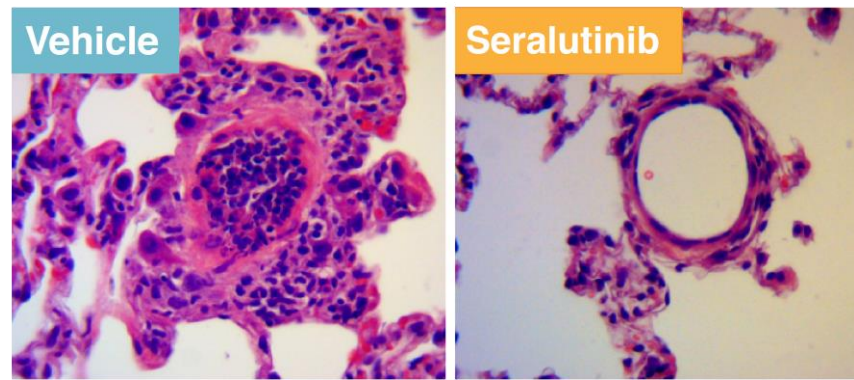
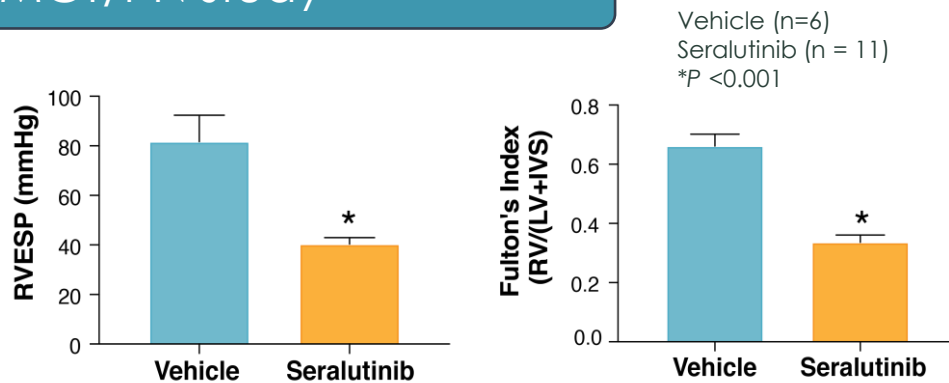
HPASMC: human pulmonary arterial smooth muscle cell; HLF: human lung fibroblast
Source: Ten Freyhaus, Arterioscler Thromb Vasc Biol 2015, 35(5):1236; Barst, J Clin Invest 2005, 115(10):2691; Gomez-Arroyo, et al, Am J Physiol (2012) 302(10):L1014; Sawada, et al, J Exp Med (2014) 211 (2):263; Talati, et al, PLoS One (2014) 9(4):e94119; Abid, et al, Eur Respir J 2019, 10:54(4):1802308; Savai, et al., Am J Respir Crit Care Med 2012, 186(9):897; Montani, et al., Am J Respir Crit Care Med 2011, 184(1):116

Seralutinib Demonstrates Efficacy in the SU5416/Hypoxia and MCT/PN Models

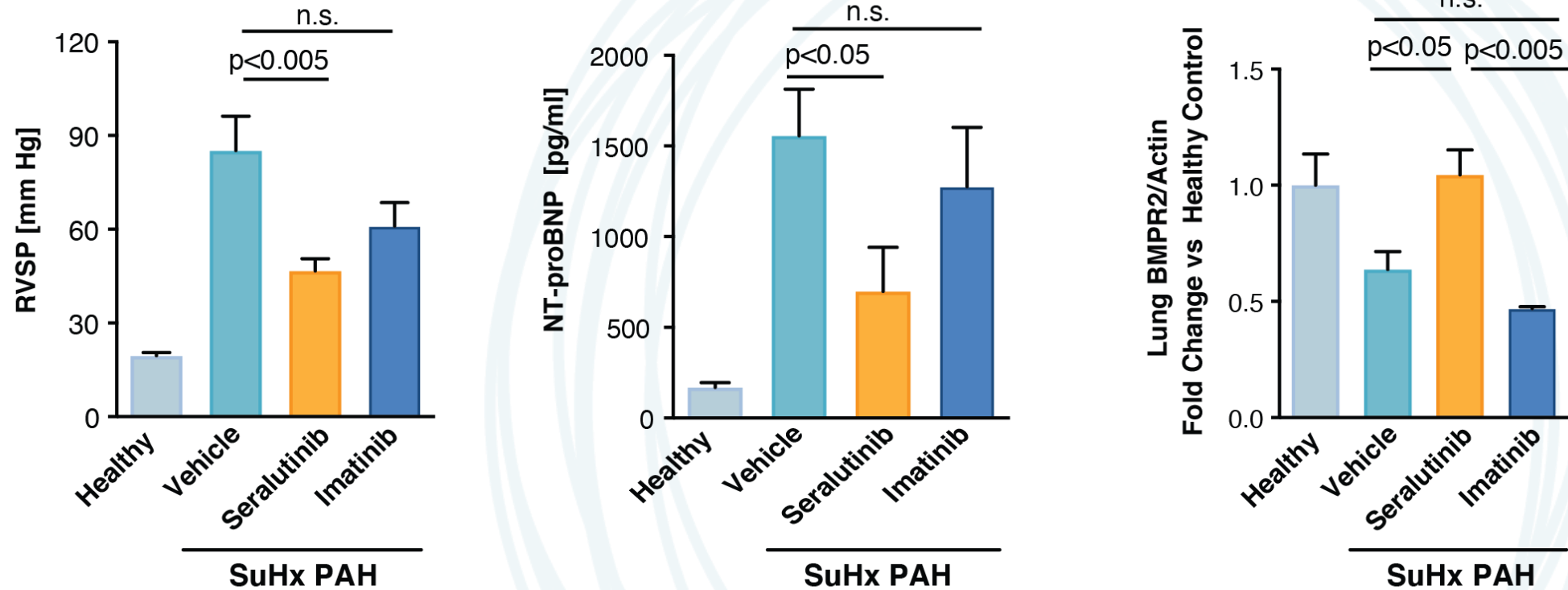
SU5416/H telemetry study



MCT/PN study



Inhaled Seralutinib Outperformed Oral Imatinib in a Head-to-Head Preclinical SuHx PAH Study



Data presented as Mean +/- SEM. Statistical analysis was performed using one-way ANOVA with Dunnett's multiple comparisons test (Healthy n=8; Vehicle n=7; Seralutinib n=9; Imatinib n=7)

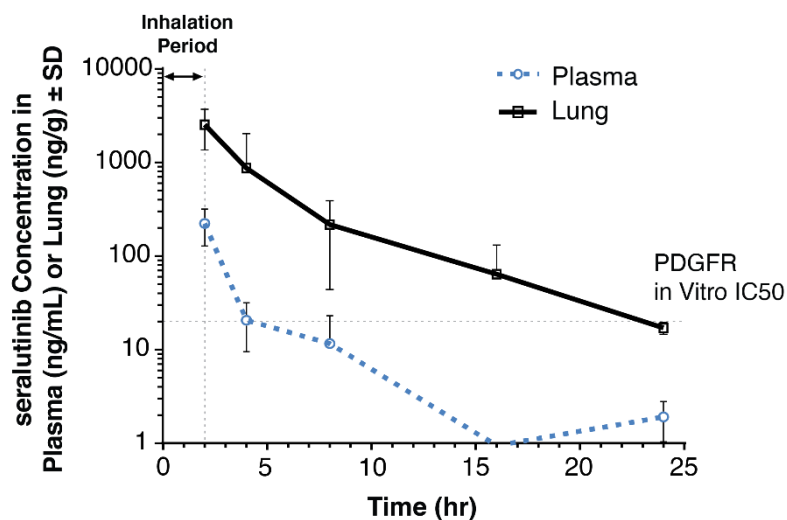
- Seralutinib treatment led to a significant improvement in RVSP
- Seralutinib reduced circulating levels of NT-proBNP and increased lung BMPR2 protein expression

