Corporate Presentation

JANUARY 2025

CLIMBBIO

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”); expectations regarding the therapeutic benefits, clinical potential and clinical development of budoprutug and CLYM116; the trial design for planned clinical trials of budoprutug; plans to optimize the administration of budoprutug;the anticipated benefits of Climb Bio’s license agreement with Mabworks; anticipated timelines for preclinical data and initiating clinical trials of budoprutug and CLYM116; 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. and its license agreement with Beijing Mabworks Biotech Co., Ltd. (“Mabworks”); 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 and CLYM116 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; competing successfully with other companies that are seeking to develop treatments for systemic lupus erythematosus, immune thrombocytopenia, membranous nephropathy, IgA nephropathy and other immune-mediated diseases; maintaining or protecting intellectual property rights related to budoprutug, CLYM116 and/or its other product candidates; managing expenses; and raising the substantial additional capital needed, on the timeline necessary, to continue development of budoprutug, CLYM116 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

1 in 7 Americans suffer from an immune-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
Committed to developing better treatments for patients with immune-mediated disease

Broad Potential
Programs focused on clinically validated biology and have potential for development in multiple indications

Well Resourced
Funded through 2027 enabling delivery of key value inflection points across multiple programs

Experienced Team
Track record of identifying potential best-in-class assets, operational excellence and delivering results

