

Corporate Presentation

NOVEMBER 2024



Forward Looking Statements

This presentation contains “forward-looking statements” within the meaning of the Private Securities Litigation Reform Act of 1995, including without limitation statements regarding: future expectations, plans and prospects for Climb Bio, Inc. (“Climb Bio”); the anticipated benefits of the acquisition of Tenet Medicines, Inc.; expectations regarding budoprutug’s therapeutic benefits, clinical potential and clinical development; the trial design for planned clinical trials of budoprutug; plans to optimize the administration of budoprutug; the anticipated timelines for initiating clinical trials of budoprutug; the sufficiency of Climb Bio’s cash resources for the period anticipated and other statements containing the words “anticipate,” “believe,” “continue,” “could,” “estimate,” “expect,” “intend,” “may,” “plan,” “potential,” “predict,” “project,” “should,” “target,” “would,” “will,” “working” and similar expressions. Forward-looking statements are based on management’s current expectations of future events and are subject to a number of risks and uncertainties that could cause actual results to differ materially and adversely from those set forth in, or implied by, such forward-looking statements. Climb Bio may not actually achieve the plans, intentions or expectations disclosed in these forward-looking statements, and you should not place undue reliance on these forward-looking statements. These risks and uncertainties include, but are not limited to, important risks and uncertainties associated with: the ability of Climb Bio to timely and successfully achieve or recognize the anticipated benefits of its acquisition of Tenet Medicines, Inc.; changes in applicable laws or regulation; the possibility that Climb Bio may be adversely affected by other economic, business and/or competitive factors; Climb Bio’s ability to advance budoprutug on the timelines expected or at all and to obtain and maintain necessary approvals from the U.S. Food and Drug Administration and other regulatory authorities; obtaining and maintaining the necessary approvals from investigational review boards at clinical trial sites and independent data safety monitoring boards; replicating in clinical trials positive results found in early-stage clinical trials of budoprutug; competing successfully with other companies that are seeking to develop treatments for systemic lupus erythematosus, immune thrombocytopenia and membranous nephropathy and other immune-mediated diseases; maintaining or protecting intellectual property rights related to budoprutug and/or its other product candidates; managing expenses; and raising the substantial additional capital needed, on the timeline necessary, to continue development of budoprutug and any other product candidates Climb Bio may develop. For a discussion of other risks and uncertainties, and other important factors, any of which could cause Climb Bio’s actual results to differ materially from those contained in the forward-looking statements, see the “Risk Factors” section, as well as discussions of potential risks, uncertainties and other important factors, in Climb Bio’s most recent filings with the U.S. Securities and Exchange Commission. In addition, the forward-looking statements included in this presentation represent Climb Bio’s views as of the date hereof and should not be relied upon as representing Climb Bio’s views as of any date subsequent to the date hereof. Climb Bio anticipates that subsequent events and developments will cause Climb Bio’s views to change. However, while Climb Bio may elect to update these forward-looking statements at some point in the future, Climb Bio specifically disclaims any obligation to do so, except as required by law.



Together We Can Reach Higher Ground

At Climb Bio, we believe elevating relationships leads to more meaningful insights, better answers, and ultimately, to more inspired medicines for patients living with immune-mediated diseases



More than 2.5 million Americans suffer from a B-cell mediated disease



Committed to enhancing the patient experience



Driven to becoming a leader in development for immune-mediated diseases

Building A Leading Immune-Mediated Disease Focused Company



Immune-Mediated Disease Focus

Focused solely on immune-mediated disease with budoprutug, a potentially best-in-class anti-CD19 antibody, our cornerstone asset



Broad Potential

Budoprutug in development for pMN, ITP, and SLE with the prospect of expanding into additional indications as well as a potential subcutaneous formulation



Well Resourced

Funded through 2027 enabling delivery of key value inflection points through the initiation of multiple clinical programs and a subcutaneous formulation clinical trial



Experienced Team

Track record of execution and operational results

Team Highlights

Building a highly-credentialed and experienced development organization focused on execution



Aoife Brennan
President and CEO



William Bonificio
Interim CBO



Brett Kaplan
COO



Nishi Rampal
SVP, Clinical Development



Kate Hecht
SVP, Program Management



Jan Hillson
Senior Clinical Advisor



Emily Pimblett
CAO



Gang (Gary) Hao
VP, CMC



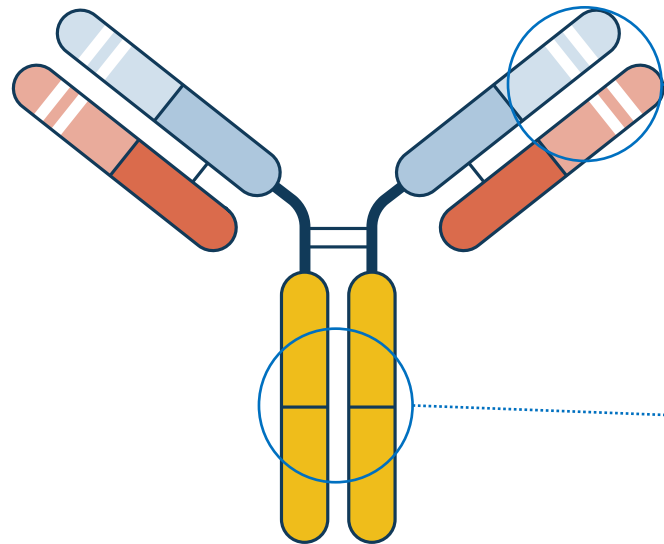
Janaki M. Subramanyam
VP, Regulatory Affairs



Jay Mitchell
VP, Clinical Operations

Budoprutug: Fc-Enhanced Anti-CD19 mAb

Designed to treat immune-mediated diseases



Budoprutug is a highly potent anti-CD19 mAb containing a low-fucosylated Fc region, leading to enhanced effector function and highly potent ADCC

Unique attributes driving differentiation & positioning

HIGH AFFINITY

18 pM

binding affinity to CD19 to counter low antigen density

ADCC-ENHANCED

>100x potency

vs. wild-type IgG1 to drive deep & durable B cell depletion

HIGH CONCENTRATION

≥175 mg/mL

with low viscosity for low volume, SC injection

✓ **Potential for best-in-class efficacy**

Rapid, deep, and durable B cell depletion at doses as low as 100 mg

✓ **Opportunity for patient-tailored approach to treatment**

Potential to provide IV and/or SC offerings where favorable to patient and point-of-care

