CNS-Penetrant BTK Franchise Investor Day

October 11, 2021

Forward Looking Statements

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In some cases, you can identify forward-looking statements by terms such as "may," "will," "should," "expect," "plan," "anticipate," "could," "intend," "target," "project," "contemplates," "believes," "estimates," "predicts," "potential" or "continue" or the negative of these terms or other similar expressions. These statements involve known and unknown risks, uncertainties and other important factors that may cause our actual results, performance or achievements to be materially different from any future results, performance or achievements expressed or implied by the forward-looking statements. These known risks and uncertainties are described in detail in our filings with the Securities and Exchange Commission (the "SEC") from time to time. Because forward-looking statements are inherently subject to risks and uncertainties, some of which cannot be predicted or quantified and some of which are beyond our control, you should not rely on these forward-looking statements as predictions of future events. The events and circumstances reflected in our forward-looking statements may not be achieved or occur and actual results could differ materially from those projected in the forward-looking statements. Except as required by applicable law, we do not plan to publicly update or revise any forward-looking statements contained herein, whether as a result of any new information, future events, changed circumstances or otherwise. All forward-looking statements are qualified in their entirety by this cautionary statement, which is made under the safe harbor provisions of the Private Securities Litigation Reform Act of 1995 and we undertake no obligation to revise or update this presentation to reflect events or circumstances after the date hereof.

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CNS-Penetrant BTK Franchise Investor Day Agenda

Topic	Presenter
Introductions & CNS Penetrant-BTK Inhibitor Overview	**Faheem Hasnain**
I. Standing Apart From the Crowd: Selective, CNS-Penetrant Franchise for Neurological Conditions	**Laura Carter, PhD**
III. Spearhead into the Clinic: GB5121 in Primary CNS Lymphoma and CNS Oncology Diseases	**Laura Carter, PhD** **Richard Aranda, MD**
IV. Returning to Roots: GB7208 in Multiple Sclerosis and Other CNS Autoimmune Disorders	**Brett Skolnick, PhD** **Isharat Yusuf, PhD**
V. Closing Remarks	**Faheem Hasnain**
Q&A	

Presenters on Today's Call

Faheem Hasnain

Co-Founder, Chairman and Chief Executive Officer

Laura Carter, PhD

Chief Scientific Officer

Richard Aranda, MD

Chief Medical Officer

Brett Skolnick, PhD

Vice President, Clinical Development

Rachel Altura, MD

Vice President, Clinical Development

Isharat Yusuf, PhD

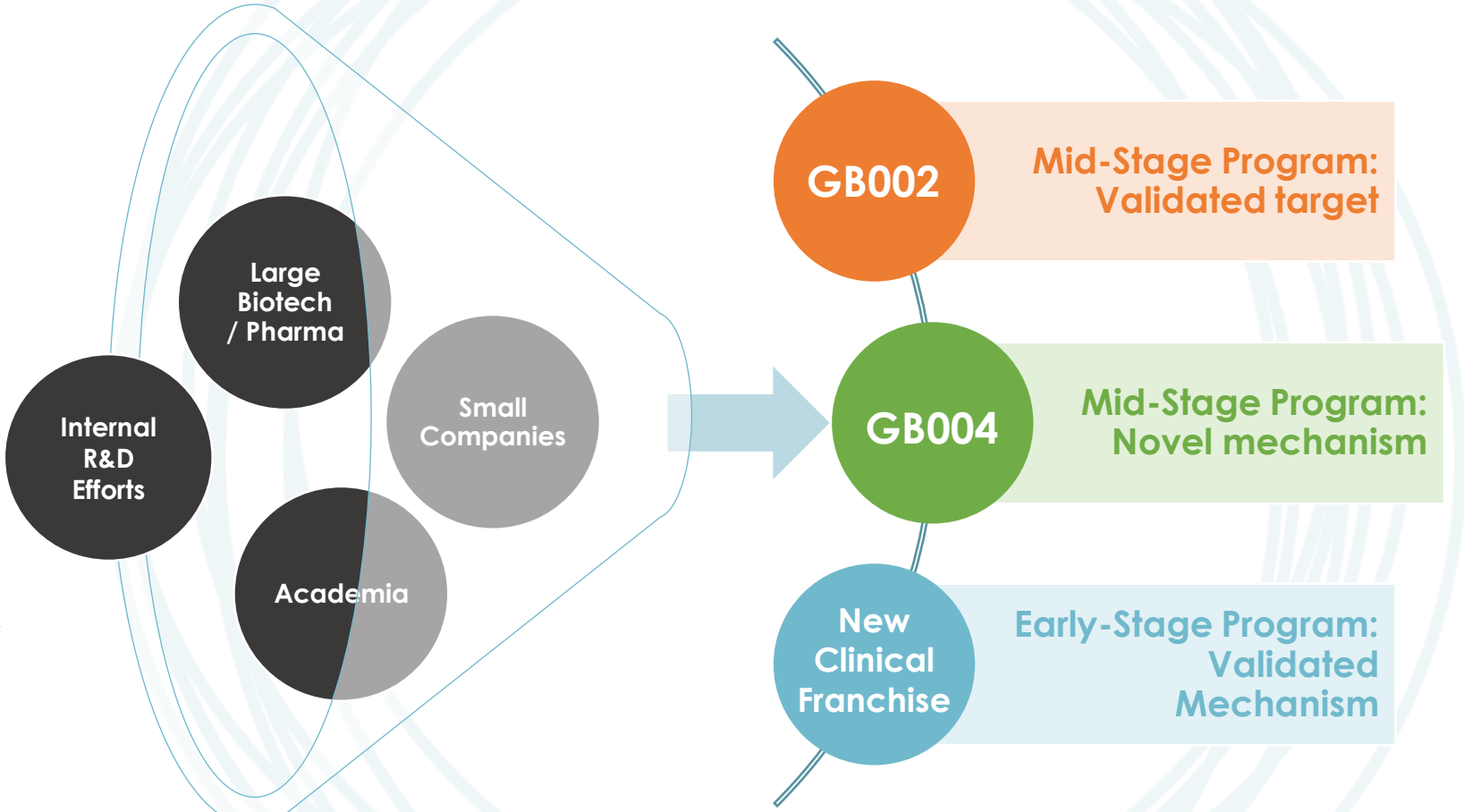
Senior Director, Pharmacology, Biology

The State of Gossamer Bio

Immunology Focus	• Disruptive research and development engine focused on the disease areas of immunology, inflammation and oncology
Diversified Pipeline	Click to add text • **2** ongoing proof-of-concept Phase 2 studies in PAH and UC
World-Class Talent	• Deeply experienced leadership team with proven track record of developing innovative clinical assets

Gossamer's Core Immunology Capabilities Inform a Rigorous Pre-Clinical Vetting Process

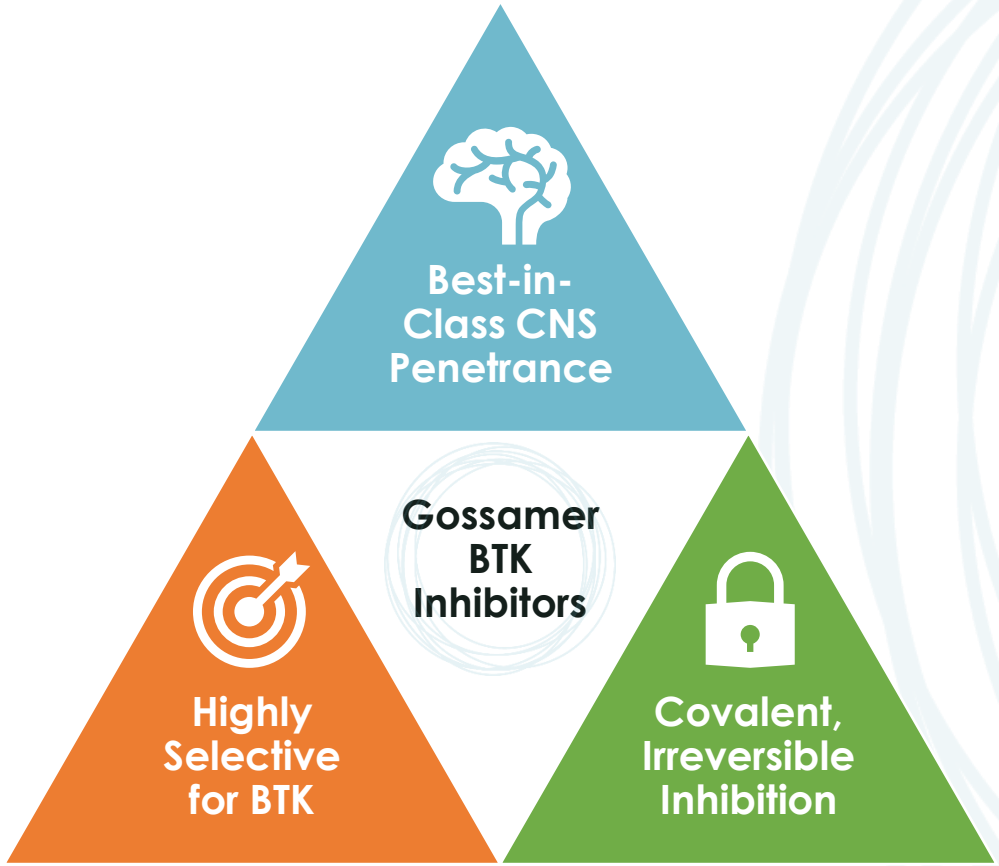
- Deep and wide-ranging relationships
- Team with a track record of success
- Decades long history in immunology



High bar for progressed product candidates: hundreds of external assets and 10+ internally-driven research programs* assessed over past 24 months

*Additional internal research projects continue in development.