CLIMBBIO

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

Emily Pimblett
SVP, Finance

Kate Hecht
SVP, Program Management

Jan Hillson
Senior Clinical Advisor

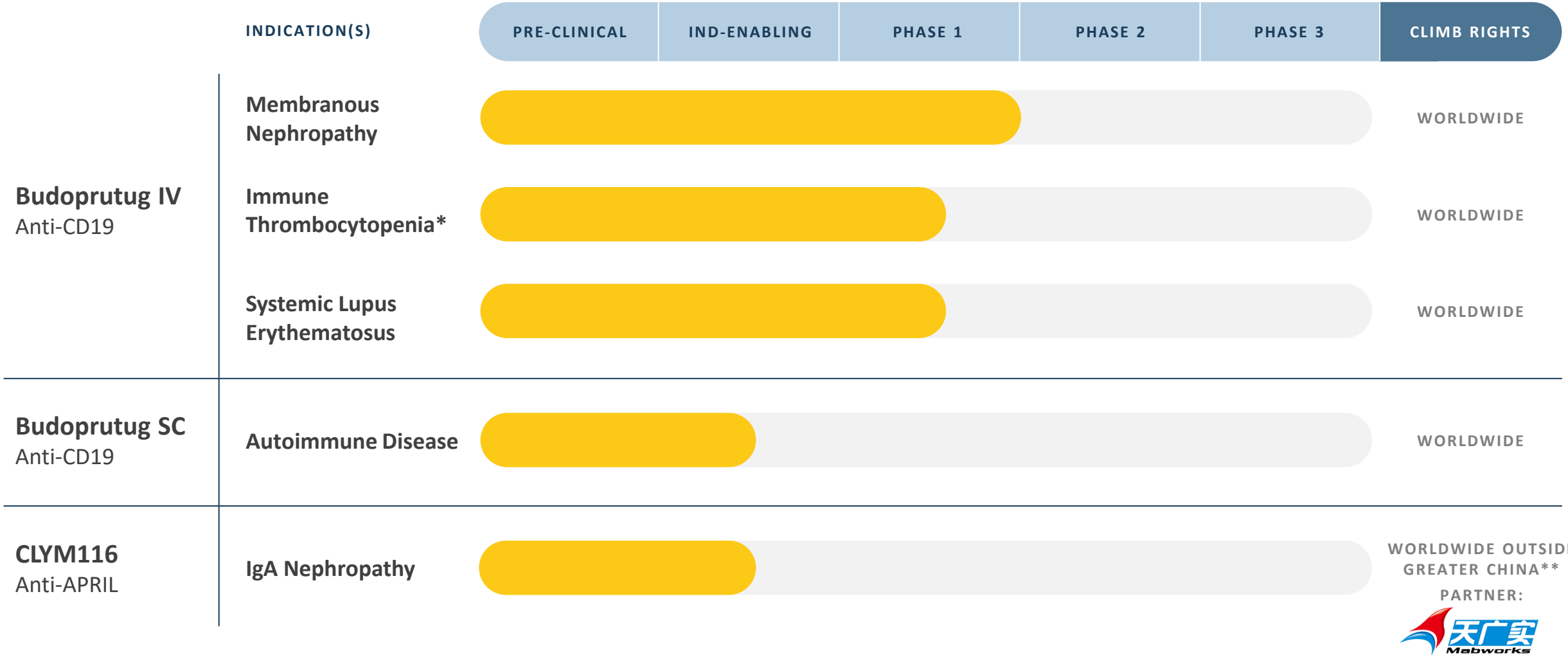
Gang (Gary) Hao
VP, CMC

Janaki M. Subramanyam
VP, Regulatory Affairs

Jay Mitchell
VP, Clinical Operations

Growing Pipeline Focused on Immune-Mediated Diseases

Developing best-in-class treatments for patients with immune-mediated diseases



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*Subject to investigational new drug application clearance **Greater China defined as mainland China, Hong Kong, Macau, and Taiwan
APRIL = a proliferation-inducing ligand, IV = intravenous, SC = subcutaneous

License Expands Pipeline with a Potential Best-in-class anti-APRIL mAb

Furthers goal to become a leader in developing new treatment options for immune-mediated diseases

Strategic Rationale

- Complementary to Climb's anti-CD19 antibody, budoprutug
- Leverages Climb's clinical and regulatory capabilities and Mabworks' development and manufacturing capabilities
- Inhibition of APRIL is a clinically validated mechanism and potentially disease modifying approach for IgAN
- CLYM116 is a highly potent anti-APRIL mAb with half-life extension and enhanced APRIL degradation

Meaningful Opportunity

- IgAN is the most common primary glomerular disease worldwide; est. >110,000 cases in the US
- Growing market, expected to reach > $10B annually over coming 10 years
- Opportunity to address limitations of APRIL-targeted therapies currently in development

Key Deal Terms

- Exclusive worldwide license outside of Greater China*
- Upfront of $9m, additional payments upon the achievement of specified development, regulatory and commercial milestones, and low- to mid-single digit royalites

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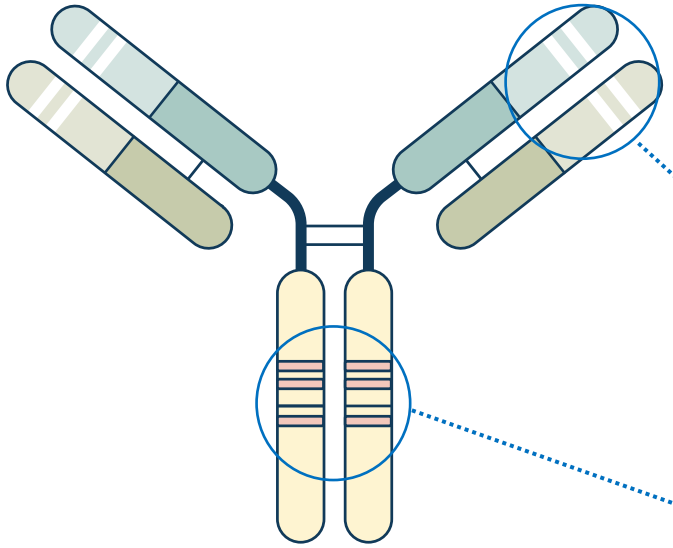
*Greater China defined as mainland China, Hong Kong, Macau, and Taiwan

APRIL = a proliferation-inducing ligand, mAb = monoclonal antibody, IgAN = IgA nephropathy

CLYM116: Fc-Engineered Anti-APRIL mAb

Designed to address limitations of APRIL-targeted therapeutics currently in development



CLYM116 is a highly potent anti-APRIL mAb designed to prevent APRIL signaling through a novel mechanism

Unique attributes driving differentiation & positioning

NOVEL MECHANISM

Blocking APRIL binding and enhancing degradation of APRIL

HIGH AFFINITY

pH-dependent binding to APRIL

FC-ENGINEERED

Clinically validated Fc mutations to increase serum half-life and diminish effector function

- **Potential for best-in-class efficacy**
  Rapid, deep, and durable inhibition of APRIL signaling
- **Favorable tolerability profile**
  Avoids potential immunosuppression associated with BAFF inhibition
  Engineered to silence effector function
- **Optimized dosing**
  Subcutaneous administration with potential for less frequent dosing, reducing patient burden