✓ **Optimized dosing and tolerability**

Potential for induction and maintenance dosing paradigm with favorable safety, tolerability profile

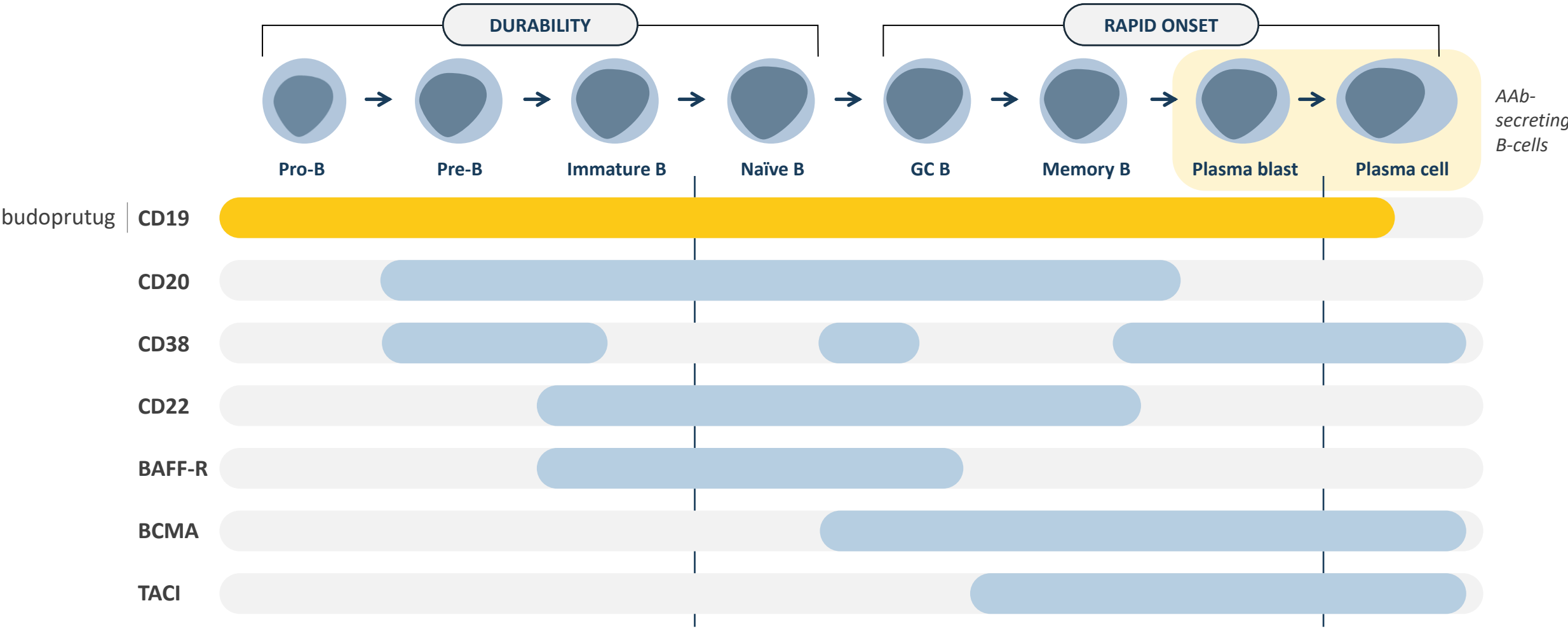
✓ **Pipeline-in-a-molecule potential**

3 distinct opportunity sets:

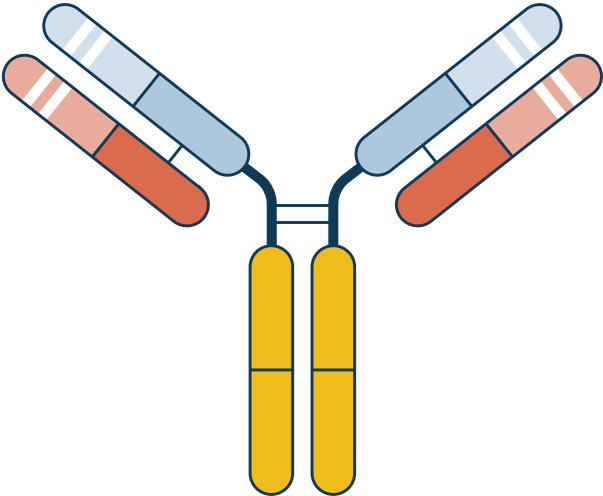
IgG4-Mediated, Complex Systemic, & Primarily Single Organ IgG1 – 3

CD19 Expression on Autoantibody-Secreting Cells (ASCs)

CD19-targeted therapy potentially enables rapid onset and durability



Budoprutug: Anti-CD19 mAb Designed to Treat a Broad Range of B-Cell Mediated Diseases



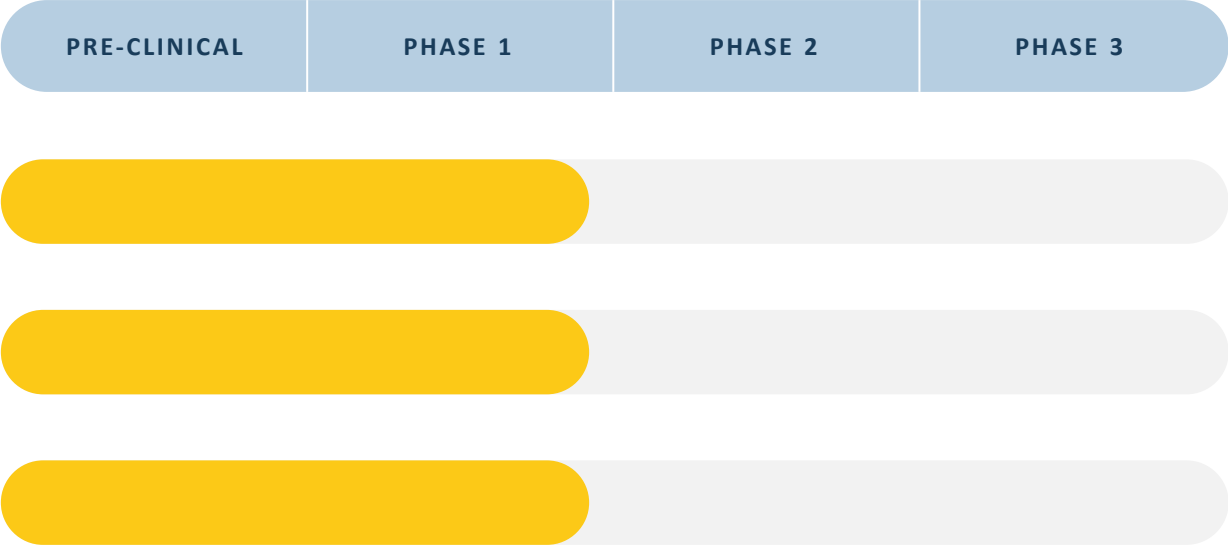
Budoprutug
Anti-CD19
Fc⁺, mAb

INDICATION(S)