Gossamer's First Internal Research Pipeline Candidates: CNS-Penetrant BTK Inhibitors



Molecules designed specifically to address neuro-oncology and neuroinflammatory diseases

GB5121: Lead Neuro-Oncology Candidate

- Initial indication, relapsed / refractory primary CNS lymphoma provides a potential opportunity for an accelerated path to market
- First-in-human studies to begin Q4:21

GB7208: Lead Neuro-inflammatory / Neuro-degenerative Candidate

- Superior CNS penetrance / efficacy in pre-clinical models vs. tolebrutinib
- First-in-human studies to begin 2H:22

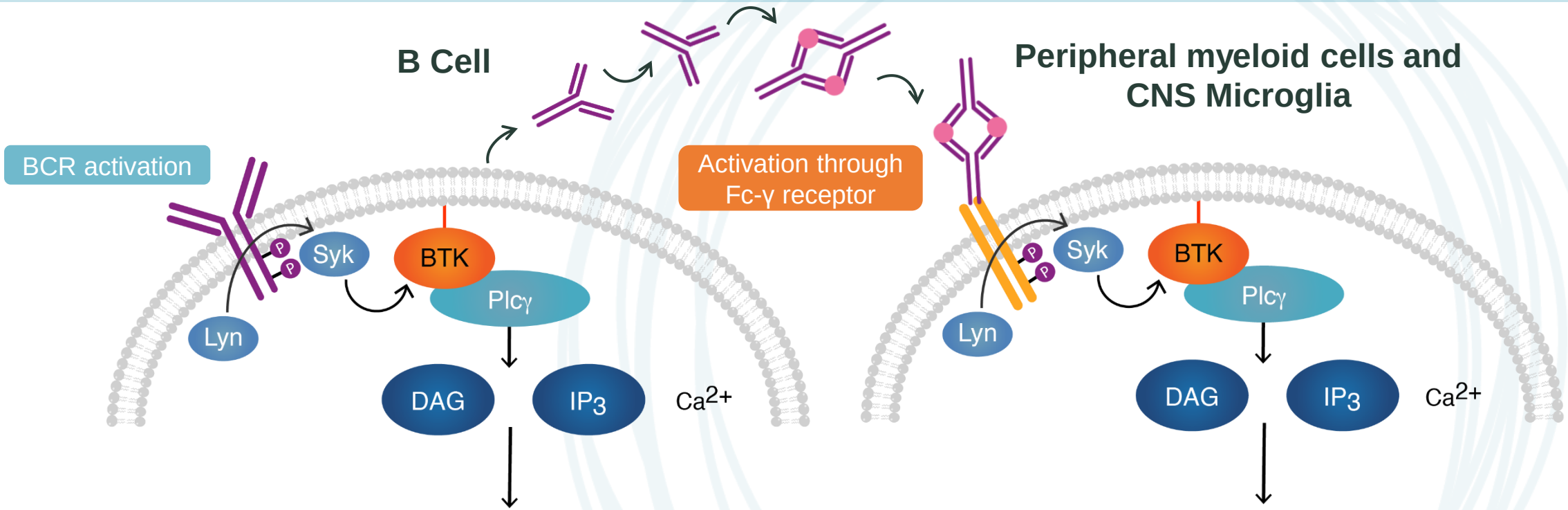
Developed in-house with patent protection expected to extend into 2040s

BTK = Bruton's tyrosine kinase.

I.
Standing Apart From the Crowd:
Selective, CNS-Penetrant Franchise for Neurological Conditions

Laura Carter, PhD

BTK Plays Critical Role in B Cell & Myeloid Cell Function



- **B-cell maturation, activation, chemotaxis, trafficking, adhesion**
- **Autoantibody production**
- **Cytokine secretion**

- **Cytokine secretion**
- **Inflammatory mediators**

- BTK is a critical signaling node in B cells, peripheral myeloid cells and CNS-resident microglia
- Inhibition of BTK has proven to be effective in the treatment of numerous lymphomas and is being investigated in inflammatory conditions

BCR = B cell receptor.

BTK Inhibition Validated in Oncology, Field Shifting Focus to MS & Inflammation

BTK Inhibitors in Hematologic Cancers

- 3 BTK inhibitors FDA approved for hematologic cancers
- High response rate & long durations of response
- **Current Limitations:**
  - Non-selective inhibitors have off-target side effects limiting therapeutic window
  - Development of BTK mutations can result in loss of response
  - Sub-optimal CNS exposure requires increased (& often intolerable) doses in CNS diseases

BTK Inhibitors in Inflammation

- Initial development mainly focused on systemic inflammatory diseases, e.g., asthma, RA and SLE
- BTK pathogenesis in neuroinflammatory and neurodegenerative processes driven by microglia make conditions like MS attractive
- **Current Limitations:**
  - Limited CNS exposures to date have limited potential therapeutic impact
  - Poor selectivity, off-target effects, and / or reversibility limit therapeutic window

MS = multiple sclerosis; RA = rheumatoid arthritis; SLE = systemic lupus erythematosus.

GB5121and GB7208 Stand Apart as BTK Inhibitors Optimized for Neurology Indications

Selected Approved & Development-Stage BTK Inhibitors

ibrutinib acalabrutinib zanabrutinib tirabrutinib fenebrutinib evobrutinib tolebrutinib
orelabrutinib pitrobrutinib branebrutinib BIIB-091 **GB5121** **GB7208**

ibrutinib orelabrutinib zanabrutinib branebrutinib
acalabrutinib evobrutinib tirabrutinib tolebrutinib
GB5121 **GB7208**

Characteristic #1:
Irreversible BTK Binding
Covalently bound molecules provide target coverage for extended periods of time at transient doses, potentially providing enhanced potency and selectivity and a prolonged duration of action

tolebrutinib

GB5121 **GB7208**

Characteristic #2:
High Brain Penetrance in Preclinical Models
Treating neuro-oncology, neuro-inflammatory, and neuro-degenerative diseases driven by B cells require high target occupancy in the CNS at systemically tolerable doses

GB5121

GB7208

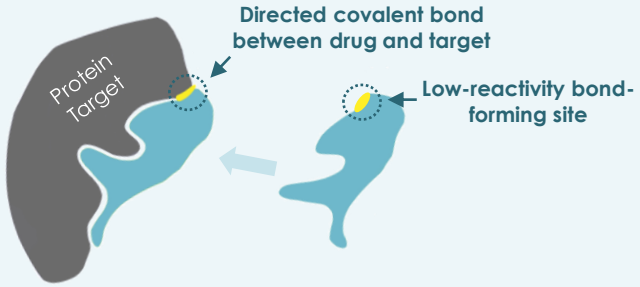
Characteristic #3:
High Selectivity
Avoidance of off-target, covalent binding to other kinases potentially improves systemic safety and tolerability, broadening the therapeutic index

Ideal BTKi profile for neuro-oncology, -inflammatory, and -degenerative disorders

BTKi = Bruton's tyrosine kinase inhibitor.

Covalent Inhibitors Provide Advantages Over Reversible Inhibitors

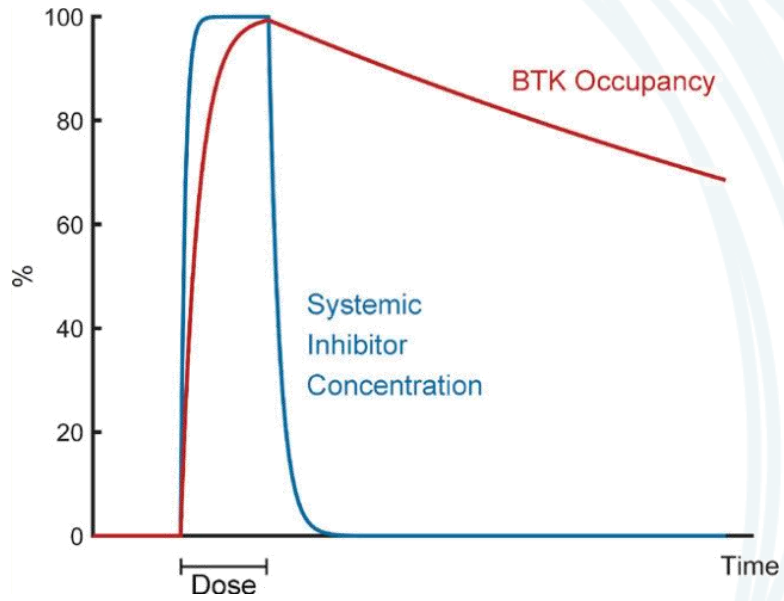
Covalent Inhibitors



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



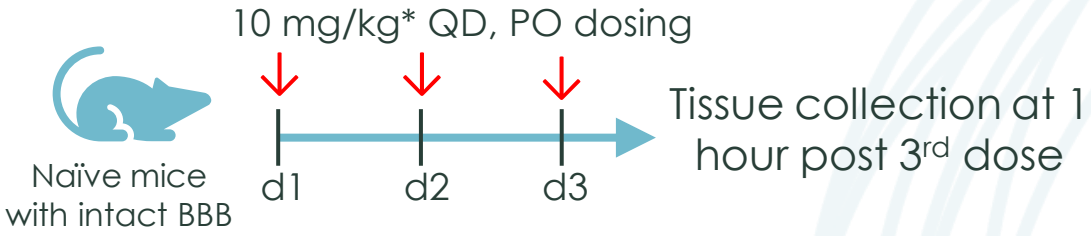
Rapid, irreversible binding to BTK, coupled with fast clearance, enables achievement of high BTK occupancy for extended periods of time with faster systemic clearance of the inhibitor.