Seralutinib Utilizes Convenient Dry Powder Inhaler



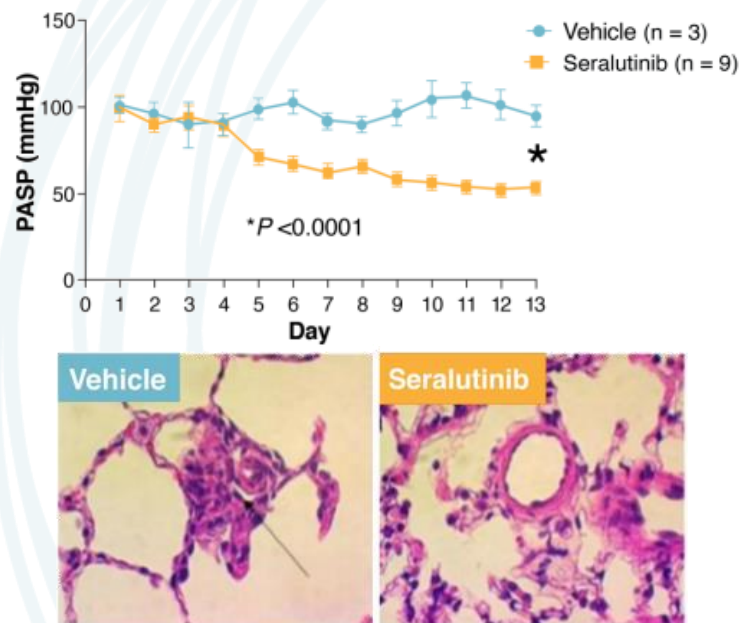
Gossamer Took A Systematic Approach to Arrive at 45 – 90mg BID Dosing for Seralutinib

Seralutinib Designed to be an Inhaled Therapeutic



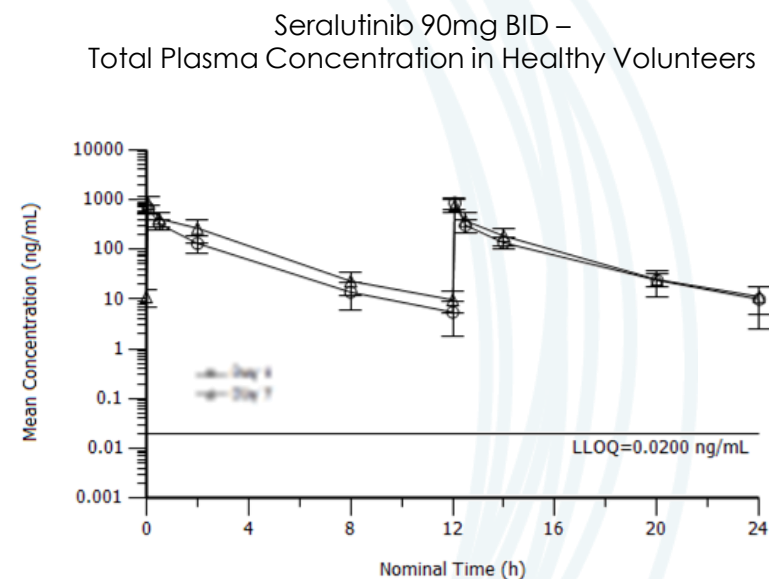
- ✓ Much greater (~30x mean) lung-to-plasma ratio and lung half life (~6 hours vs. 3 hours in plasma) observed in rats

Human Dose Scaled to Efficacious Results in Rats



- ✓ Allometric and direct scaling for inhalation products[†] suggests ~12.8 mg/kg BID dose efficacious in SuHx PAH model translates to ~90 mg BID in humans

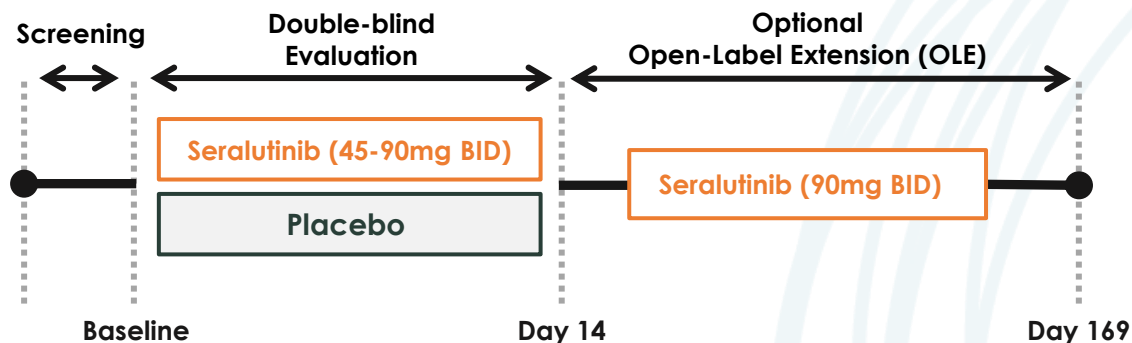
Phase 1 PK Confirms Scaling and IC50 Coverage of Target Kinases



- ✓ 45mg and 90mg BID doses selected to maintain IC50 coverage in the lung above the IC50 values of PDGFR α , PDGFR β , c-KIT, and CSF1R based on Phase 1 NHV data

[†]Allometric and direct scaling approaches as described in JE Philips; Pharmacology & Therapeutics, 178 (2017) Therese Ericsson et al; Pharm Res, July 2017
BID: twice daily; SD: standard deviation; PASP: pulmonary arterial systolic pressure; SuHx: sugen hypoxia; NHV: normal healthy volunteers

Overview of Phase 1b Study in Patients with PAH



- First subjects enrolled Q1:20; study interrupted by COVID-19 related site closures in the spring of 2020, limiting ability for patients to continue treatment on OLE
- Site re-openings in the fall of 2020 allowed enrollment of additional subjects, including opportunity for roll-over to the OLE
- A total of 8 subjects enrolled and completed the double-blind period (6 Seralutinib, 2 Placebo) with 2 subjects continuing and completing the OLE

Study Objectives

Primary

- To evaluate the safety and tolerability of inhaled seralutinib

Secondary

- To evaluate the pharmacokinetics (PK) of seralutinib

Exploratory

- To evaluate pharmacodynamic (PD) biomarker on blood samples

Inclusion Criteria and Dosing

Key Inclusion Criteria

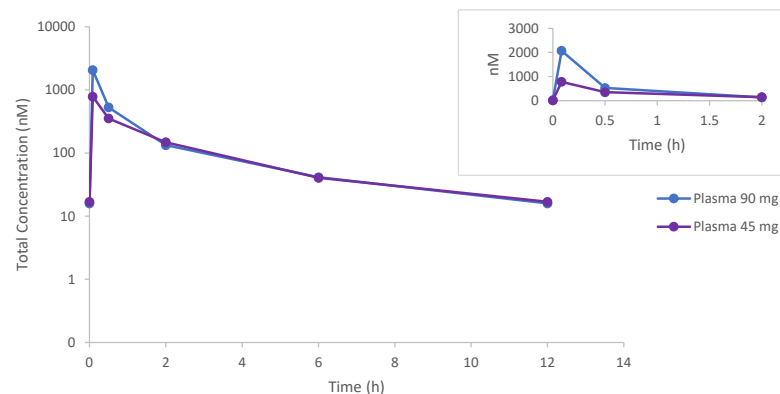
- Diagnosis PAH, WHO group 1, FC II-IV
- Prior cardiac catheterization data c/w PAH
- Baseline 6-minute walk >100 m
- On PAH background medications