Membranous Nephropathy

Immune Thrombocytopenia*

Systemic Lupus Erythematosus



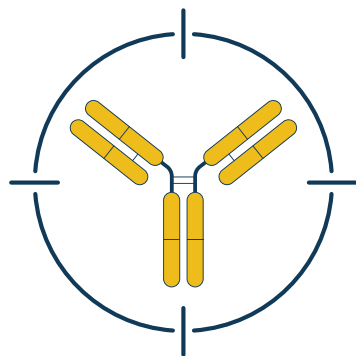
CD19 is a promising target antigen for AAb-mediated diseases as a clinically-validated MoA

Additional potentially addressable indications across multiple therapeutic areas

Aiming to advance potentially best-in-class mAb to late-stage clinical trials

Corporate Strategy & Vision

Climb is well-positioned to advance budoprutug across three distinct opportunity sets



Primarily IgG4-Mediated

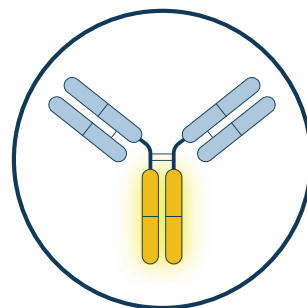
Clear pathophysiology supporting targeting of CD19-expressing B-cells

Lead indication

Primary Membranous Nephropathy

Opportunity to Differentiate

Potential for “immune reset”, improved efficacy



Primarily Single Organ IgG1 - 3

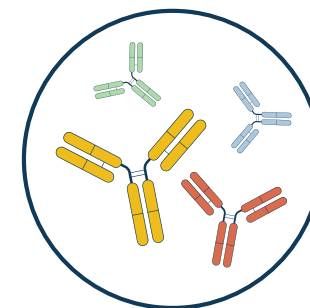
Orphan diseases with compelling clinical proof-of-concept using B-cell depletion

Lead indication

Immune Thrombocytopenia

Opportunity to Differentiate

Demonstrate efficacy in relapsing and/or refractory patients



Complex Systemic

Multi-organ, systemic diseases with heterogenous patient populations

Lead indication

Systemic Lupus Erythematosus

Opportunity to Differentiate

Improve efficacy while balancing safety, tolerability, and convenience

Pipeline In A Molecule

Multiple potential next wave indications

Primarily IgG4-Mediated

MG (MuSK)
~7k

MN
~70k

CIDP
~20k

IgG4-RD
~20k

PV
~15k

Primarily single organ IgG1 - 3

MS
~730k

ANCA-AAV
~140k

MG (AChR)
~68k

ITP
~65k

BP
~40k

NMOSD
~25k

CIDP
~10k

Complex Systemic

RA
~1300k

Sjogren's
~340k

SLE
~240k

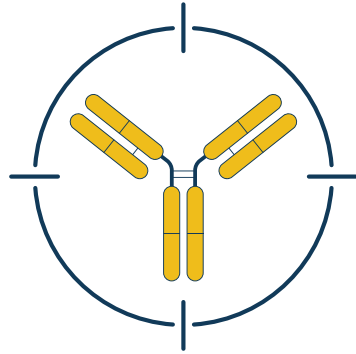
SSc
~85k

MG MuSK= Myasthenia Gravis muscle-specific tyrosine kinase; SLE = Systemic Lupus Erythematosus, MN = Membranous Nephropathy, ITP = Immune Thrombocytopenia NMOSD = Neuromyelitis optica spectrum disorder, BP = Bullous pemphigoid, ANCA-AAV = antineutrophil cytoplasmic antibody-associated vasculitides, SSc = Systemic sclerosis; CIDP = Chronic inflammatory demyelinating polyradiculoneuropathy, IgG4-RD = IgG4 related disease, RA = Rheumatoid arthritis, MS = Multiple sclerosis

Prevalence references: SLE (Izmirly 2021), ITP (internal research), MN (internal research), MG (Ye 2024), SSc (Fan 2020), ANCA-AAV (Berti 2017), CIDP (Laughlin 2009), BP (Wertenteil 2018), NMOSD (Briggs 2024), IgG4-RD (Wallace 2023), RA (Hunter 2017), Sjogren's (Maciel 2017), MS (Wallin 2019)

Primarily IgG4-Mediated

Primary membranous nephropathy



Primarily IgG4-Mediated

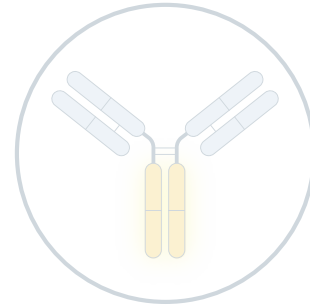
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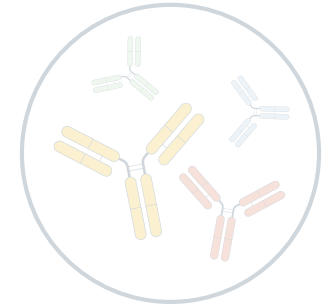
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Complex Systemic