Advantages:

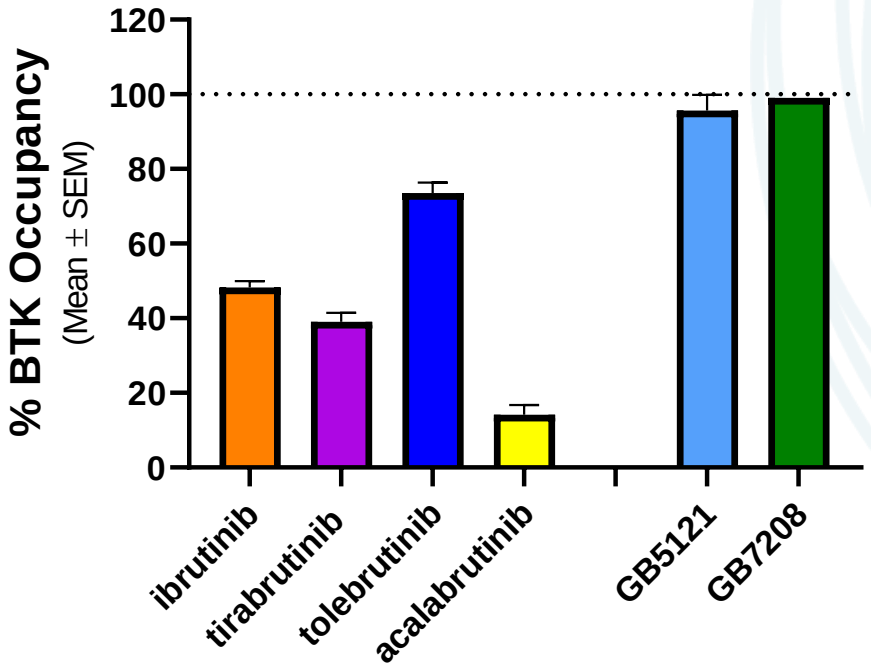
- Enhanced Potency
- Selectivity
- Prolonged Duration of Action

Source: Singh, J. et al. Nat Rev Drug Discov 10, 307–317 (2011)

GB5121 and GB7208 Demonstrate Superior Brain Penetrance in Preclinical Models



Brain Target Occupancy

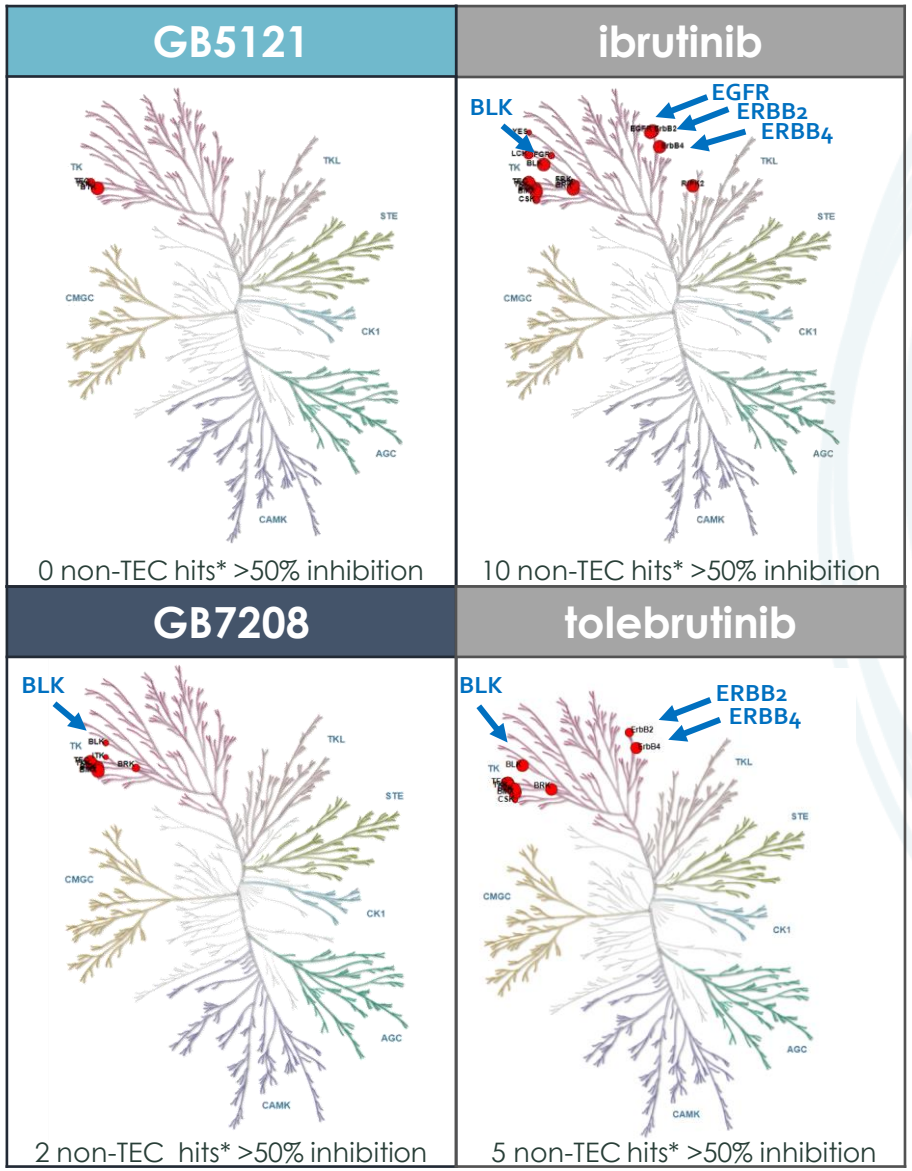


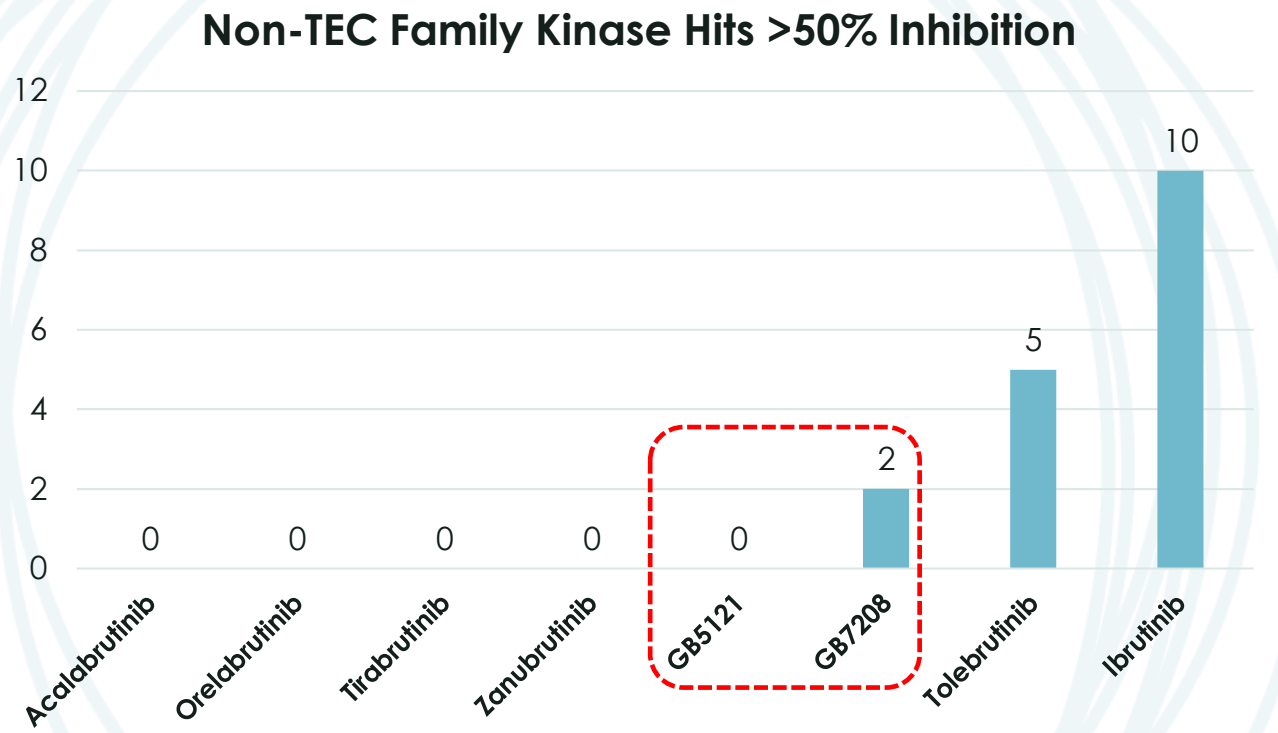
Compound	Company / Phase	Mouse Brain Target Occupancy
GB5121	Gossamer Bio / Preclinical	
GB7208	Gossamer Bio / Preclinical	
Tolebrutinib	Sanofi (Principia) / Phase 3	
Ibrutinib	Abbvie & J&J / Approved	
Tirabrutinib	Ono Pharma / Phase 2 (US), Approved (Japan)	
Acalabrutinib	AstraZeneca / Approved	

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





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.