Dosing

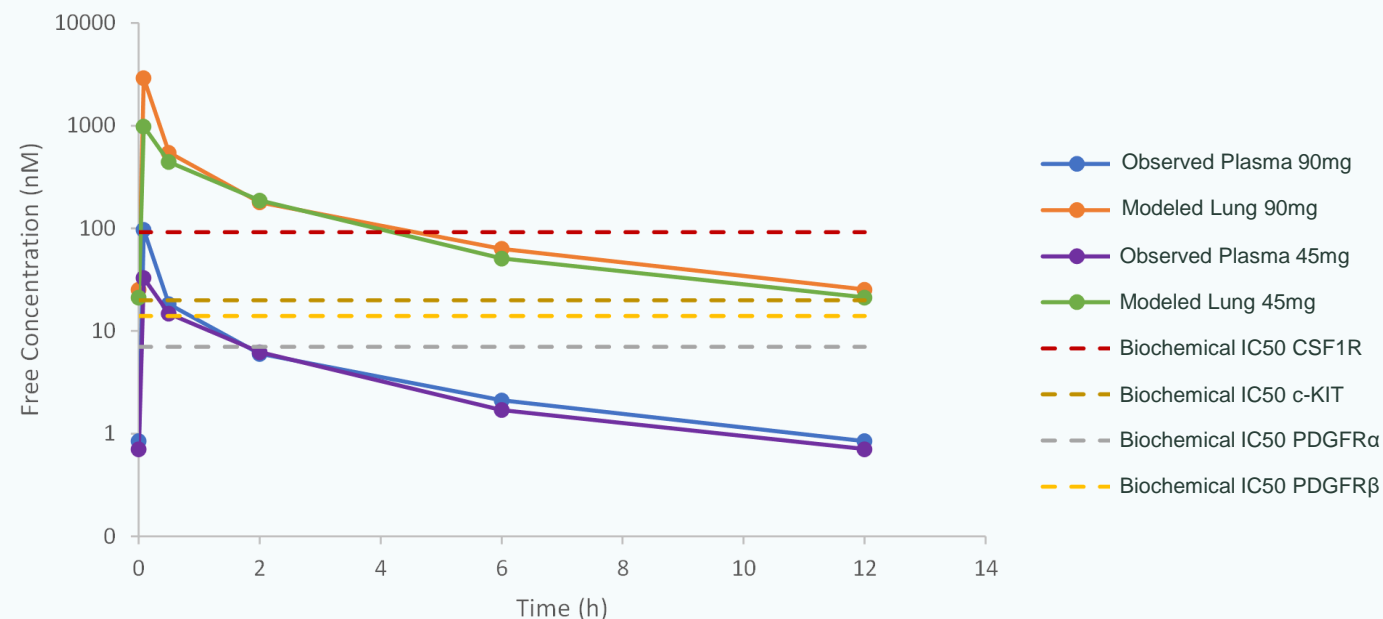
- 45 mg (wk 1) to 90 mg (wk 2) BID dose escalation first 14 days
- 90 mg BID in OLE days 15 - 169

Phase 1b PK Profile, Target Engagement Data, and Extrapolated Lung Concentrations Support Target Coverage of Dose Range Predictions

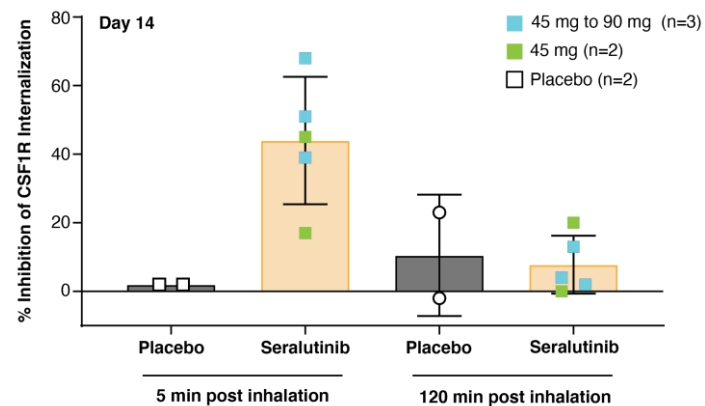
Systemic Total Drug PK in PAH Patients Matches Normal Healthy Volunteer Experience



Observed Phase 1b Free Plasma and Modeled Free Lung Concentrations Support Target Coverage in Lungs and Systemic Sparring



Seralutinib Transiently Inhibits CSF1R in Plasma



- Seralutinib systemic PK in PAH characterized by T_{max} of 5-6 min and half-life of ~4 hours following a single inhaled dose
- Blood target engagement biomarker (CSF1R) data consistent with free drug concentration levels considering biochemical IC50 of CSF1R
- With the extrapolated lung exposures of ~30x and in-vitro biochemical IC50's, seralutinib doses of 45-90 mg BID are anticipated to provide target coverage in the lung over 24 hours

Summary of Key Outcomes From Phase 1b Study in PAH with Seralutinib

Summary of Outcomes By Section of Study

Double-blind period

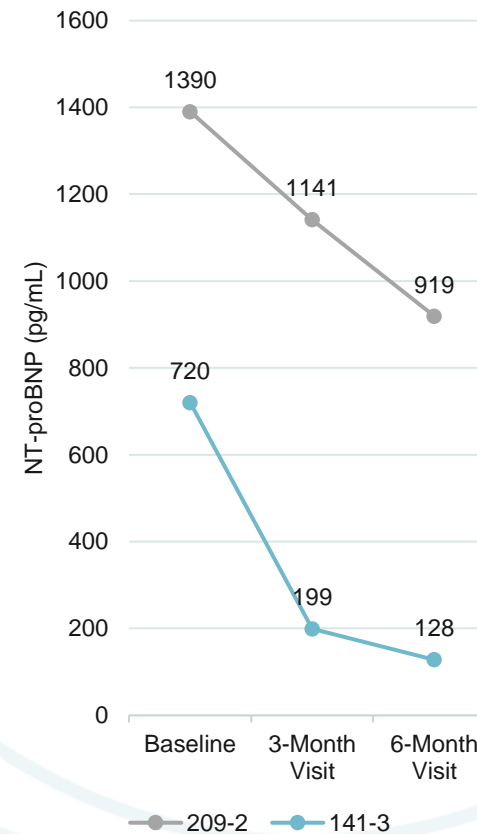
- All 8 subjects completed the 14-day treatment period
- No SAEs reported
- The most frequently reported AEs were:
 - Cough (mild-moderate)
 - Headache (mild)
- There were no clinically significant changes in labs, ECGs, PFTs, or vital signs
- Evidence of peripheral blood target engagement (e.g., CSF1R)

Open-Label Extension (OLE)

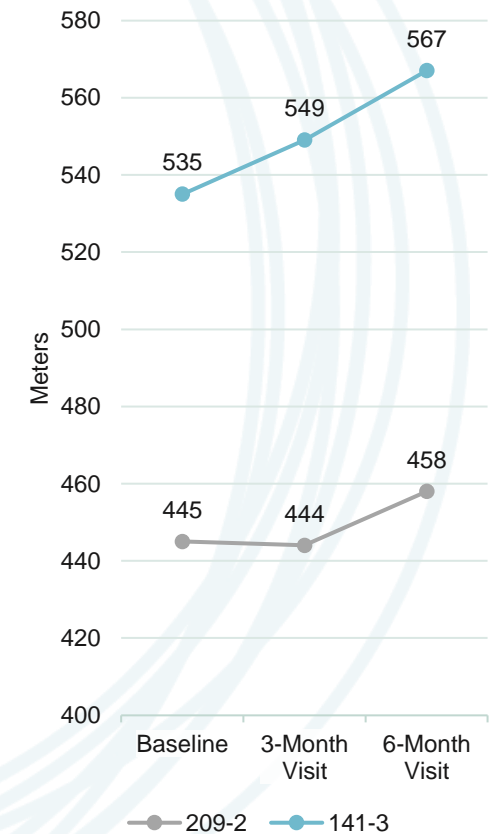
- 2 subjects entered and completed the OLE receiving 90 mg BID
- No SAEs reported and no safety concerns identified with longer term dosing
- NT-ProBNP levels decreased and 6-minute walk distances increased

Biomarker & Functional Outcomes for OLE Patients

Δ in NT-proBNP from Baseline



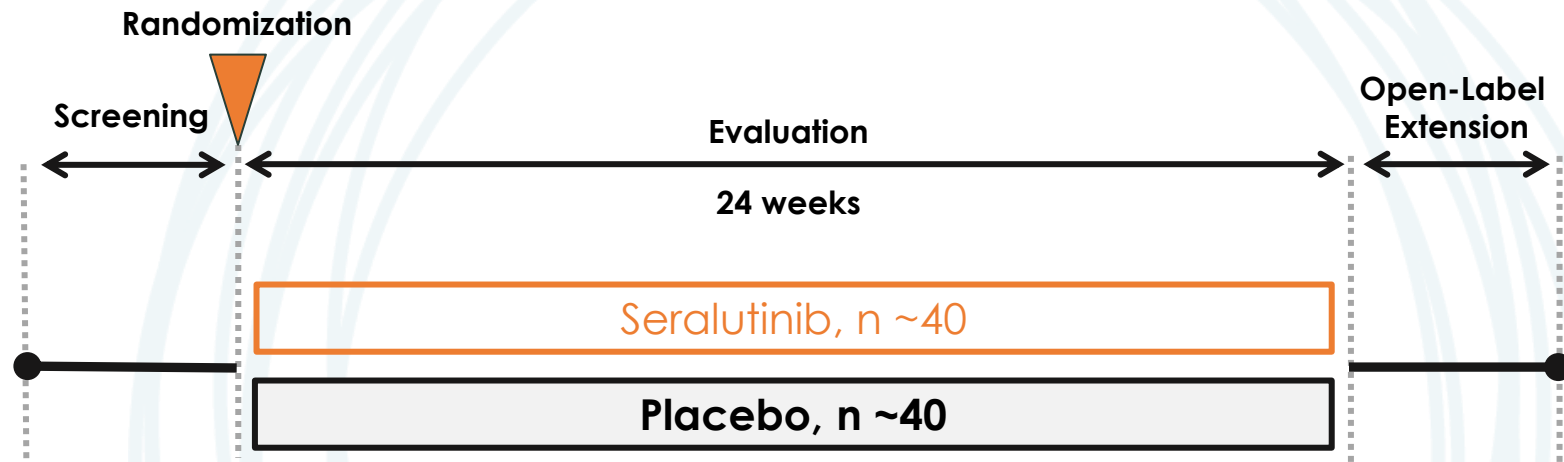
Δ in 6MWD from Baseline



TORREY: Phase 2 Study of Seralutinib in Patients With Functional Class II and III PAH