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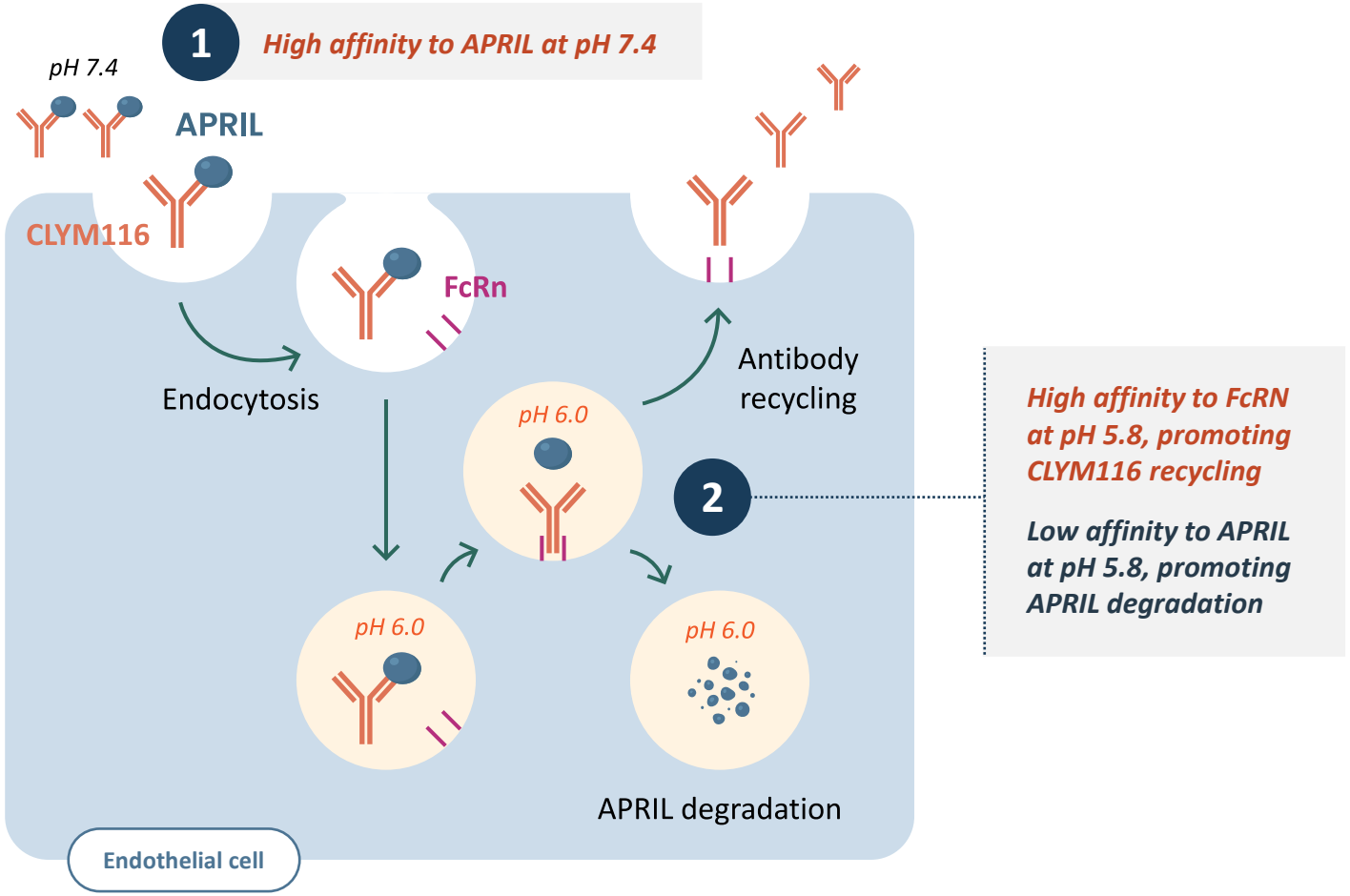
APRIL = a proliferation-inducing ligand, BAFF = B-cell activating factor, mAb = monoclonal antibody

CLYM116: Mechanism of Action

Novel mechanism of action employs a pH-dependent bind-and-release design, coupled with Fc-engineering