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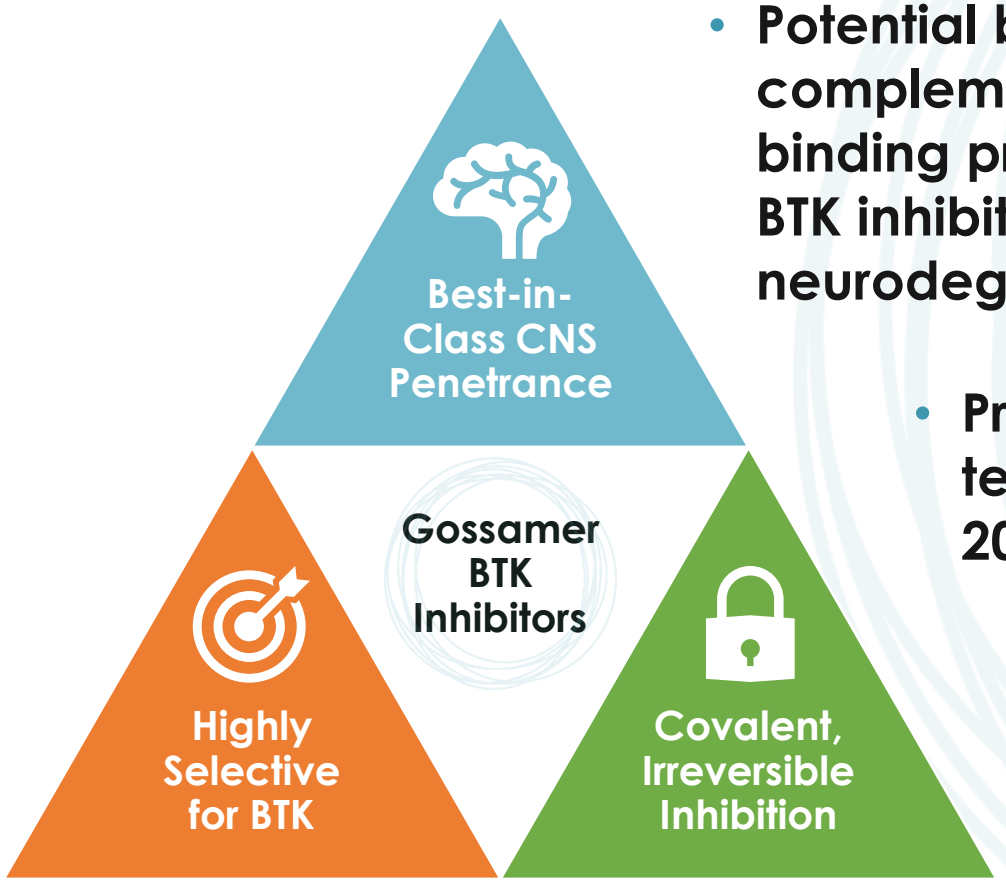
GB5121 and GB7208 Are Differentiated BTK Inhibitors Primed to Make Transition to the Clinic



- **Potential best-in-class preclinical CNS penetration, complemented by high degrees of selectivity and covalent binding profile position GB5121 and GB7208 as differentiated BTK inhibitors for neuro-oncology, neuroinflammatory, and neurodegenerative disorders**
  - **Programs developed in house by Gossamer research team, with patent protection expected to extend into 2040s**
  - **Gossamer has significant clinical development expertise in both neuroinflammatory conditions and hematologic cancers**

III.
Spearhead into the Clinic:
GB5121 in Primary CNS Lymphoma and CNS Oncology

Laura Carter, PhD

Richard Aranda, MD

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 Relapsed / Refractory PCNSL for Initial Indication for BTK Franchise?

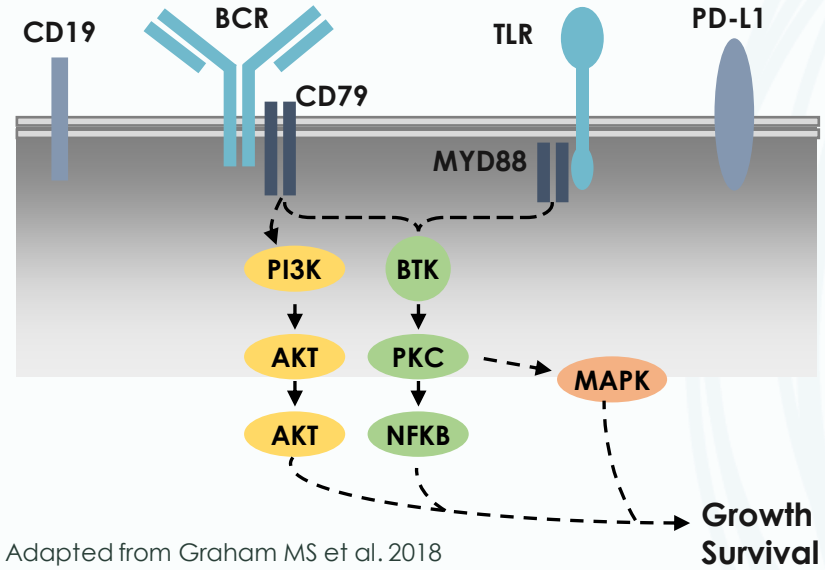
The Right Molecule for the Right Indication

- ✓ **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 penetrance and selectivity primed to address challenges
- ✓ **Ability to move fast**
  - Rapid proof-of-concept
  - Potential for accelerated path to approval

R/R = relapsed / refractory; OS = overall survival; DoR = duration of response.

Rationale for BTK Inhibition in PCNSL

BTK Inhibition Targets a Key Survival Node in PCNSL

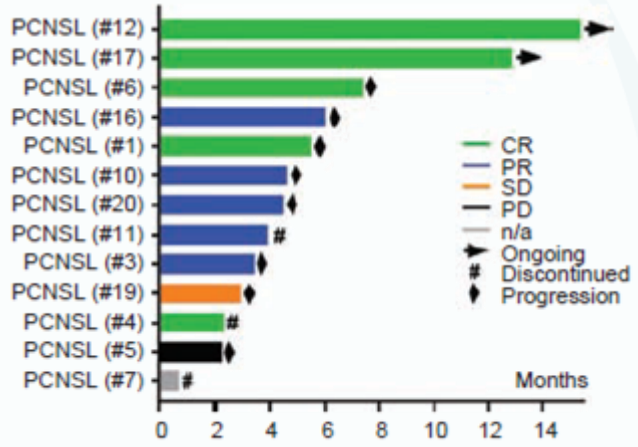


Adapted from Graham MS et al. 2018

- 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

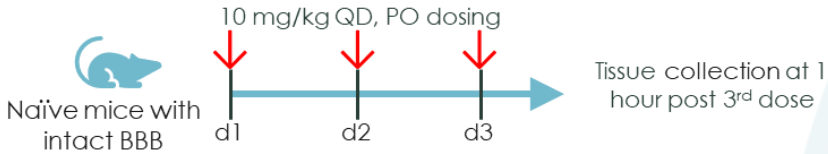


ibrutinib (Grommes 2017)

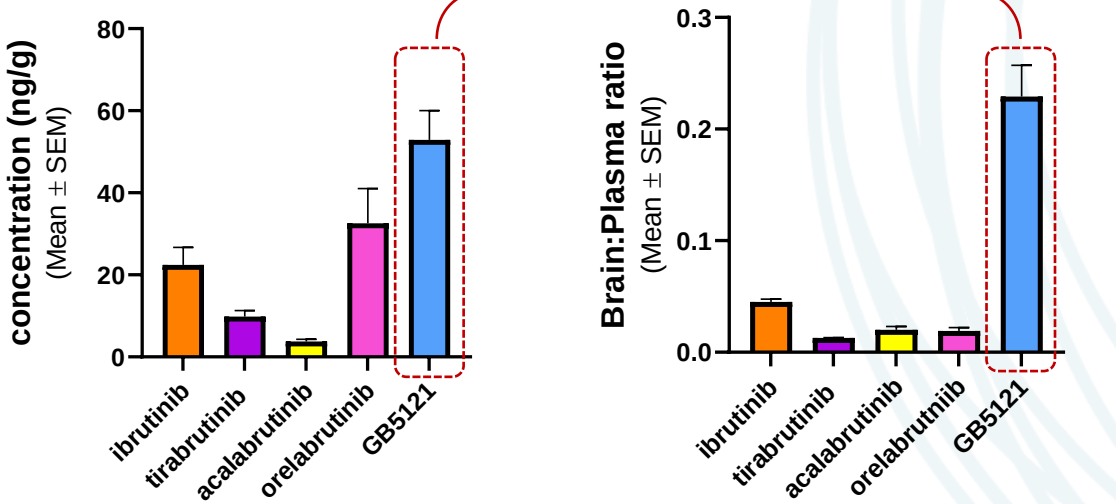
- Limited CNS-penetrance of ibrutinib necessitates use of very high doses, resulting in a poor safety profile
- Duration of response of ibrutinib is limited, which has been hypothesized to be related to insufficient CNS target coverage, leading to secondary escape mutations

Sources: Graham MS, Deangelis LM, Best Prac Res Clin Haematol, 2018 31(3), 262-269; Grommes C. et al. Cancer Discov. 2017 Sep 7(9):1018-1029.

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



GB5121 achieves higher CNS penetrance with lower plasma exposures



Brain Concentration

Brain : Plasma Ratio

- 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

Source: Internal data on hand.
QD = once-daily; PO = oral administration; BBB = blood brain barrier; SEM = standard error of the mean; DLBCL = diffuse large B cell lymphoma.

Phase 1a Study in Healthy Volunteers Expected to Initiate in Q4:2021

Part A

Single Ascending Dose

Cohorts 1 – 6

Multiple Ascending Dose

Cohorts 7 – 9

Parts B and C

Food Effect

Preliminary CYP3A DDI

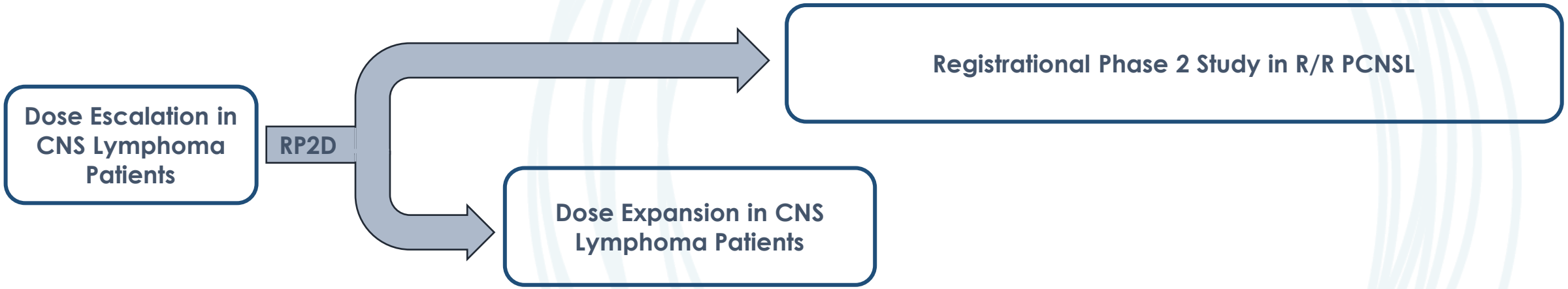
Endpoints	**Primary:** Evaluate safety, tolerability, and PK of escalating single and multiple doses of GB5121 **Secondary:** PK in cerebral spinal fluid, prelim. food and formulation effect on PK, prelim. CYP3A DDI evaluation
Timing	**Initiation:** Q4:2021 **Top-line Data:** 1H:2022

DDI = drug-drug interaction; PK = pharmacokinetics.