**TORREY
STUDY**



Patient Population	Functional Class II and III PAH patients on standard background therapy (including triple therapy); PVR ≥ 400 dyne*s/cm ⁵
Endpoints	Primary: PVR Change from Baseline at Week 24 Key Secondary: 6MWD Change from Baseline at Week 24 [†]
Dosing Regimen	Starting dose: 60mg BID, escalated up to 90mg BID (protocol allows for down-titration to 45mg BID)

PVR = pulmonary vascular resistance; 6MWD = six-minute walk distance.

Source: clinicaltrials.gov/NCT04456998

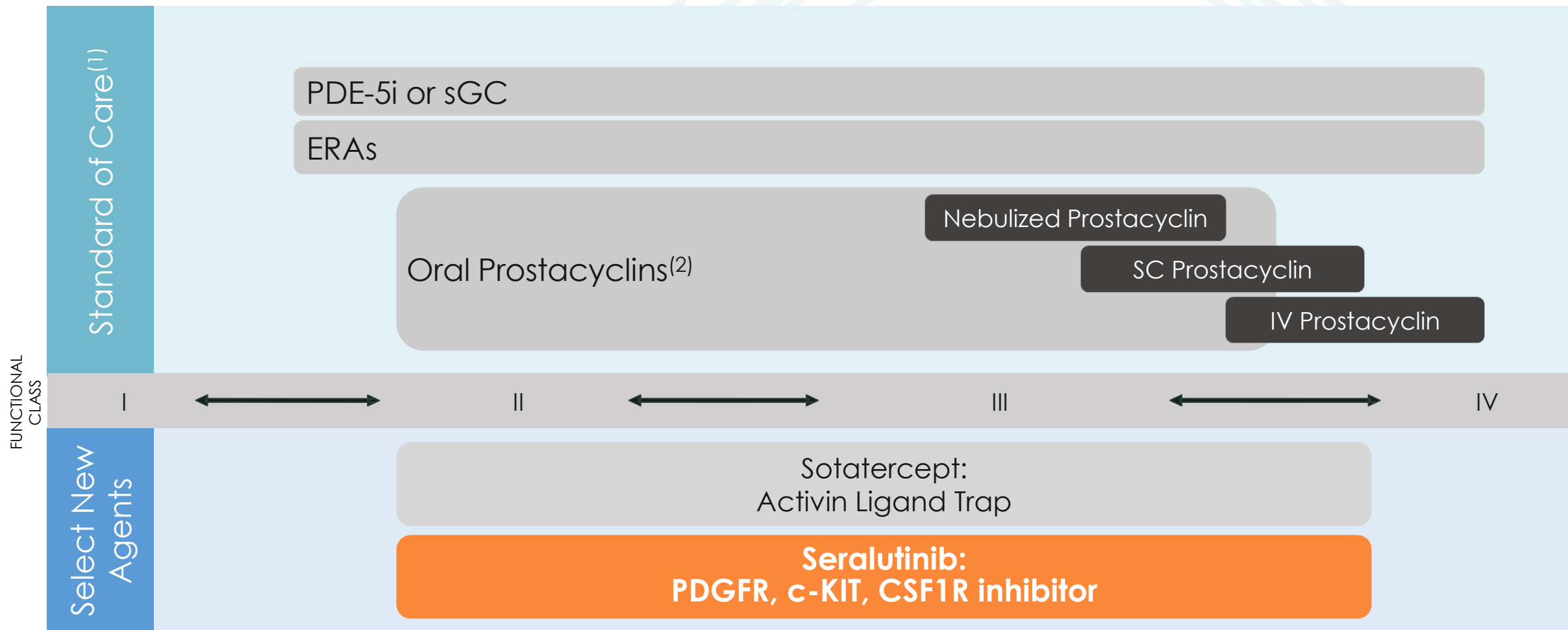
[†]Trial is not powered to demonstrate a statistically significant difference in 6MWD.

Placebo Arm PVR Changes vs. Baseline in Prior Phase 2 and Phase 3 Clinical Studies

	Sotatercept PULSAR Phase 2^a	Ralinepag Phase 2^b	Selonseritib ARROW Phase 2^c	Imatinib IMPRES Phase 3^d	Selexipag Phase 2^e	Riociguat PATENT Phase 3^f	Imatinib Phase 2^g
Year of Study Start	2018	2014	2014	2009	2008	2008	2006
Study Duration	24 weeks	22 weeks	24 weeks	24 weeks	17 weeks	12 weeks	24 weeks
N (Placebo Patients)	32	21	37	98	10	126	31
Background Medicine Breakdown	9% Mono 38% Double 53% Triple	52% Mono 48% Double	8% Mono 57% Double 35% Triple	58% Double 42% Triple	70% Mono 30% Double	52% No PAH Tx 48% Mono	19% Mono 49% Double 32% Triple
Baseline PVR	797	598	743	1181	867	1228	1118
Mean Δ in PVR from Baseline at Study End	-16	+26	+6	+12	+224	-9	-79

- a. Humbert, et al. NEJM, 2021; <https://www.clinicaltrials.gov/ct2/show/NCT03496207>
- b. Torres, et al. Eur Resp Journ, 2019; <https://www.clinicaltrials.gov/ct2/show/NCT02279160>
- c. Rosenkranz, et al. Lancet Resp, 2022; <https://www.clinicaltrials.gov/ct2/show/NCT02234141>
- d. Hoeper, et al. Circulation, 2013; <https://www.clinicaltrials.gov/ct2/show/NCT00902174>
- e. Simonneau, et al. Eur Resp Journ, 2012; <https://clinicaltrials.gov/ct2/show/NCT00993408>
- f. Ghofrani, et al. NEJM, 2013; <https://clinicaltrials.gov/ct2/show/NCT00810693>
- g. Ghofrani, et al. Am J Respir Crit Care Med, 2010; <https://clinicaltrials.gov/ct2/show/NCT00477269>

Seralutinib Offers a New, Multifaceted Approach to Treating PAH, Differentiating it From the Competitive Landscape



Seralutinib is being evaluated on-top of background therapy (≥ 1 therapy)

1) Galiè N et al. Eur Respir J 2015; 46(4):903-75;

2) Klinger JR et al. Chest 2019; 155(3): 565-586 (Klinger et al 2019 [CHEST guidelines] removed oral prostacyclin treatments in all FCs).

FC = Functional Class; SC = subcutaneous.