Variable Region exhibits **pH-dependent binding to APRIL**, resulting in both potent blocking of APRIL to its receptors *and* promotion of lysosomal APRIL degradation

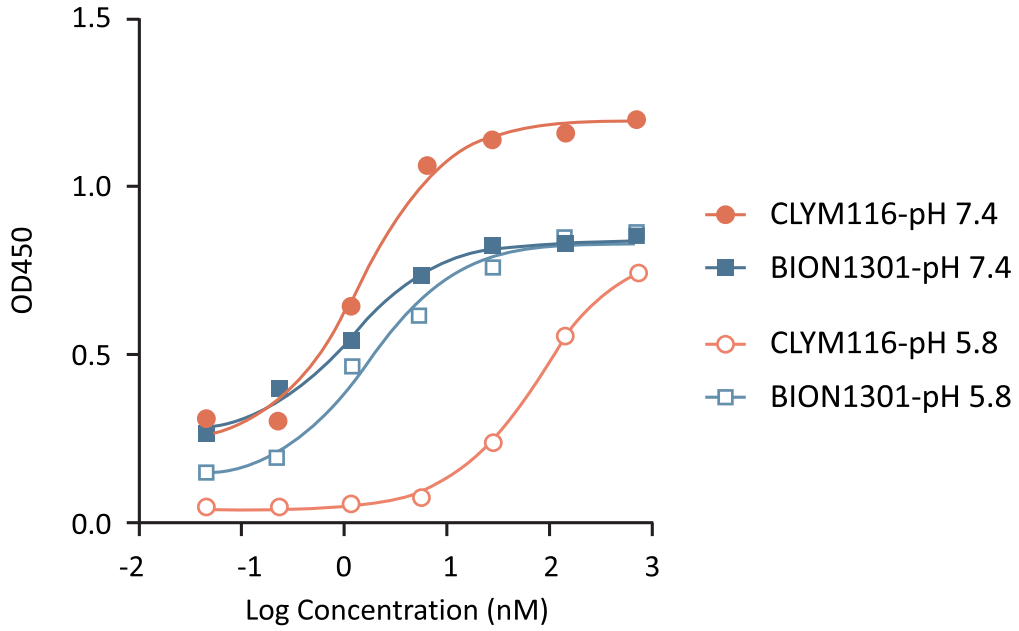
Fc Region **engineered to promote antibody recycling** and reduce clearance of CLYM116, resulting in longer half-life

APRIL = a proliferation-inducing ligand

CLYM116: Engineered For Potency and Rapid Clearance of APRIL

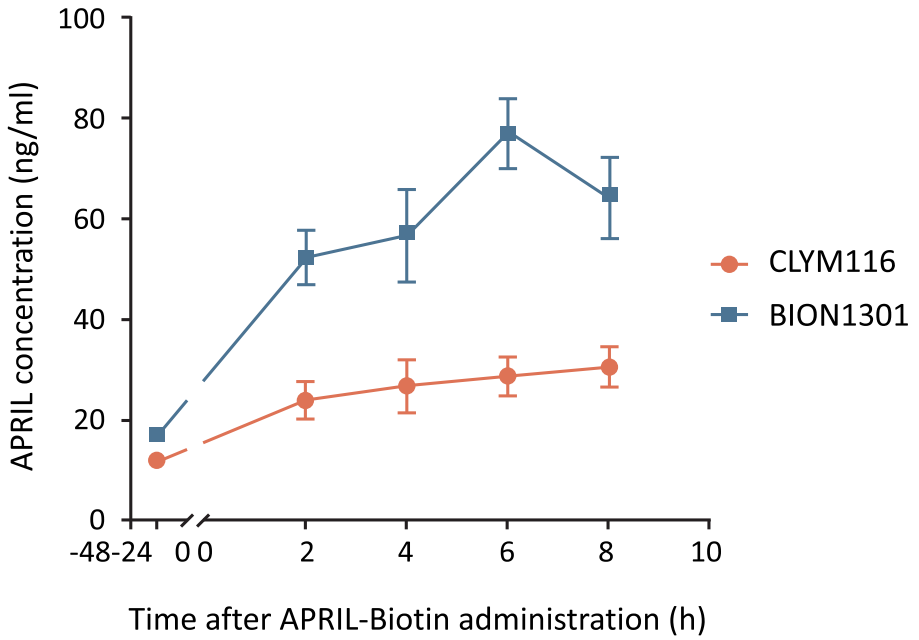
pH-dependent binding of CLYM116 to APRIL led to deep and durable clearance of APRIL in vivo

pH-dependent ELISA assay



- CLYM116 potently bound APRIL at pH 7.4; poor binding at pH 5.8
- Benchmark anti-APRIL mAb did not demonstrate this pH-dependent binding profile