Phase 1b/2 Expected to Initiate in 1H:2022, Providing Potential Path to Registration

Phase 1b Portion – Expected Initiation: 1H:22

Phase 2 Portion – Expected Initiation: 1H:23

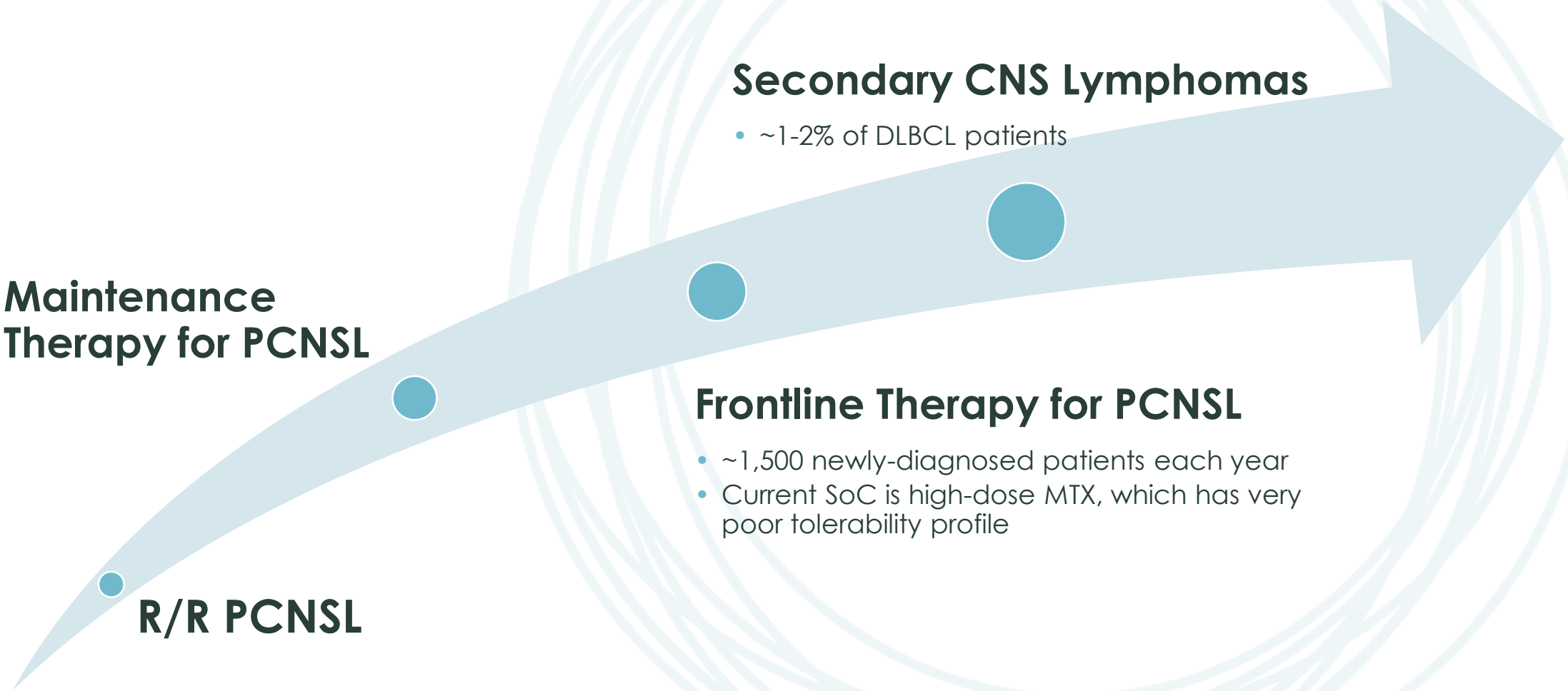


Patient Population	R/R primary and secondary CNS lymphoma patients
Endpoints	**Primary:** Safety, tolerability, recommended Phase 2 dose **Secondary:** ORR, Duration of Response

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

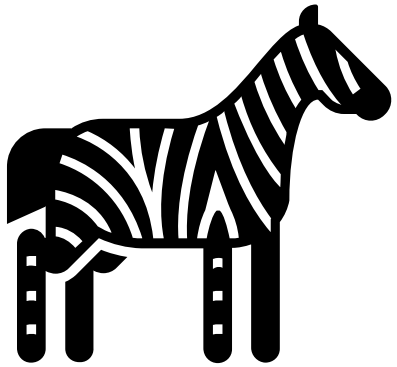
ORR = Overall Response Rate; OS = Overall Survival; PFS = Progression Free Survival; R/R = relapsed / refractory, RP2D = recommended Phase 2 dose.

Opportunities to Expand Beyond R/R PCNSL



R/R = relapsed / refractory; PCNSL = primary central nervous system lymphoma; DLBCL = diffuse large B cell lymphoma; SoC = standard of care; MTX = methotrexate.

GB5121 is a Differentiated BTK Inhibitor Poised to Meet a High Unmet Need



Underserved Rare Disease Population with High Unmet Need

BTKi's Effective, but at Higher-Than-Labeled Doses Causing Tolerability Issues

More Efficiently Penetrates CNS Resulting in Lower Doses

Selectivity Results in Improved Tolerability

Potential Quick Path to Registrational Study

BTKi = Bruton's tyrosine kinase inhibitor.

IV.
Returning to Roots:
GB7208 in Multiple Sclerosis and Other CNS Autoimmune Disorders

Brett E. Skolnick, PhD

Isharat Yusuf, PhD

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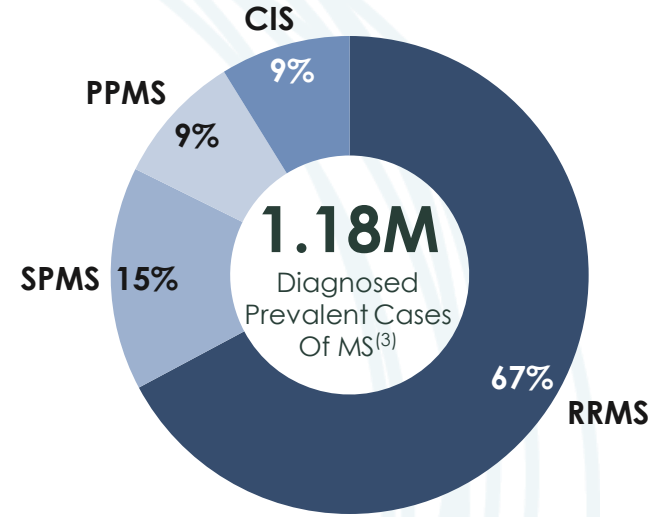
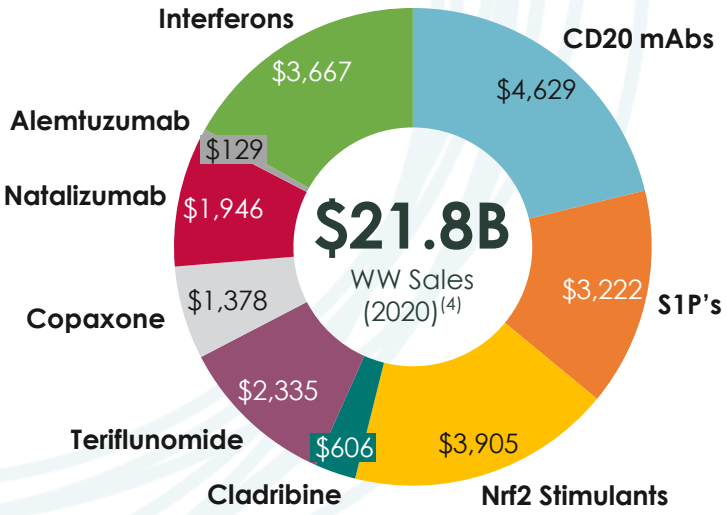
Multiple Sclerosis (MS) Background

- **Despite a multitude of approved therapies for MS, unmet medical needs remain across the disease spectrum**
  - RRMS pts. accumulate disability despite being on therapy[1]
  - 16–27% of treated RRMS pts. convert to SPMS in less than 10 yrs.[2]
  - Only modest efficacy reported in PPMS with approved therapy
- **Opportunity to enhance risk / benefit over approved therapies**
  - Several therapies have safety profiles requiring monitoring
  - IV/SC admin. often required to achieve higher levels of efficacy
  - No approved therapy acts directly on microglia, which are implicated in disease progression & disability accumulation