GB5121 & GB7208

Covalent, CNS-Penetrant BTK Inhibitors

Primary CNS Lymphoma (PCNSL) and
Multiple Sclerosis (MS)



GB5121 & GB7208: CNS-Penetrant, BTK Inhibitors

Product Candidates Description

- 2 oral, small molecule, CNS-penetrant, irreversible BTK Inhibitors optimized for CNS penetration and kinase selectivity
- Developed in-house with patent protection expected to extend into 2040s

GB5121: Lead Neuro-Oncology Candidate

- Initial indication, relapsed / refractory primary CNS lymphoma (PCNSL) provides a potential opportunity for an accelerated path to market
- Advanced into first-in-human clinical trial in 4Q21

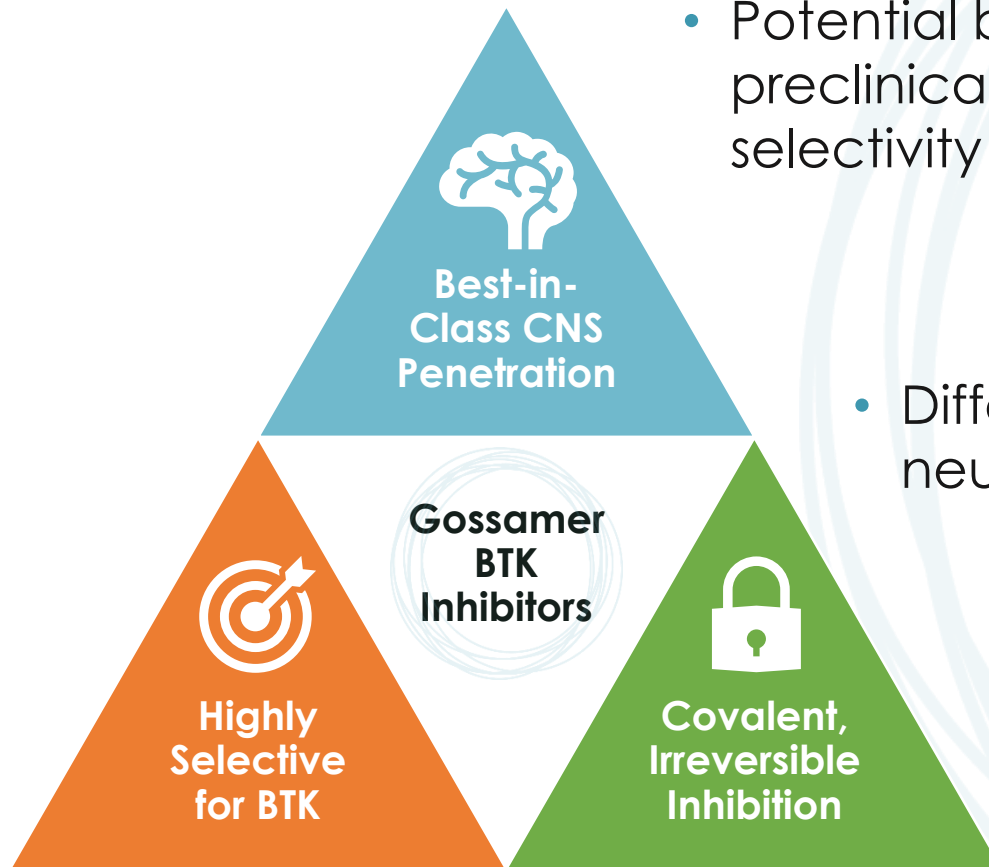
GB7208: Lead Neuro-inflammatory / Neuro-degenerative Candidate

- Superior CNS penetration / results in preclinical mouse models vs. tolebrutinib at studied doses
- First-in-human studies to begin 1H23*

INDICATION	RESEARCH	PRECLINICAL	PHASE 1	PHASE 2	PHASE 3	Next Catalyst
Primary CNS Lymphoma	GB5121: Phase 1 Ongoing (Healthy Volunteers) Phase 1b/2 Planned (PCNSL)					Initiate Phase 1b/2 (2Q22)
Multiple Sclerosis	GB7208: Preclinical					Initiate Phase 1* (1H23)

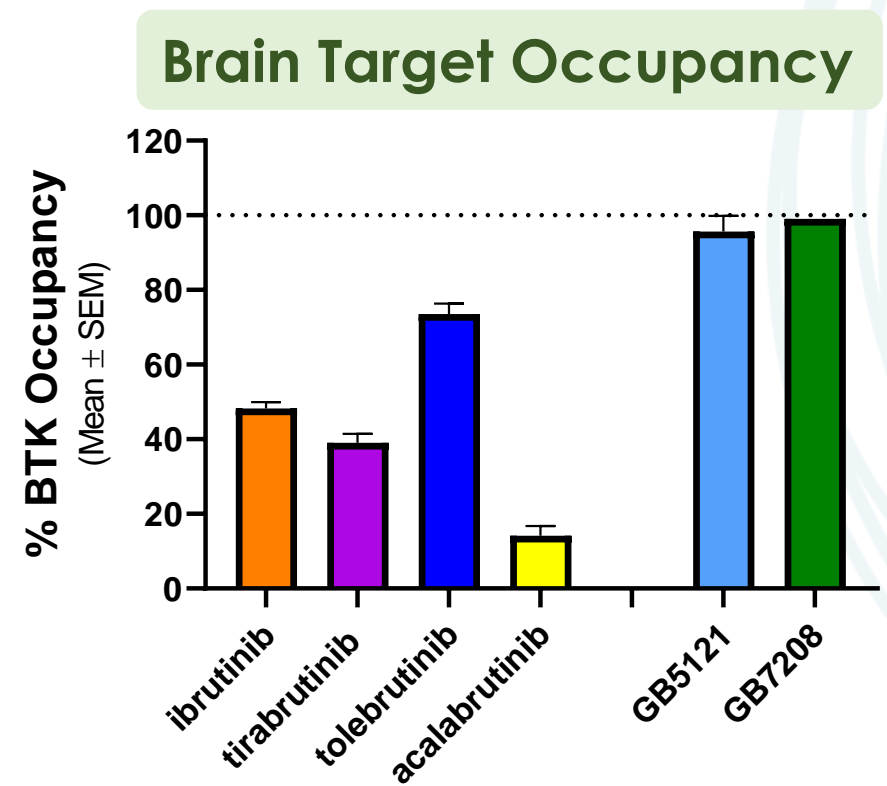
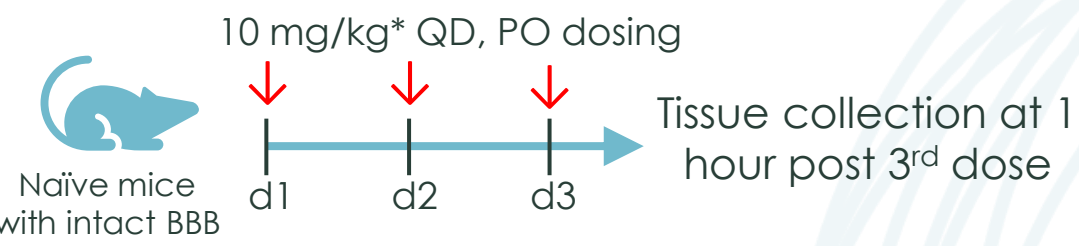
*Healthy volunteer study, pending the outcomes of our ongoing preclinical work and seralutinib Phase 2 TORREY Study topline results.

Gossamer's CNS-Penetrant BTK Inhibitors



- Potential best-in-class preclinical CNS penetration based on preclinical mouse models, complemented by high degrees of selectivity and covalent binding profile
- Differentiated BTK inhibitors for neuro-oncology, neuroinflammatory, and neurodegenerative disorders
- Gossamer has significant clinical development expertise in both neuroinflammatory conditions and hematologic cancers

GB5121 and GB7208 Demonstrate Superior Brain Penetration in Preclinical Mouse Models