APRIL degradation in mice after administration of anti-APRIL antibodies*

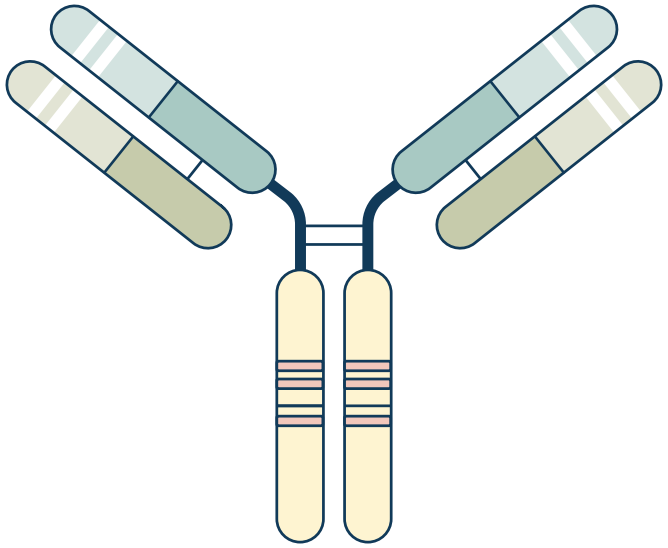


- CLYM116 depleted circulating APRIL and demonstrated enhanced clearance vs. benchmark anti-APRIL mAb

*Wild type C57 mice; antibodies 10mg/kg, single dose sc at time 0; Human APRIL 15mg/kg, single dose sc at 36 h; APRIL concentration assessed every 2 h thereafter

APRIL = a proliferation-inducing ligand, ELISA = enzyme-linked immunosorbent assay, h = hours, mAb = monoclonal antibody, OD = optical density, sc = subcutaneous

CLYM116: Potential Best-in-Class Anti-APRIL mAb for IgAN



CLYM116
Anti-APRIL
Fc+, mAb

INDICATION	PRE-CLINICAL	IND-ENABLING	PHASE 1	PHASE 2	PHASE 3	CLIMB RIGHTS:
IgA Nephropathy						WORLDWIDE OUTSIDE GREATER CHINA

PARTNER: 天广实 Mabworks

Program is currently in IND-enabling studies and data from ongoing preclinical studies expected to be shared later in 2025

APRIL is a clinically validated target for IgA Nephropathy

CLYM116 was designed to have reduced dosing frequency, faster response, and strong efficacy

Aiming to advance potentially best-in-class mAb through IND enabling studies

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Fc+ = Fc-engineered, IgAN = IgA nephropathy, mAb = monoclonal antibody

IgA Nephropathy (IgAN): Indication Overview

Pathophysiology	• IgA Nephropathy (IgAN), also known as Berger's Disease is an **autoantibody mediated** disease caused by **deposition of immune complexes**, comprising IgA and IgG, **in the glomeruli** • Median age of diagnosis in western patients is 40-45 years old
Symptoms & Diagnosis	• Symptoms: hematuria, **proteinuria**, high blood pressure, edema • Diagnosis: kidney biopsy
Epidemiology	• IgAN is the **most common primary glomerular disease worldwide** with an **estimated >110,000 cases in the US** and higher prevalence in Europe & Asia • **Growing market**, expected to reach **> $10B annually** over coming 5-10 years
Natural History	• **30-40% of untreated patients will develop kidney failure** within 10 years of diagnosis • Accelerated approval based on reduction in proteinuria with full approval based on stabilization of eGFR
Standard of Care	• Treatment is aimed at preserving kidney function through reduction of blood pressure and proteinuria • **Target maintain urine protein excretion < 0.5g/24 hours** and ideally normalization of protein excretion, i.e. < 0.3g/24 hours • Updated KDIGO guidelines likely to recommend additional treatment when proteinuria > 0.5g/24 hours