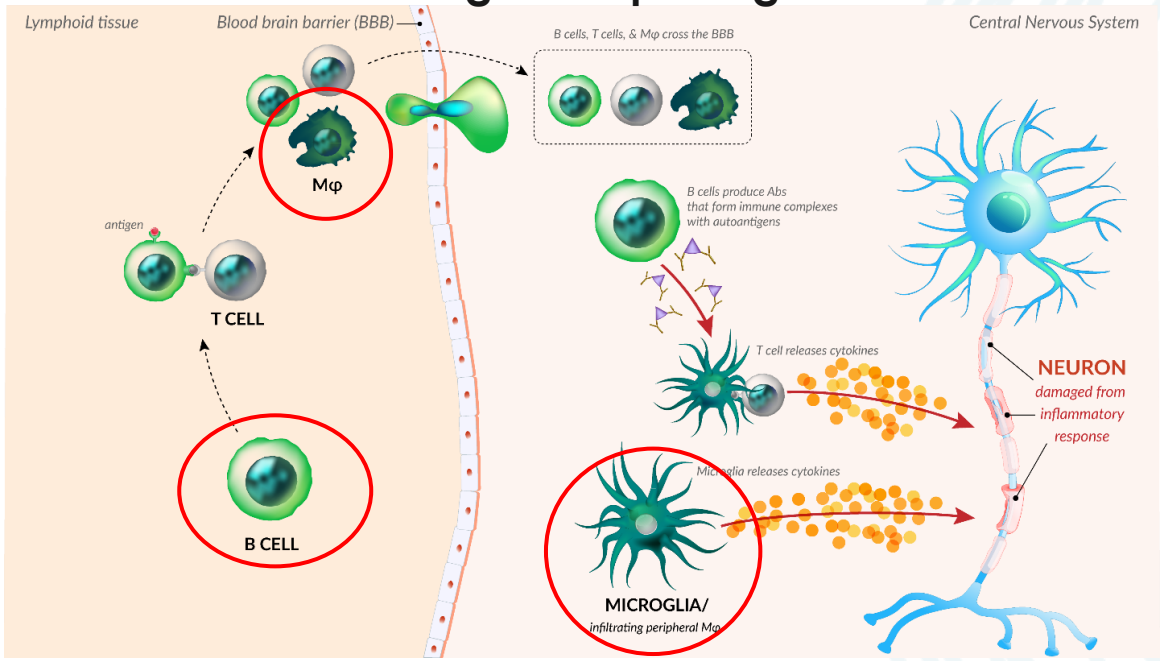




1) Tilling K et al. Health Technol Assess 2016;20:1-48
2) Brown J et al. JAMA. 2019;321(2):175-187
3) Clarivate, Sept. 2021 – Major Markets Only: US, Europe, & Japan
4) EvaluatePharma, Sept. 2021 – Branded sales only
RRMS = Relapsing-Remitting Multiple Sclerosis; pts. = patients; SPMS = Secondary Progressive Multiple Sclerosis; PPMS = Primary Progressive Multiple Sclerosis; CIS = Clinically Isolated Syndrome; SC = subcutaneous injection; mAbs = monoclonal antibodies.

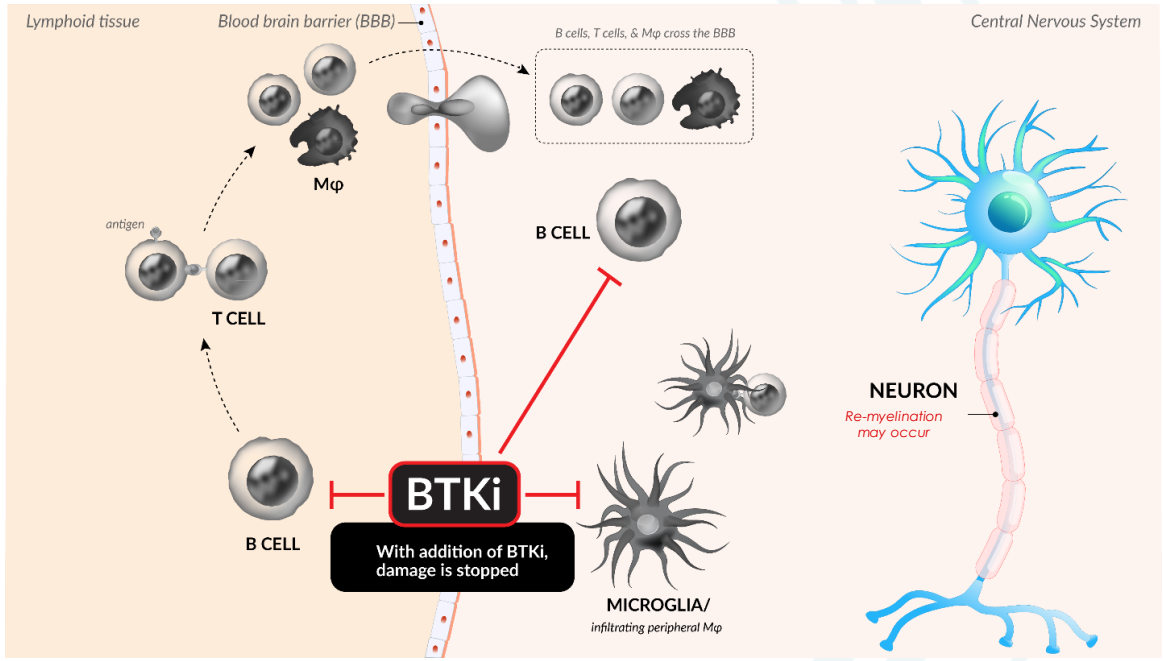
BTK Inhibition Targets Multiple MS Mechanisms

BTK plays a role in both peripheral and CNS-resident cells contributing to MS pathogenesis



- FcR-mediated signaling by immune complex drives pathology of myeloid cells both in periphery and CNS
- B cells, both in the periphery and CNS contribute to pathogenesis in MS
- Presence of B cell follicles in CNS correlates with poor prognosis in SPMS

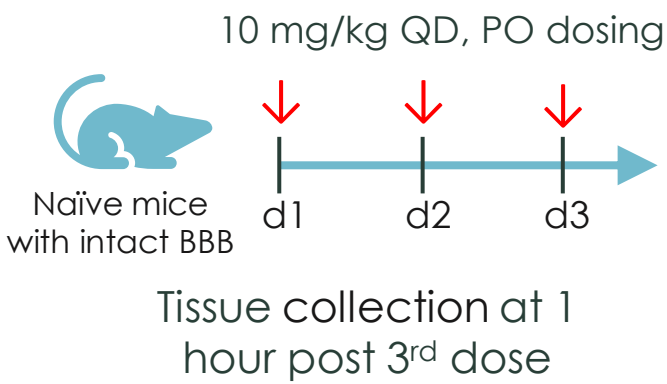
BTKi both in the periphery and CNS targets multiple pathogenic mechanisms



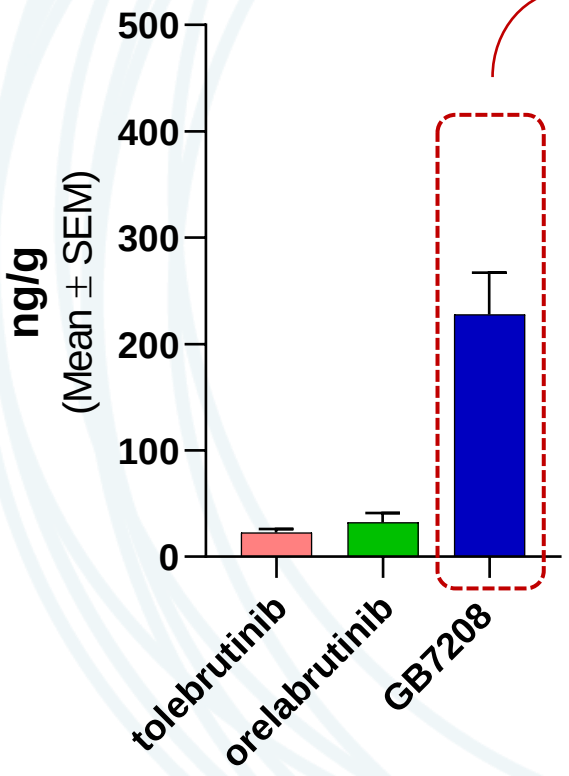
- CNS-penetrant BTKi targets pathogenic mechanisms in both the periphery and CNS
- GB7208 is differentiated from current therapies in MS, such as B cell depletion (ocrelizumab) and S1P receptor modulators

SPMS = Secondary Progressive Multiple Sclerosis; BTKi = Bruton's tyrosine kinase inhibitor.

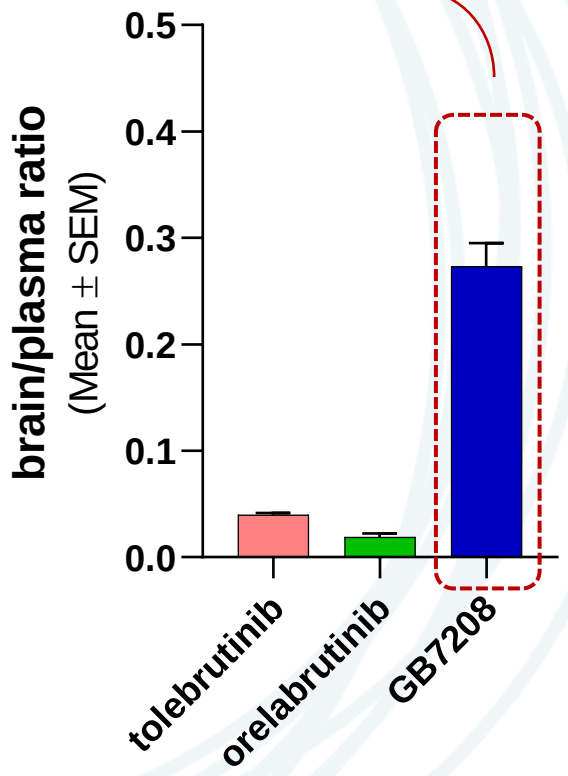
GB7208 Demonstrates Superior Brain Penetrance in Preclinical Models vs. Selected BTK Inhibitors in Development for MS