Multi-organ, systemic diseases with heterogenous patient populations

Lead indication

Systemic Lupus Erythematosus

Opportunity to Differentiate

Improve efficacy while balancing safety, tolerability, and convenience

Primary Membranous Nephropathy (pMN): Indication Overview

Pathophysiology	<ul style="list-style-type: none">Primary MN (pMN) is caused by autoantibody-mediated destruction of podocytes at the glomerular basement membrane (GBM)Anti-PLA2R antibodies occur in 60% to 80% of patients with pMN
Symptoms & Diagnosis	<ul style="list-style-type: none">Symptoms: proteinuria, hypoalbuminemia, edema, dyspnea, fatigue, hyperlipidemia, nephrotic syndromeDiagnosis: kidney biopsy and/or serum anti-PLA2R antibody assays
Epidemiology	<ul style="list-style-type: none">MN incidence: 1/100K and 80% are pMN; conservatively ~100K patients in US and EU20-40% of patients are refractory to currently available lines of treatment
Natural History	<ul style="list-style-type: none">Severity of proteinuria is associated with poor outcomes and severe proteinuria (i.e., >10g/day) leads to end stage renal disease in ~50% of patients by 5 yearsPatients are at increased risk for kidney failure and life-threatening thromboembolic eventsRemission of proteinuria is an approvable endpoint in nephrotic patients
Standard of Care	<ul style="list-style-type: none">Treatment is aimed at reducing proteinuria, though all therapies are off-labelKDIGO guidelines recommend treatment based on risk, primarily based on eGFR and PLA2R levelsRituximab (RTX) is considered 1st line therapy; MENTOR trial showed complete remission w/ RTX superior to calcineurin inhibitors (CNIs) at 24 months

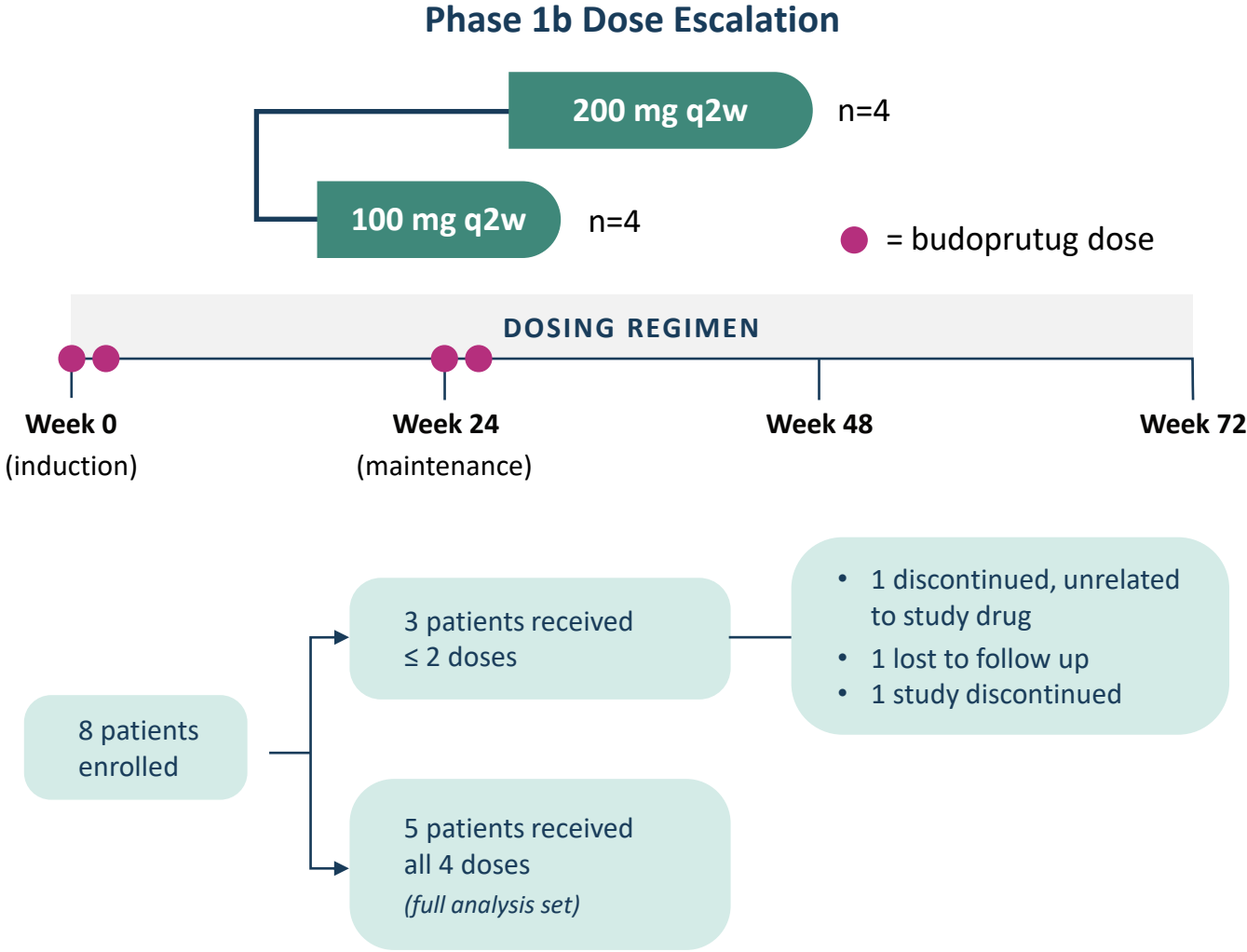
PLA2R = phospholipase A2 receptor; eGFR = estimated glomerular filtration rate; KDIGO = kidney diseases improving global outcomes
Ronco P 2021; Teisseyre M 2022; KDIGO 2021 guidelines; Ahmad, S.B. 2020; Swaminathan S 2006; Donadio JV 1988; Schieppati A 1993; Shiiki H 2004