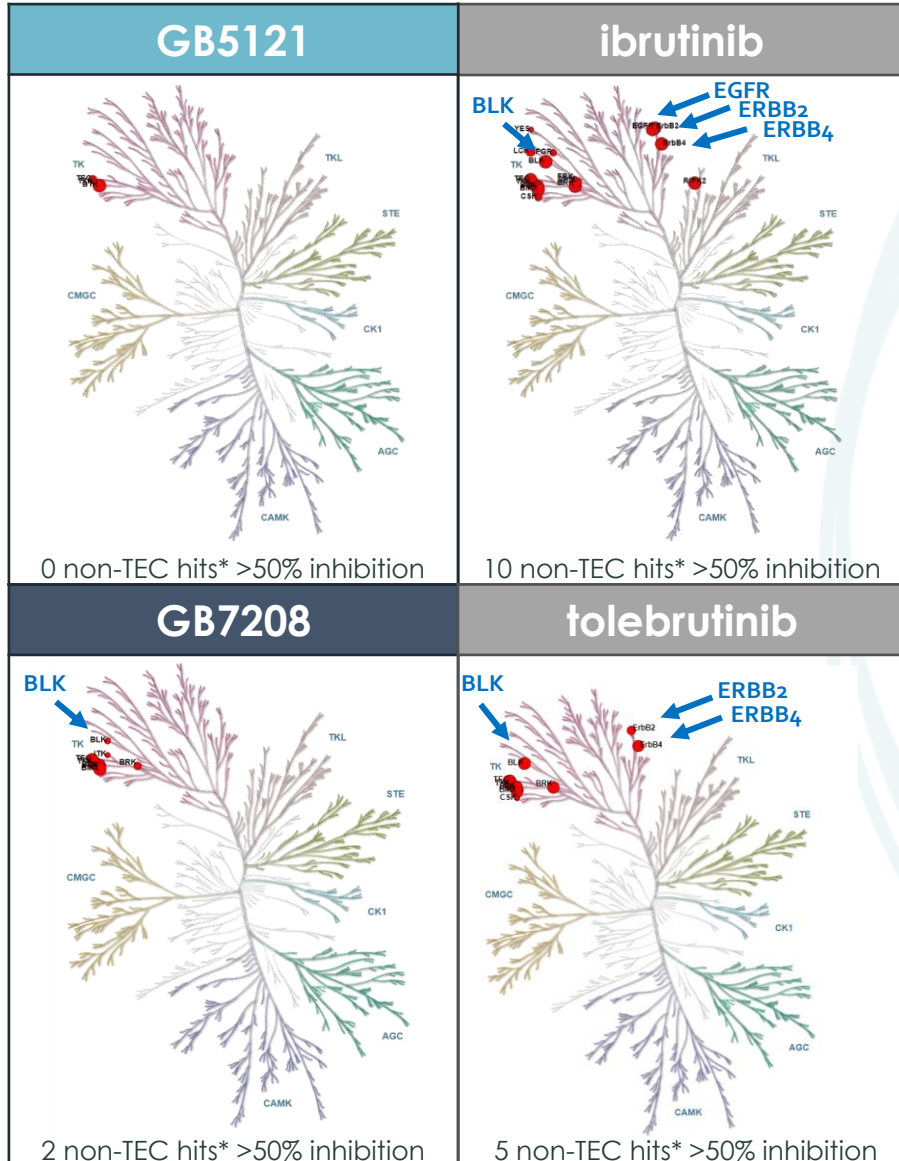
Compound	Company / Phase	Mouse Brain Target Occupancy
GB5121	Gossamer Bio / Phase 1	<div></div>
GB7208	Gossamer Bio / Preclinical	<div></div>
Tolebrutinib	Sanofi (Principia) / Phase 3	<div></div>
Ibrutinib	Abbvie & J&J / Approved	<div></div>
Tirabrutinib	Ono Pharma / Phase 2 (US), Approved (Japan)	<div></div>
Acalabrutinib	AstraZeneca / Approved	<div></div>

Based on internally generated data in naïve mice with intact BBB.

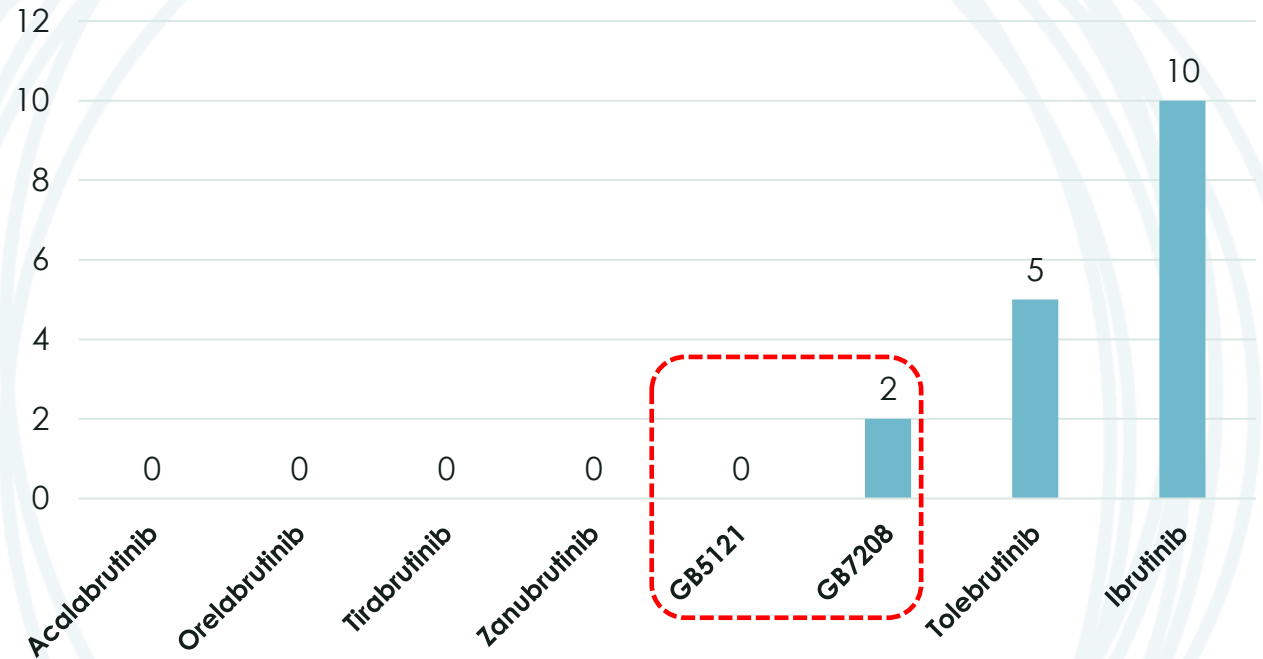
*10 mg/kg = comparable to tolebrutinib clinical dose of 60mg QD in MS (based on allometric scaling). QD = once-daily; PO = oral administration; BBB = blood brain barrier; SEM = standard error of the mean.



GB5121 and GB7208 Are Highly Selective BTK Inhibitors Based on Kinome Scans



Non-TEC Family Kinase Hits >50% Inhibition



Kinome scans suggest GB5121 and GB7208 are highly selective compounds

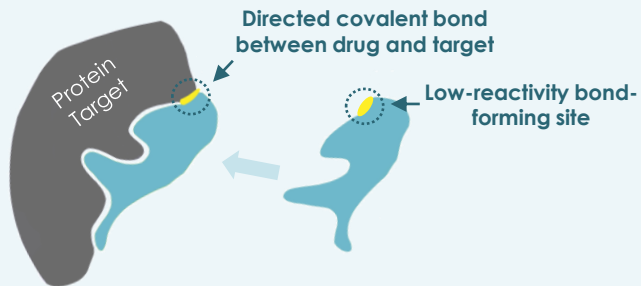
* Non-TEC family kinases inhibited >50% at 1uM concentrations

Red denotes >50% inhibition, → denotes non-TEC covalent off-target interaction

Source: Internal data on hand.

Covalent Inhibitors Provide Advantages Over Reversible Inhibitors

Covalent Inhibitors

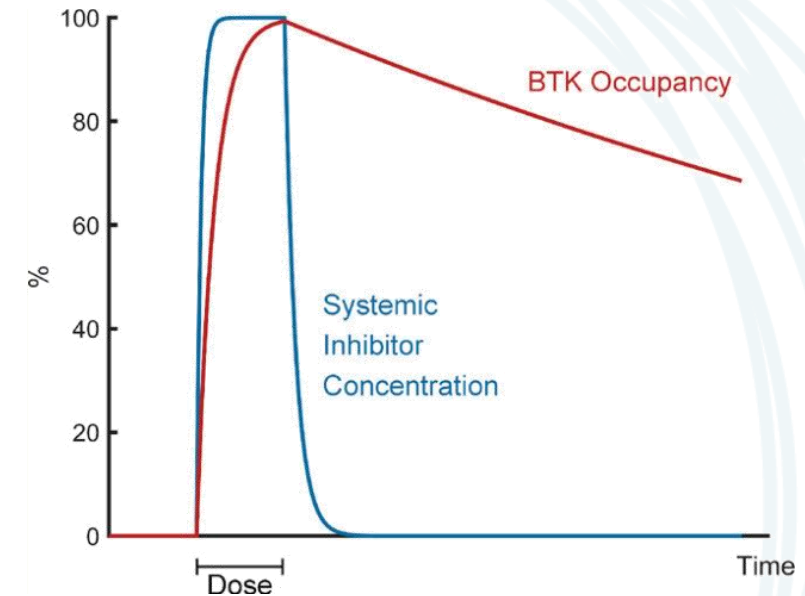


Covalent irreversible drugs bind specifically to a drug target and form a precisely direct, permanent bond with their target

Reversible Inhibitors



Traditional reversible drugs are in equilibrium with their target continually binding, unbinding and rebinding



Rapid, irreversible binding to BTK, coupled with fast clearance, enables achievement of high BTK occupancy for extended periods of time with faster systemic clearance of the inhibitor.