GB7208 achieves >10x higher CNS exposures vs. other CNS-penetrant BTKi's



Brain Concentration



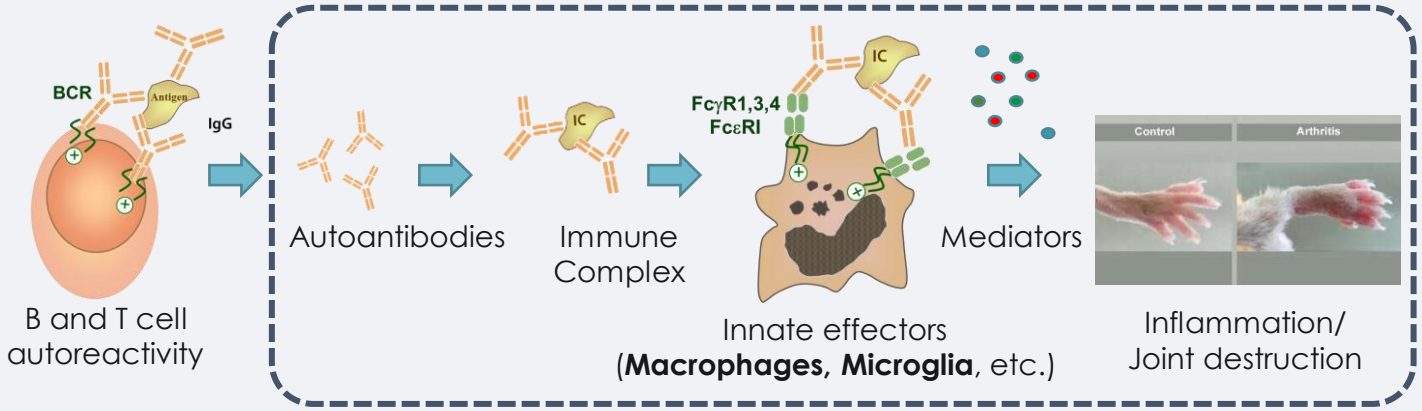
Brain : Plasma Ratio

Source: Internal data.
MS = multiple sclerosis; BBB = blood brain barrier; QD = once daily; PO = oral dosing; SEM = standard error of the mean.

GB7208 Outperforms Tolebrutinib in BTK-Dependent Peripheral Disease Model

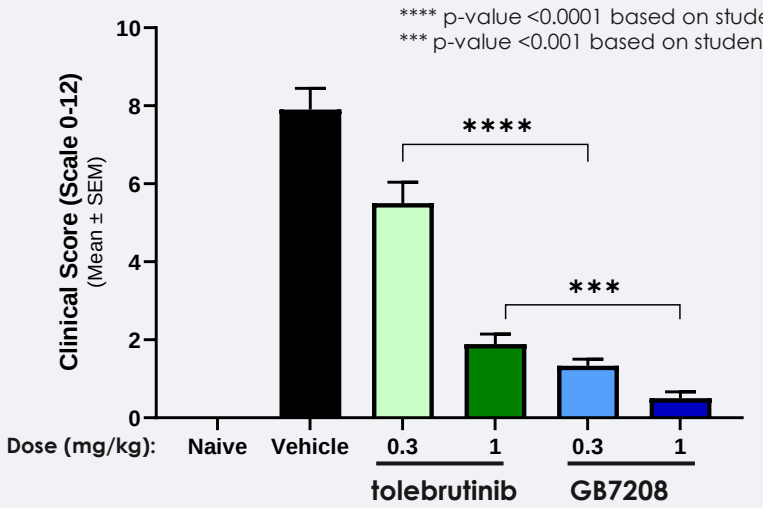
Arthritis Model Is A Gold Standard Pre-clinical Model For BTK Inhibitors

Collagen Antibody-Induced Arthritis (CAIA) Model Captures FcR Driven Inflammation And Disease



Gillooly KM, et al. 2017; Kagari, T et al. 2003

GB7208 Significantly Reduces Disease In CAIA Model

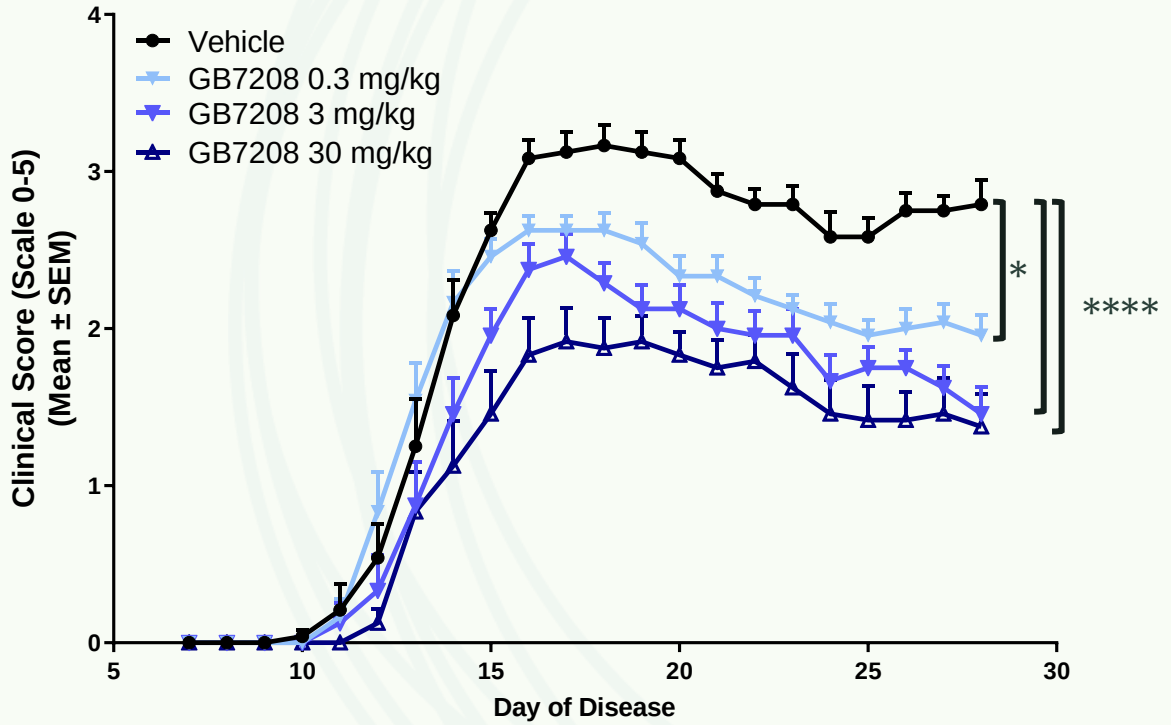


- FcR mediated activation of innate effector cells (macrophages, microglia) is thought to augment neuroinflammation *(Takai, T et al. 2002; Pellerin, K et al., 2021)*
  - Reactive microglia in MS show increased FcγR expression (*Ulvestad E, et al., 1994*)
- **In a head-to-head preclinical study, GB7208 shows superior results vs. tolebrutinib at multiple doses**

Sources: Internal data; Pellerin K. Brain 2021 144(8): 2361-2374; Takai T. Nat Rev Immunol. 2002 2(8): 580-592; Ulvestad E et al. Adv Neuroimmunol. 1994 4(3): 273-281.
BCR = B cell receptor; IFNγ = interferon gamma; SEM = standard error of the mean.

GB7208 Shows Dose-dependent Efficacy in EAE Model of Multiple Sclerosis

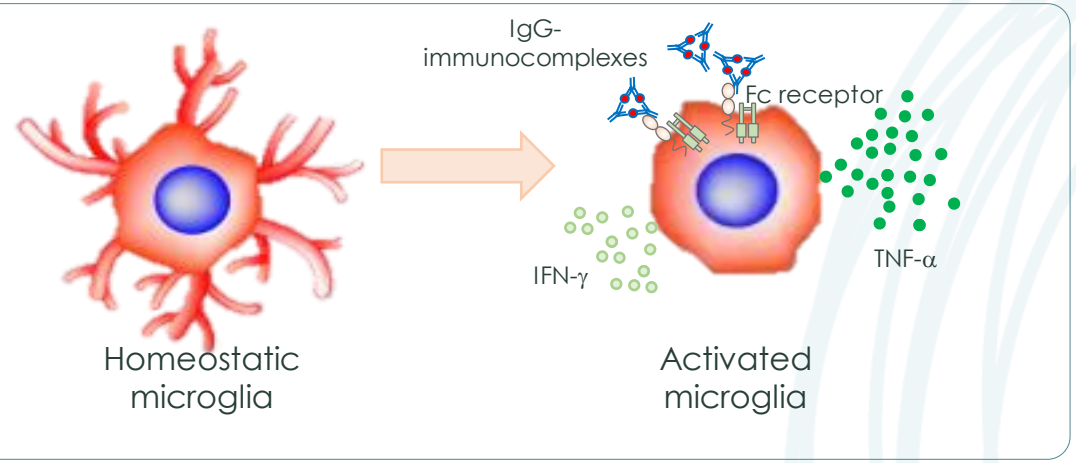
Mice Immunized with MOG_{35-55}
GB7208 Dosed Prophylactically, QD, PO

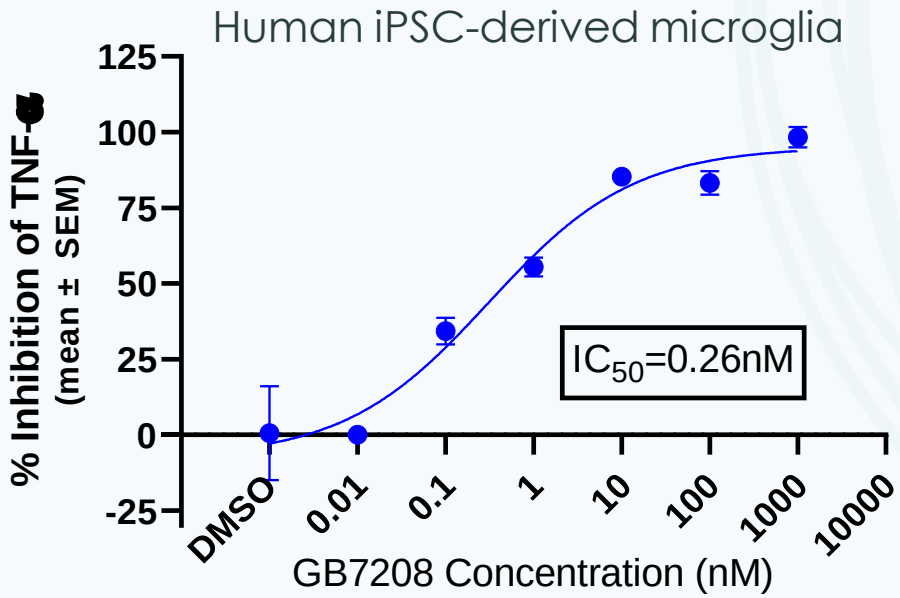


GB7208 demonstrates dose-dependent effect in an EAE model, despite model being primarily driven by activity of peripheral T cells

Source: Internal data; *p-value <0.05, ****p-value <0.0001significance determined by one-way ANOVA of end clinical score.
EAE = experimental autoimmune encephalomyelitis; QD = once daily; PO = oral dosing; SEM = standard error of the mean.