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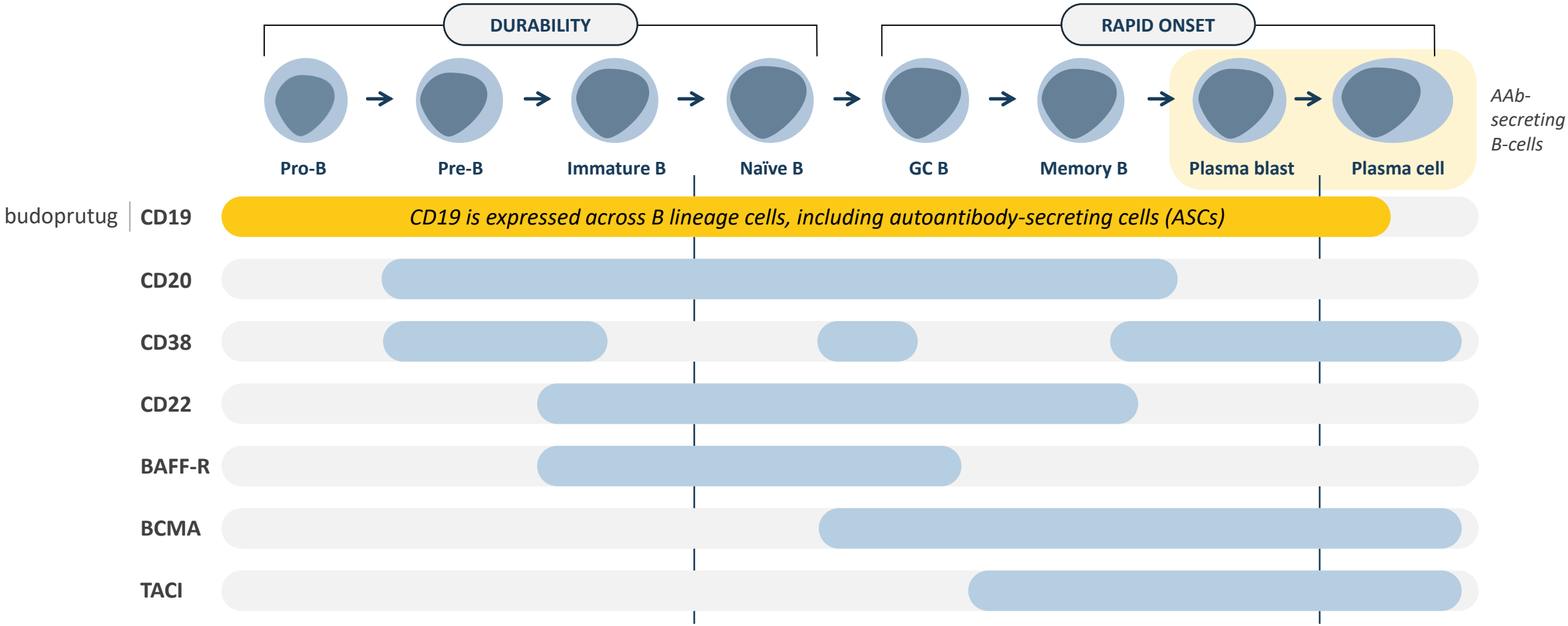
Stamellou 2023, Caster 2024, Lerma 2023, Barbour 2024, Barratt J 2024, Paul E. 2024, Pattrapornpisut P 2021

eGFR = estimated globmerular filtration rate, g = grams, KDIGO = Kidney Disease Improving Global Outcomes