Advantages:

- Enhanced Potency
- Selectivity
- Prolonged Duration of Action

Primary CNS Lymphoma (PCNSL) Background

- ~1,500 new diagnosed patients / year in US⁽¹⁾
- **Median OS, from diagnosis in US, is 26 months⁽²⁾**
 - ~6 months in elderly, where >20% receive no treatment
- 1L SoC is polychemotherapy on backbone of high-dose methotrexate (HD-MTX)
 - ~50% durable remission, associated with significant late neurotoxicity
- **Prognosis remains poor: no approved R/R treatment**
 - Median recurrence at 10 – 18 months⁽³⁾
 - Median OS for R/R is 2 months without treatment⁽³⁾



1) Grommes, C et al. *J Clin Oncol*. 2017 Jul 20;35(21):2410-2418

2) Mendez JS, et al. *Neuro-Oncology*. 2018;20(5):687-694

3) Houillier C, et al. *Neurology*. 2020;94:e1027-e0139

OS = overall survival; 1L = first line; SoC = standard of care; R/R = relapsed / refractory.

Why R/R PCNSL for Initial Indication for GB5121?

Potentially The Right Molecule for the Right Indication

✓ **High unmet need:**

- 1) no approved R/R treatments
- 2) median OS only 2 months without treatment

✓ **BTKs show promise, but limited CNS exposure and safety / tolerability profile lead to disappointing results**

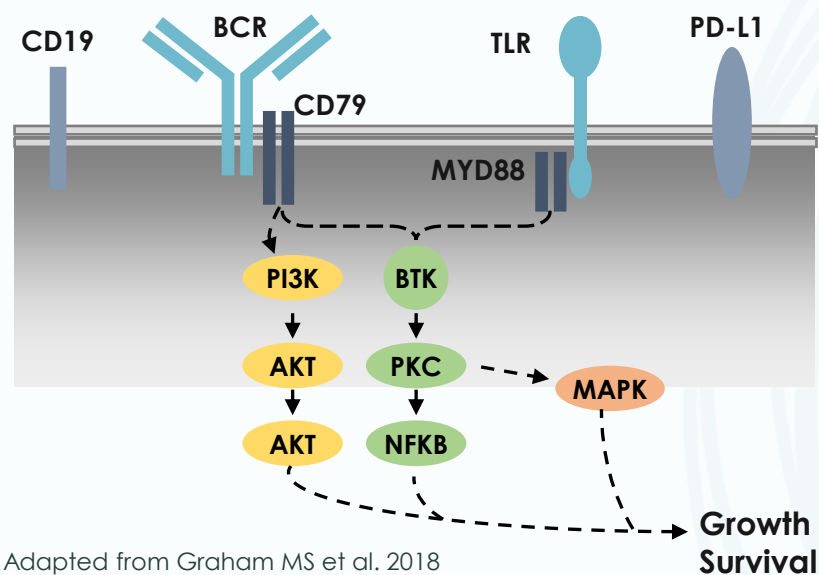
- Ibrutinib has ability to achieve responses with higher than labeled doses
- Safety / tolerability issues often result in treatment cessation and short DoR
- GB5121's brain penetration and selectivity potentially primed to address challenges

✓ **Ability to move fast**

- Rapid proof-of-concept
- Potential for accelerated path to approval

Rationale for BTK Inhibition in PCNSL

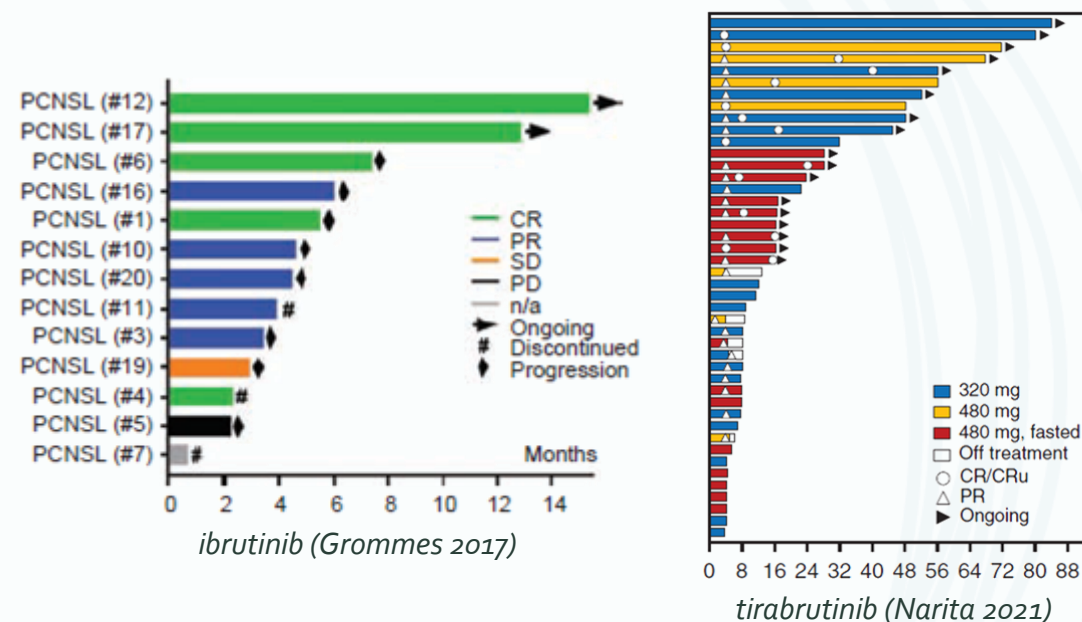
BTK Inhibition Targets a Key Survival Node in PCNSL



- PCNSL is an aggressive non-Hodgkin lymphoma restricted to the CNS without evidence of systemic spread
- Most PCNSLs are ABC-DLBCLs that carry the MyD88 driver mutations

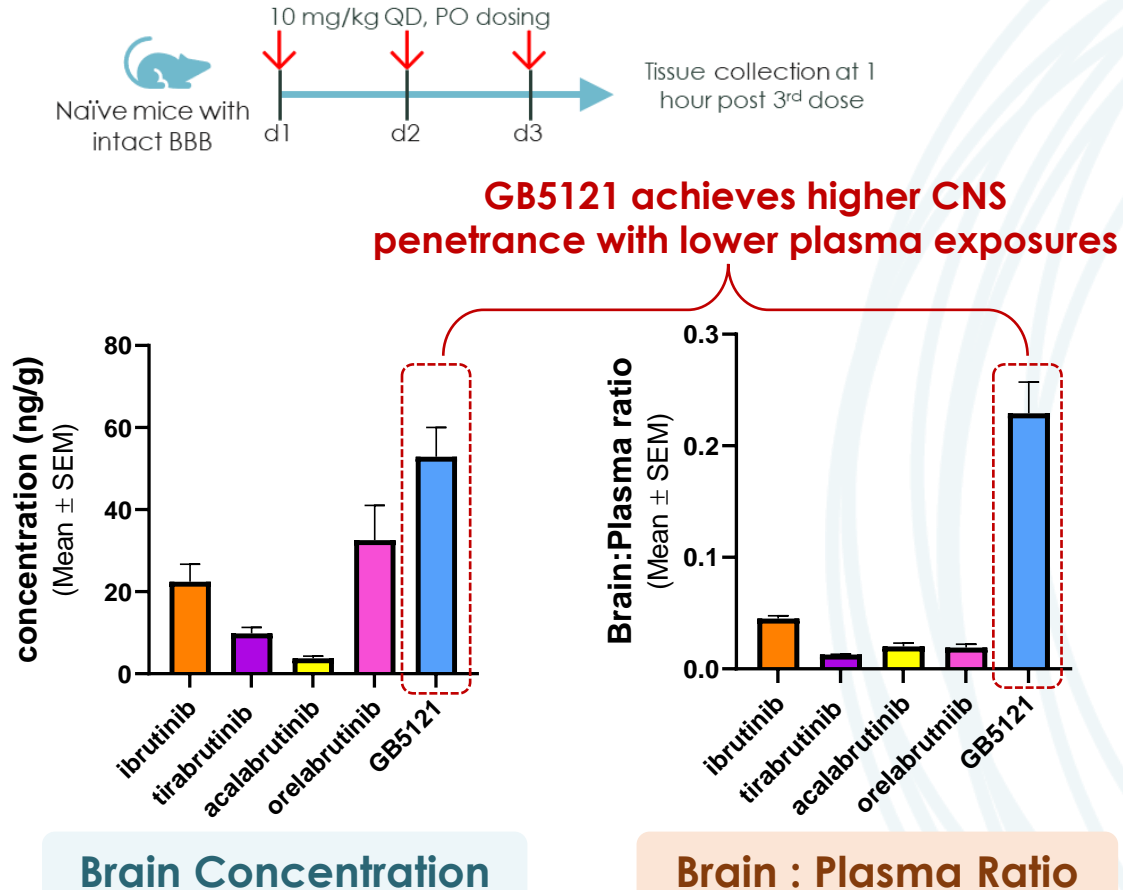
BTKi Efficacious in PCNSL Patients

ibrutinib 77% ORR, tirabrutinib 64% ORR



- Limited CNS-penetrance necessitates use of very high doses, resulting in a poor safety profile
- Duration of response is limited, which has been hypothesized to be related to insufficient CNS target coverage, leading to secondary escape mutations