Budoprutug Phase 1b Study Design

Proof-of-concept, open label, dose escalating study in adult subjects with pMN

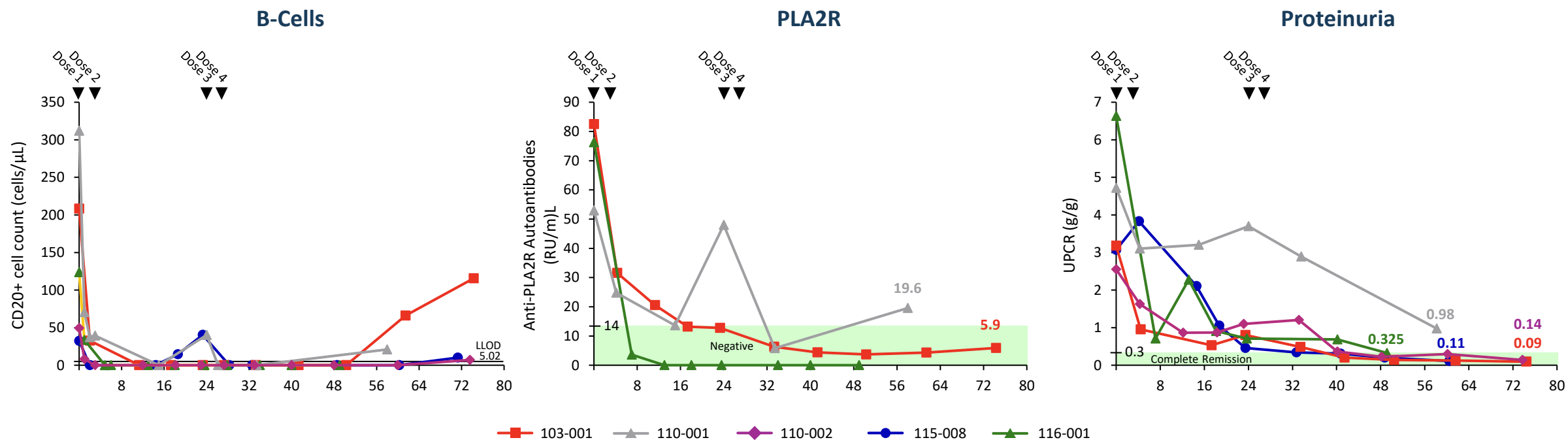
ELIGIBILITY	<ul style="list-style-type: none"> • UPCR ≥ 2.0 g/g • B-cell count $>LLN$ (80 cells/μL)
DESIGN	<ul style="list-style-type: none"> • Dose escalation & expansion • 18-month follow-up
DOSING	<p>2 doses 14 days apart</p> <ul style="list-style-type: none"> • 100 mg • 200 mg
ENDPOINTS	<ul style="list-style-type: none"> • Safety, tolerability & PK • PD markers (B-cells, PLA2R) • Proteinuria response

PARAMETER	BASELINE (MEAN)
B-cells	145 cells/ μ L
PLA2R	71 RU/mL
UPCR	4.03 g/g



Budoprutug administration was associated with resolution of proteinuria and immunological remission

Data for pMN subjects (n = 5) who have completed ≥48-weeks



Complete remission achieved in 60% (3/5) of patients at Week 48

- Partial remission (>50% reduction in UPCR + UPCR < 3.5 g/g) achieved in all (5/5) subjects
- Complete B-cell depletion (CD20⁺ count < 5 cells/μL) achieved in all (5/5) subjects
- Anti-PLA2R Ab negativity (< 14 RU/mL) achieved in all (3/3) evaluable subjects
- 2 subjects on study that have not entered complete remission have achieved PLA2R negativity (serological remission)



Safety

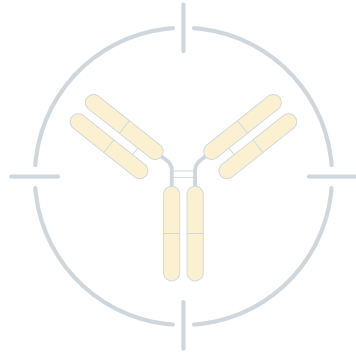
Budoprutug was generally well tolerated at doses of up to 200mg

8 Patients received at least one injection of budoprutug and are included in the safety analysis population

- ✓ There were no deaths on study
- ✓ There were 3 SAEs, none of which were considered to be related to budoprutug by the investigator
- ✓ No discontinuations due to AE
- ✓ No dose limiting toxicities (DLTs) were observed
- ✓ 4 patients reported infections on study of which 3 were cases of COVID-19 and 1 was bacterial pneumonia

Primarily Single Organ IgG1 - 3

Immune thrombocytopenia



Primarily IgG4-Mediated

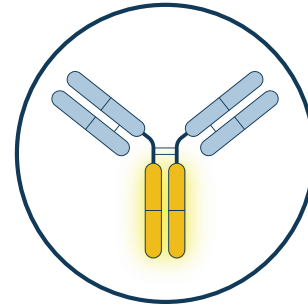
Clear pathophysiology supporting targeting of CD19-expressing B-cells

Lead indication

Primary Membranous Nephropathy

Opportunity to Differentiate

Potential for “immune reset”, improved efficacy



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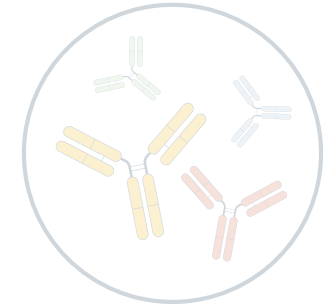
Orphan diseases with compelling clinical proof-of-concept using B-cell depletion

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Demonstrate efficacy in relapsing and/or refractory patients



Complex Systemic

Multi-organ, systemic diseases with heterogenous patient populations

Lead indication

Systemic Lupus Erythematosus

Opportunity to Differentiate

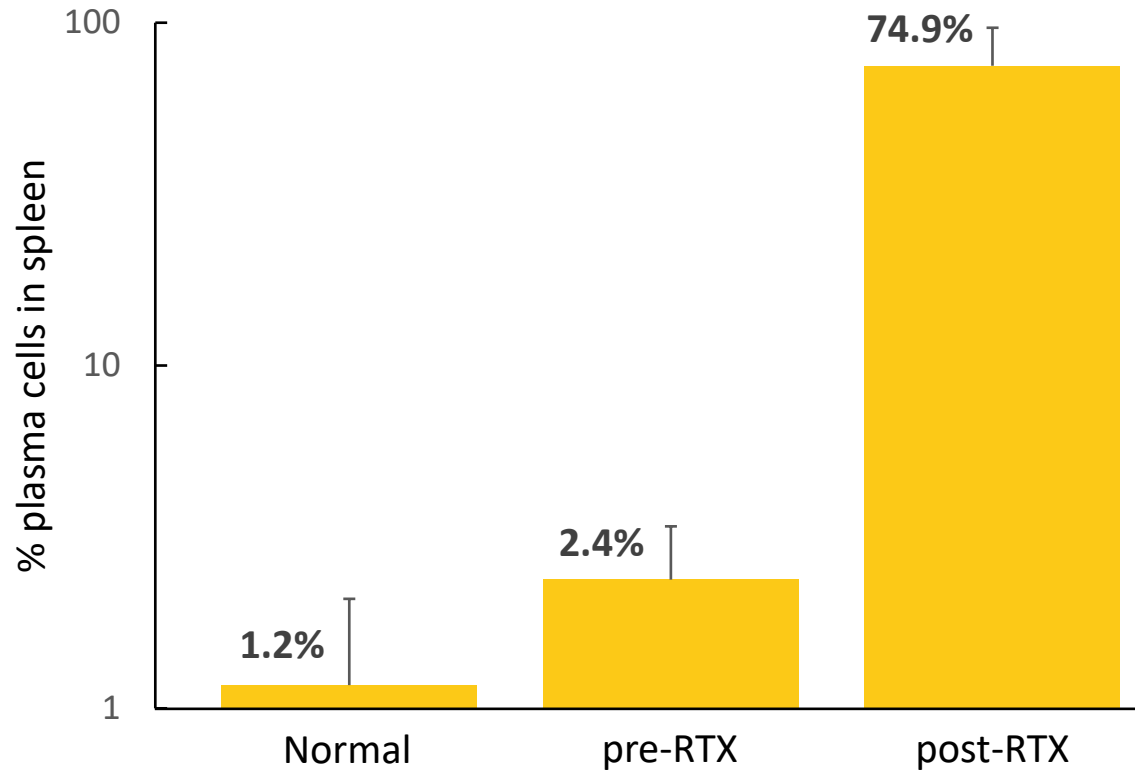
Improve efficacy while balancing safety, tolerability, and convenience