CD19: Important Target for B-cell Mediated Diseases

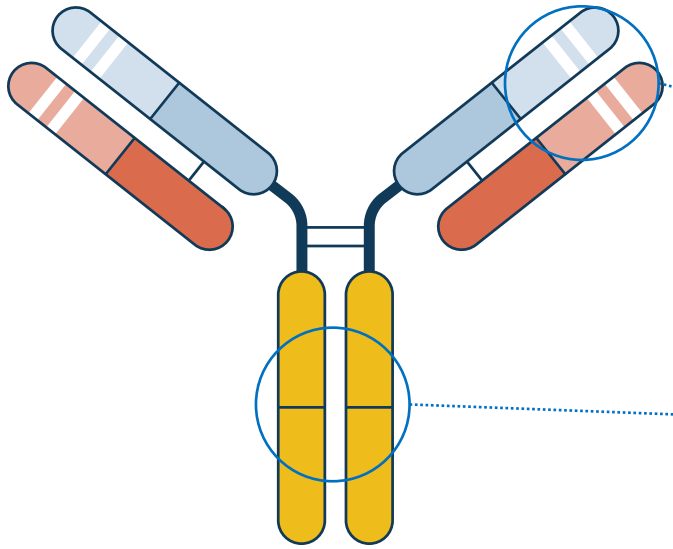
CD19-targeted therapy potentially enables rapid onset and durability of effect in B-cell mediated diseases



AAb = autoantibody, Zhang 2023

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

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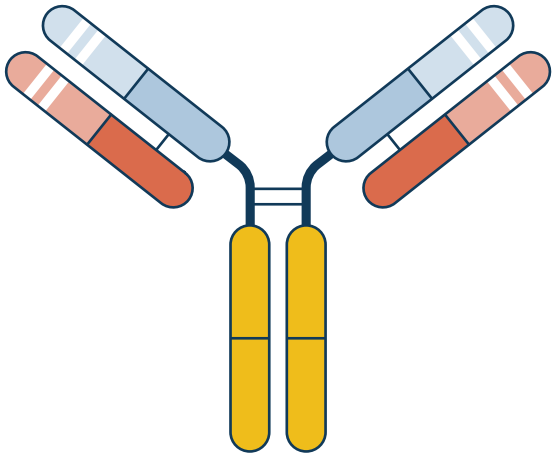
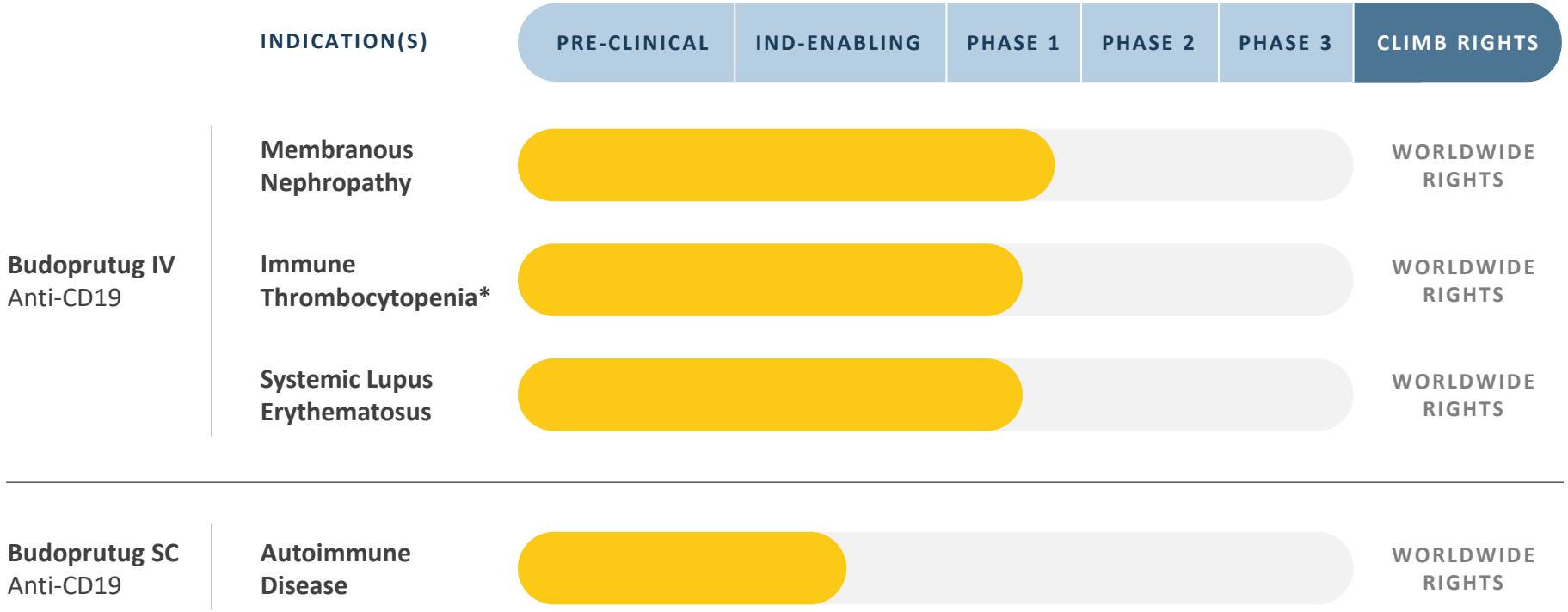
pM = picomolar, ADCC = antibody-dependent cellular cytotoxicity, IV = intravenous, SC = subcutaneous