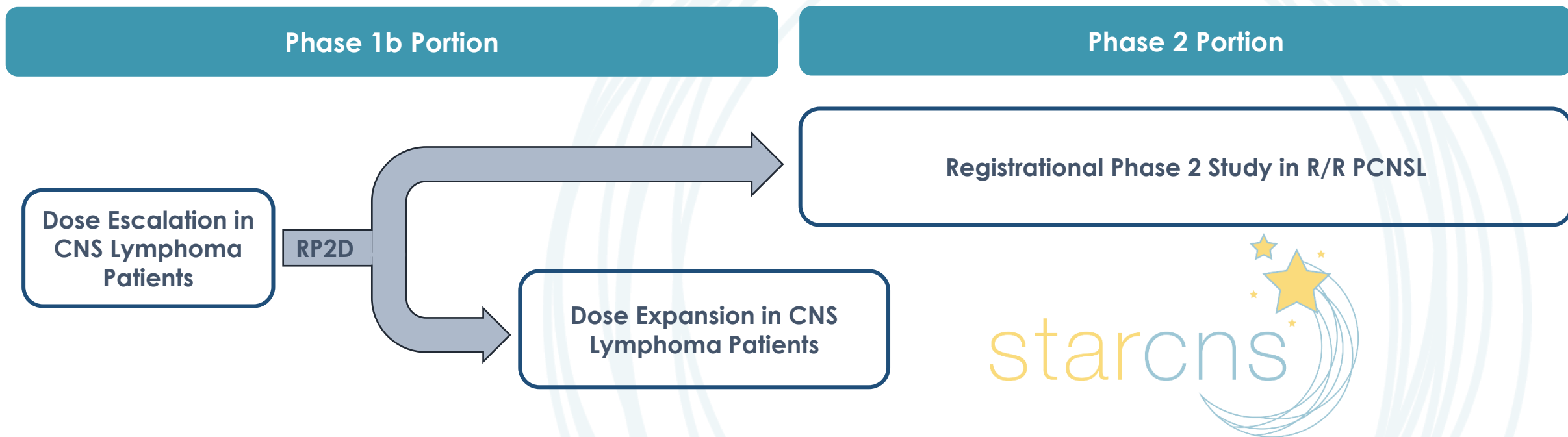
GB5121 Demonstrates Superior Brain Penetrance vs. Selected BTK Inhibitors Developed in Oncology



- Optimized for CNS penetrance and selectivity, GB5121 achieves robust drug levels and BTK occupancy in brain supporting its use in PCNSL patients
- GB5121 shows potent activity *in vitro* in DLBCL cell lines regardless of phenotype and mutational profile
- Development of *in vivo* PCNSL models underway with top academic collaborators

Excellent brain penetrance and selectivity combined with activity against DLBCL cell lines positions GB5121 for differentiated efficacy and safety in patients with CNS lymphomas

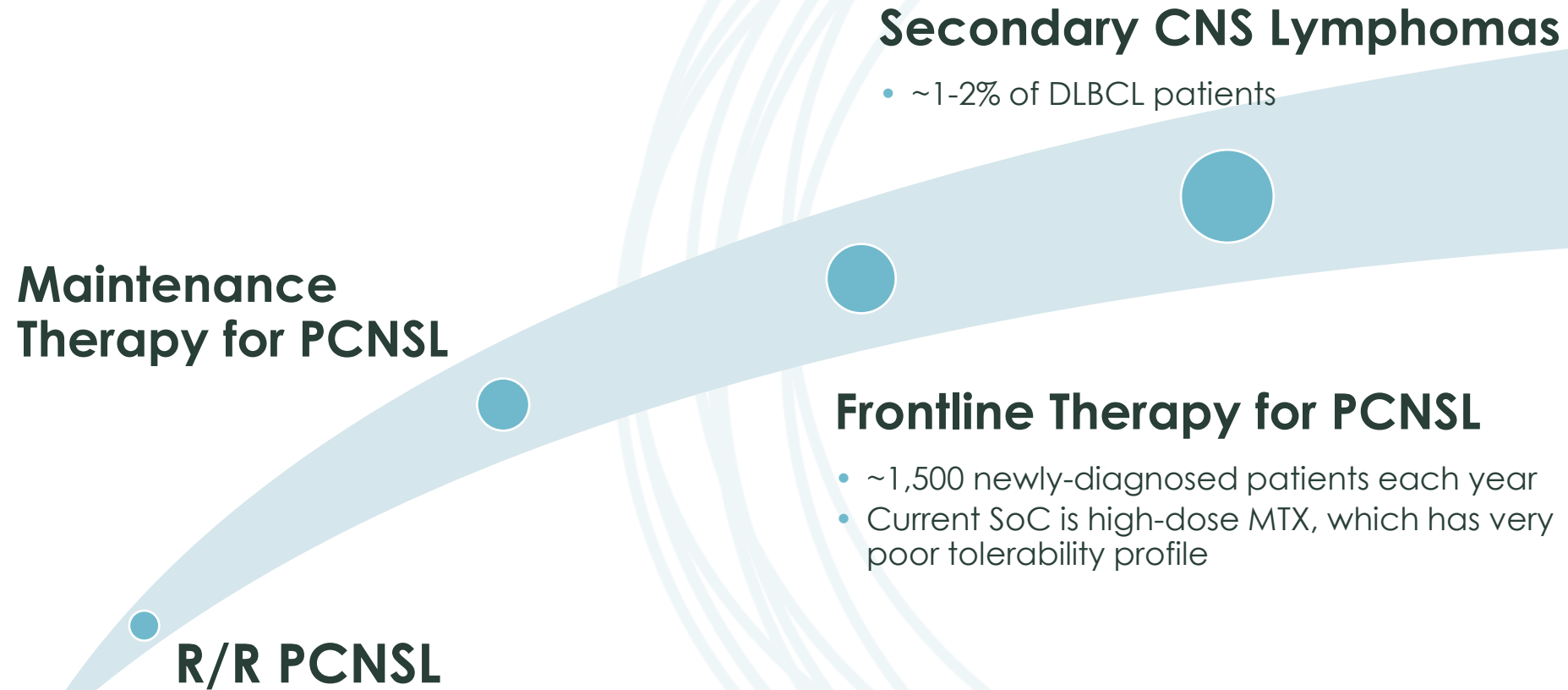
GB5121 STAR CNS Phase 1b/2 Expected to Initiate in 2Q:2022, Providing Potential Path to Registration



Patient Population	R/R primary and secondary CNS lymphoma patients, R/R primary vitreoretinal lymphoma
Endpoints	Primary: Safety, tolerability, recommended Phase 2 dose Secondary: ORR, Duration of Response

Patient Population	R/R primary CNS lymphoma patients, R/R primary vitreoretinal lymphoma
Endpoints	Primary: ORR Secondary: Duration of Response, OS, PFS, Safety, Tolerability

Opportunities to Expand Beyond R/R PCNSL





Corporate Overview and Milestones

Financial Overview

Cash, Cash Equivalents and Marketable Securities

(As of 3/31/22)

~\$272mm

Debt, *Related to Line of Credit*

(As of 3/31/22; initial tranche of credit facility, announced 5/2/19)

~\$30mm

Principal of Convertible Notes Outstanding

(As of 3/31/22)

~\$200mm

Common Shares Outstanding

(As of 5/5/22)

~77mm

Upcoming Clinical Milestones

Population	Milestone	Timing
Seralutinib (Pulmonary Arterial Hypertension)		
PAH	Phase 2 TORREY Study Topline Results	4Q 2022
GB5121 (Rare CNS Malignancies)		
PCNSL	Initiate Phase 1b/2	2Q 2022
GB7208 (Autoimmune, Multiple Sclerosis)		
Healthy Volunteers	Initiate Phase 1*	1H 2023

*Pending the outcomes of our ongoing preclinical work and pending seralutinib Phase 2 TORREY Study topline results.