Immune thrombocytopenia (ITP): Indication Overview

Pathophysiology	<ul style="list-style-type: none">• ITP is an autoimmune disease characterized by low platelets resulting in bruising and hemorrhage• Antiplatelet autoantibodies lead to accelerated removal of platelets by macrophages with bone marrow compensation
Symptoms & Diagnosis	<ul style="list-style-type: none">• Symptoms: bruising (petechiae and purpura), bleeding episodes, and fatigue• Diagnosis: Low platelet count, supported by additional blood tests i.e., CBC and blood smear, antiplatelet antibody test, bone marrow aspiration if needed
Epidemiology	<ul style="list-style-type: none">• The estimated global prevalence of ITP is around 200,000 patients worldwide• In the US, there are 81,000 adults with chronic ITP with >24,000 refractory to 2nd line treatment
Natural History	<ul style="list-style-type: none">• Most children have spontaneous remission within a few weeks or months• While adults often stabilize on 1st line therapy, the majority eventually relapse or become refractory, necessitating treatment with 2nd and at times 3rd line therapies, splenectomy in hard-to-treat situations can be considered

ITP patients likely fail rituximab due to the presence of CD19+CD20- B-cells

CD19⁺/CD20⁻ plasma cells expand within B-cell niches post anti-CD20 treatment

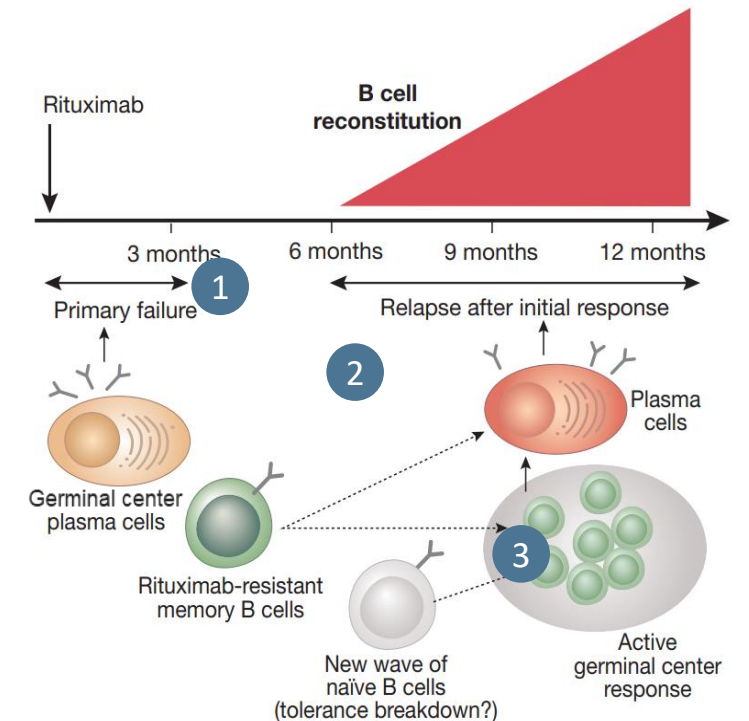


Primary failure

- 1 Pre-existing CD20⁻ PCs

Relapse after initial response

- 2 Pre-existing CD20⁻ B-cells
- 3 *De novo* CD20⁻ B-cells



ITP Phase 2 Trial Design & Objectives*

Planned single arm, open-label study focused on platelet response and B-cell depletion

POPULATION

- Insufficient response to 1 or more prior therapies
- Platelet count $<30,000/\mu\text{L}$
- B cells $> 40 /\mu\text{L}$