GB7208 Inhibits FcR-dependent Activation of Human Microglia and the Production of TNFα



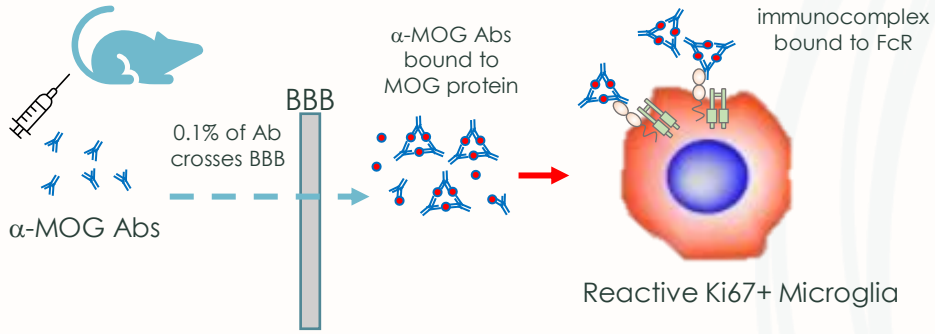


- Autoantibodies in patients with multiple sclerosis could trigger FcR-dependent microglial reactions (*Pellerin, K et al., 2021*)
- TNFα is elevated in progressive multiple sclerosis and causes excitotoxic neurodegeneration (*Rossi et al., 2014*)
- BTK is an important signaling node downstream of FcRs that regulates the activation of myeloid cells and microglia
- GB7208 dose-dependently inhibits the production of TNFα in human microglia stimulated with IgG-immunocomplexes and IFNγ

Sources: Internal data; Pellerin K. Brain 2021 144(8): 2361-2374; Rossi S et al. Mult Scler 2014 20(3): 304-312.
TNFa = tumor necrosis factor alpha; IFNγ = interferon gamma; FcR = Fc receptor; SEM = standard error of the mean.

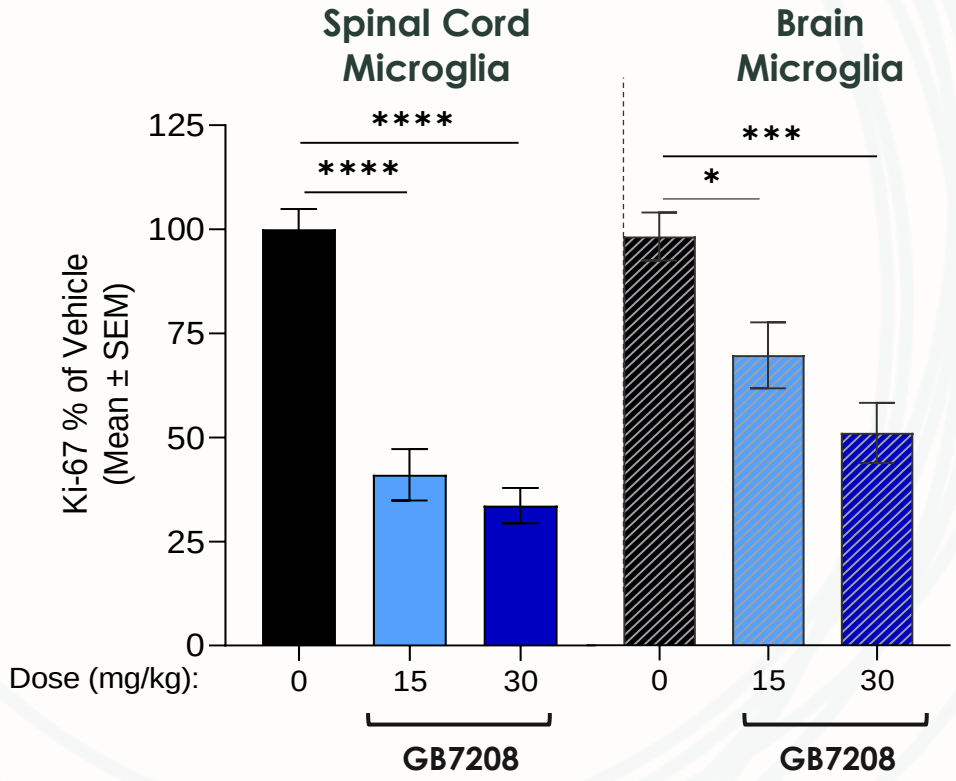
GB7208 Inhibits Proliferation in a Mouse Model of Microglia Activation Induced by Anti-MOG Autoantibodies

Anti-MOG Antibodies Drive Activation of Microglia



- MOG autoantibodies drive activation and proliferation of microglia in mice with an intact BBB
- Activation is driven by BTK-dependent FcR signaling

GB7208 Inhibits Microglia Proliferation *in vivo*



**** p-value <0.0001; *** p-value <0.001, * p-value <0.01 based on student's t-test

Sources: Pellerin K. Brain 2021 144(8): 2361-2374(left figure); Internal data(right figure)
Abs = antibodies; MOG = myelin oligodendrocyte glycoprotein; FcR = Fc receptor; BBB = blood brain barrier; SEM = standard error of the mean.

Expected Timeline to Clinic

2021	2022	
4Q	1H	2H

Today

IND-Enabling Studies

First-in-Human Phase 1

GB7208 expected to enter the clinic 2H:2022 and into MS patients in 2023

Potential Indications for GB7208

NEURO**INFLAMMATION**

- Relapsing / Remitting MS
- Secondary Progressive MS

NEURO**DEGENERATION**

- Primary Progressive MS
- Parkinson's Disease
- Alzheimer's Disease
- ALS

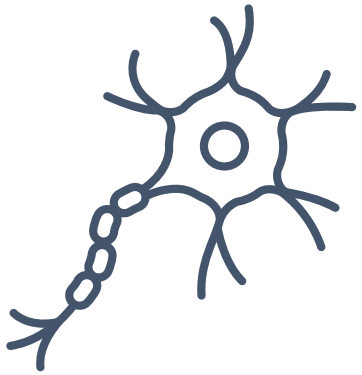
MS = multiple sclerosis; ALS = amyotrophic lateral sclerosis.

GB7208 is Positioned to Address Shortcomings of Prior BTK Generations

- Optimal treatment of neuroinflammatory and neurodegenerative diseases likely requires target engagement in both the periphery and the central nervous system
- Preclinically, GB7208 demonstrates:
  - Best-in-class CNS penetrance vs. reference molecules in development for MS
  - Potent inhibition of microglial activation
  - Superiority vs. reference molecules, including tolebrutinib, in pre-clinical models of disease activity
- GB7208 is expected to enter the clinic in 2H:22 and has potential in a variety of indications, such as RRMS and Progressive Multiple Sclerosis

RRMS = relapsing / remitting multiple sclerosis.

Optimal Characteristics for a Neuro-Inflammatory and -Degenerative Therapy



Addresses Underlying Biology

Targets Specific Cell Populations

Penetrates CNS

Selectivity Results in Improved Tolerability

Halts Disease Progression

V.
Closing Remarks

Faheem Hasnain

Diversified Clinical Immunology Pipeline

PROGRAM	CLASS (Route of Admin.)	INDICATION	RESEARCH	PRE-CLINICAL	PHASE 1	PHASE 2	PHASE 3	RIGHTS
Seralutinib (GB002)	PDGFR, CSF1R, c-KIT Inhibitor (Inhaled)	Pulmonary Arterial Hypertension (PAH)				Phase 2 Ongoing		WW
GB004	Gut-Targeted, HIF-1a Stabilizer (Oral)	Inflammatory Bowel Disease (IBD) (Ulcerative Colitis, UC)				Phase 2 Ongoing		WW
GB5121	CNS-Penetrant, BTK Inhibitor (Oral)	Primary CNS Lymphoma (PCNSL)			Phase 1 Ready			WW
GB7208	CNS-Penetrant, BTK Inhibitor (Oral)	Multiple Sclerosis (MS)						WW
Research Programs	Multiple Programs	Oncology, Immunology						WW

GOSSAMER IS WELL CAPITALIZED WITH $406 MILLION IN CASH†

ww = worldwide.
†As of 6/30/21. USD. Includes cash, cash equivalents and marketable securities.

CNS-Penetrant BTK Inhibitor Clinical Milestones

Population	Milestone	Timing
GB5121 (Oncology)		
Healthy Volunteers	Initiate Phase 1	4Q21
PCNSL	Initiate Phase 1b/2	1H22
GB7208 (Autoimmune, Multiple Sclerosis)		
Healthy Volunteers	Initiate Phase 1	2H22

ADDITIONAL CLINICAL MILESTONES DO NOT CHANGE ANTICIPATED CASH RUNWAY[1]

1) Consistent with Gossamer's last public disclosure on its cash runway, Gossamer expects the combination of current cash, cash equivalents and marketable securities, and access to its debt facility will be sufficient to fund its operating and capital expenditures into the second half of 2023.

Question & Answer Session

Thank you for joining us!