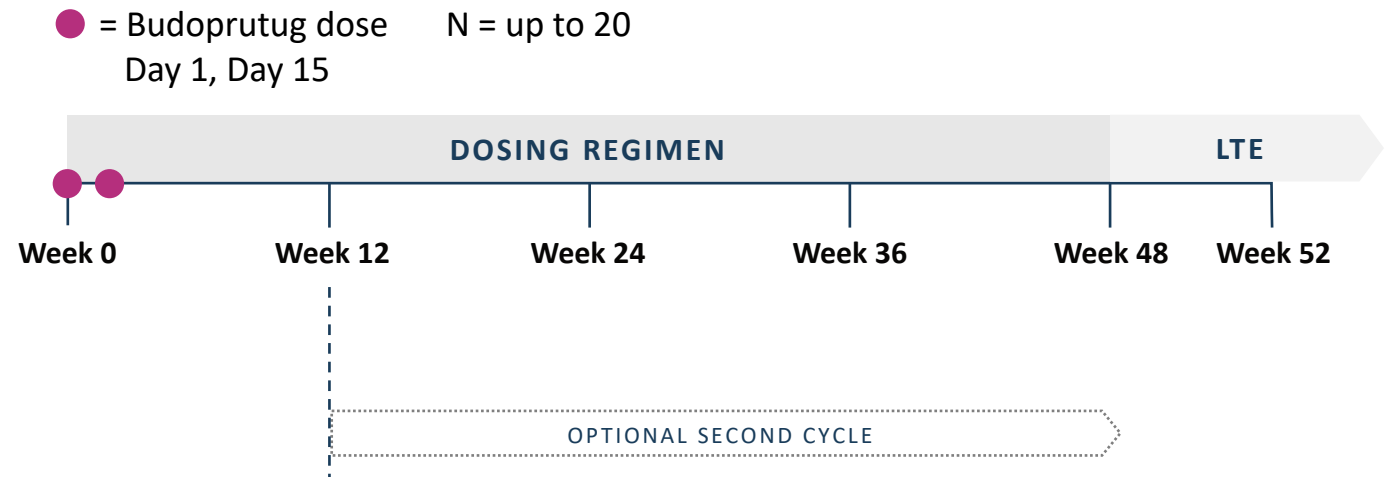
PRIMARY OBJECTIVE

- To evaluate the safety and tolerability of budoprutug in subjects with ITP
- To evaluate the efficacy of budoprutug on platelet counts

KEY SECONDARY/EXPLORATORY OBJECTIVES

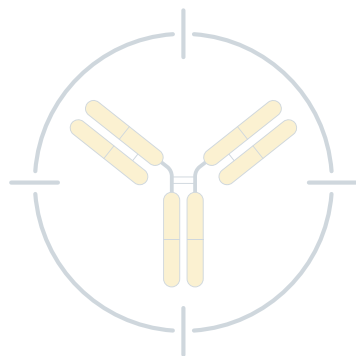
- To evaluate subject reported outcomes/quality of life (QoL) measures
- To evaluate PK/PD (dose relationship) profile in subjects with ITP

Potential for treat to target approach



Complex Systemic

Systemic Lupus Erythematosus



Primarily IgG4-Mediated

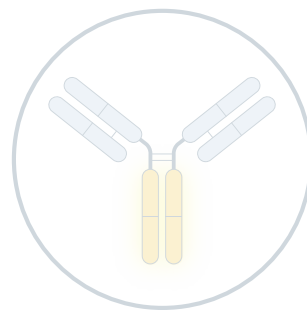
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Primary Membranous Nephropathy

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Primarily Single Organ IgG1 - 3

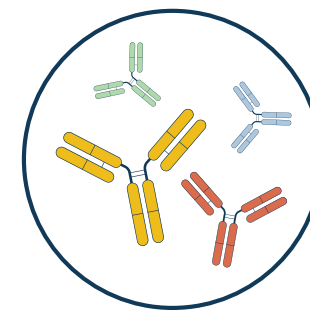
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Systemic Lupus Erythematosus (SLE): Indication Overview

Pathophysiology	<ul style="list-style-type: none">• SLE comprises a group of disorders characterized by the generation and persistence of autoreactive lymphocytes and autoantibodies that directly interfere with critical functions, target cells for destruction, and damage tissues through immune complex depositions
Symptoms & Diagnosis	<ul style="list-style-type: none">• Diagnosis is clinical, based on serology and organ system involvement without other cause• Symptoms and severity vary widely across patients. Nephritis is the most common organ system threat; fatigue and cognitive dysfunction are the most common disabling manifestations
Epidemiology	<ul style="list-style-type: none">• US burden is ~240,000 active patients; ~80,000 with lupus nephritis• Global prevalence is ~1-2 per 100,000 adults, with 9:1 female predominance
Standard of Care	<ul style="list-style-type: none">• Corticosteroids to rapidly control inflammation• Antimalarials for rash and to reduce flares• Small molecule immune suppressants to reduce corticosteroid use• Belimumab (B-cell activating factor blockade), Anifrolumab (type 1 interferon receptor blockade) for refractory disease• Rituximab used off label
Unmet Need	<ul style="list-style-type: none">• 10% – 20% are refractory to current therapies; much larger numbers are dependent on corticosteroids• Relapses, especially of nephritis, vasculitis, thrombosis, lead to cumulative damage and organ failure• Fatigue and cognitive dysfunction respond poorly, impairing participation and quality of life• Treatment-associated burden of cardiovascular mortality, infection, and neoplasm risk

Targeting B-Cells Has Shown Promise in SLE

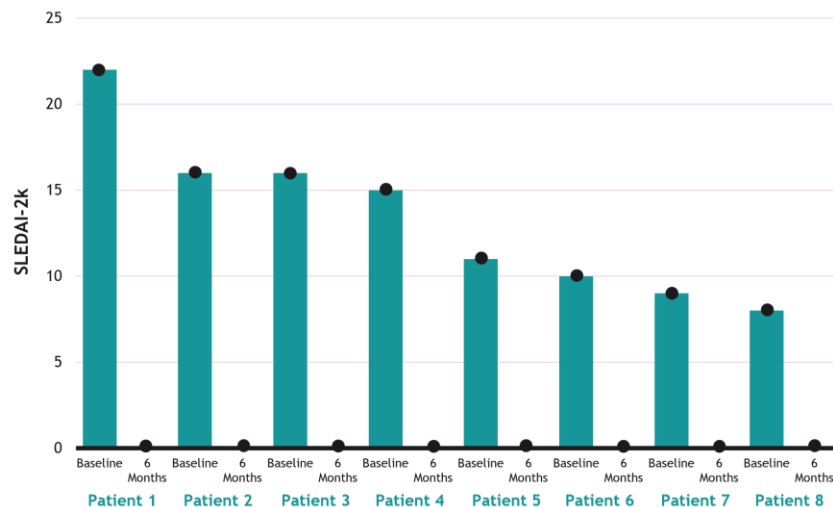
DRUG	CO.	TARGET	ROUTE	STAGE	SLE CLINICAL DATA
Rituximab	Roche	CD20	IV, SC	Phase 3 SLE and LN (failed)	Phase 3 (vs. placebo, n=257) <u>Week 52 SRI-4</u> 27.2% (vs. 22.7%)
Obinutuzumab	Roche	CD20	IV	Phase 3 LN (completed; results pending)	Phase 2 (vs placebo, n=125) <u>Week 52 CRR</u> 35% (vs 23%) <u>Week 104 CRR</u> 41% (vs 23%)
Belimumab	GSK	BAFF	SC	SLE and LN (marketed)	Phase 3 (vs. placebo, n=836) <u>Week 52 SRI-4</u> 61% (vs. 48%)

Patients with poor response to depletion of CD20+ B cells are characterized by any among:

- Inadequate CD20+ B cell depletion
- Persistence of CD19+ self-reactive B cell subsets
- Continued production of pathogenic autoantibodies by plasma cells
- Rapid recovery of pathogenic B cell subsets

Targeting B-Cells Has Shown Promise in SLE

CD19 CAR-T potentially 'curative' at 6 months

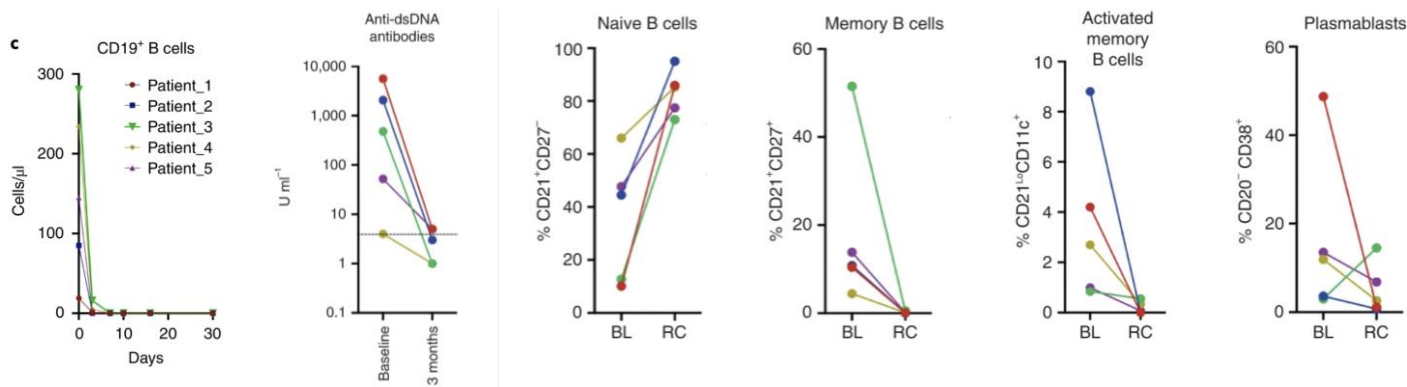


SLE patient data from Müller 2024, Mackensen 2022, SLEDAI-2K = Systemic Lupus Erythematosus Disease Activity Index 2000

Depletion of CD19+ B cells with CD19+ CAR-T cells addresses some of the shortcomings of existing therapy. Limitations include:

- Challenging to make autologous cells from highly pre-treated patients
- Toxicity associated with required conditioning therapy
- Risk of cytokine release syndrome and immune effector cell associated neurotoxicity syndrome
- Access limited by complexity and cost

Response is associated with rapid depletion of circulating B cell and autoantibodies, followed by recovery of relatively benign B cell subsets



SLE Trial Design & Objectives*

Planned open-label, dose escalation with augmented B cell and antibody analyses

POPULATION

- Diagnosis of SLE, with active disease based on SLEDAI
- Seropositive, with elevated ANA, anti-dsDNA, ENA or APL
- <20 mg prednisone by Day -28; stable limited background

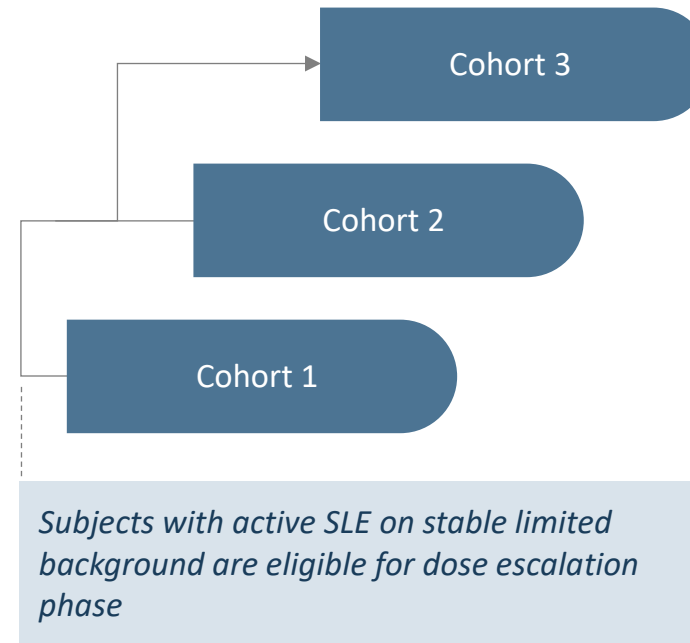
PRIMARY OBJECTIVE

- To evaluate the safety and tolerability of budoprutug in subjects with SLE

KEY SECONDARY/EXPLORATORY OBJECTIVES

- To evaluate the effects of budoprutug on B-cell depletion (prioritized pharmacodynamic [PD] response), autoantibody levels, and protective antibody levels
- To evaluate the PK and PK/PD (dose relationship) profile in patients with SLE
- To evaluate preliminary signs of clinical activity in patients with SLE
- To determine the kinetics of re-population of B-cell subsets and antibodies after depletion

Dose Escalation



Potential Dose Expansion

Dose regimen selected based on Dose Escalation

Subjects with active SLE despite adequate trial of two prior therapies
Follow until B cells return toward baseline

Multiple Clinical Milestones Over Coming 15 Months

\$218M*

67.2M*

RUNWAY THROUGH 2027

SHARES OUTSTANDING

Anticipated Milestones

Timing

- Initiation of SLE global clinical trial with first patient in[†]
- Initiation of ITP global clinical trial with first patient in[†]
- Additional non-clinical data from subcutaneous program
- Advance pMN program to late phase development
- Initiate clinical development of subcutaneous program
- Evaluate additional programs

- H1 2025
- H1 2025
- H1 2025
- 2025
- 2025
- 2025

Building A Leading Immune-Mediated Disease Focused Company



Immune-Mediated Disease Focus

Focused solely on immune-mediated disease with budoprutug, a potentially best-in-class anti-CD19 antibody, our cornerstone asset



Broad Potential

Budoprutug in development for pMN, ITP, and SLE with the prospect of expanding into additional indications as well as a potential subcutaneous formulation



Well Resourced

Funded through 2027 enabling delivery of key value inflection points through the initiation of multiple clinical programs and a subcutaneous formulation clinical trial



Experienced Team

Track record of execution and operational results