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



Budoprutug
Anti-CD19
Fc+, mAb



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

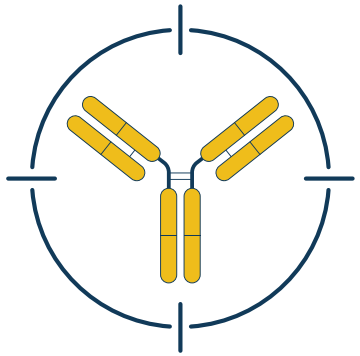
Additional potentially addressable indications across multiple therapeutic areas

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

AAb = autoantibody, Fc+ = Fc-engineered, IV = intravenous, mAb = monoclonal antibody, MoA = mechanism-of-action, SC = subcutaneous
*Subject to investigational new drug application clearance

Budoprutug Strategy & Vision

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

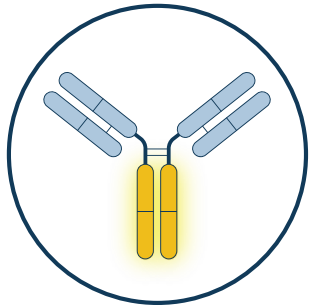


Primarily IgG4-Mediated

Clear pathophysiology supporting targeting of CD19-expressing B-cells

Lead indication
Primary Membranous Nephropathy

Opportunity to Differentiate
Potential for “immune reset”, improved efficacy

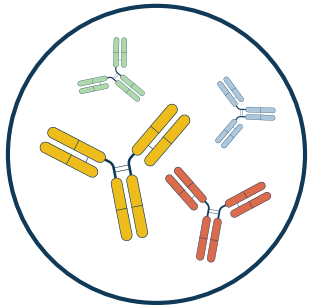


Primarily Single Organ IgG1 - 3

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

Budoprutug: 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, MG (AChR) = Myasthenia Gravis acetylcholine receptors, PV = Pemphigus Vulgaris

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)

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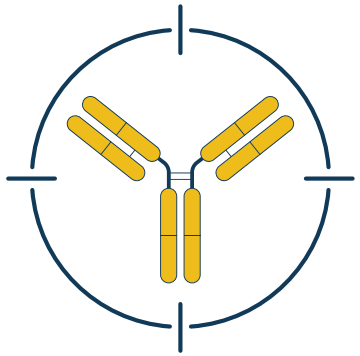
Budoprutug: Primarily IgG4-Mediated

Primary Membranous Nephropathy



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Systemic Lupus Erythematosus

Opportunity to Differentiate
Improve efficacy while balancing safety, tolerability, and convenience

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Primary Membranous Nephropathy (pMN): Indication Overview

Pathophysiology	• 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	• Symptoms: **proteinuria**, hypoalbuminemia, edema, dyspnea, fatigue, hyperlipidemia, nephrotic syndrome • Diagnosis: kidney biopsy and/or serum anti-PLA2R antibody assays
Epidemiology	• MN incidence: 1/100K and 80% are pMN; conservatively ~100K patients in US and EU • **20-40% of patients are refractory** to currently available lines of treatment
Natural History	• 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 years • Patients are at increased risk for **kidney failure and life-threatening thromboembolic events** • **Remission of proteinuria** is an approvable endpoint in nephrotic patients
Standard of Care	• Treatment is aimed at reducing proteinuria, though **all therapies are off-label** • KDIGO guidelines recommend treatment based on risk, primarily based on eGFR and PLA2R levels • Rituximab (RTX) is considered 1st line therapy**;** MENTOR trial showed complete remission w/ RTX superior to calcineurin inhibitors (CNIs) at 24 months

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eGFR = estimated glomerular filtration rate; KDIGO = kidney diseases improving global outcomes; PLA2R = phospholipase A2 receptor

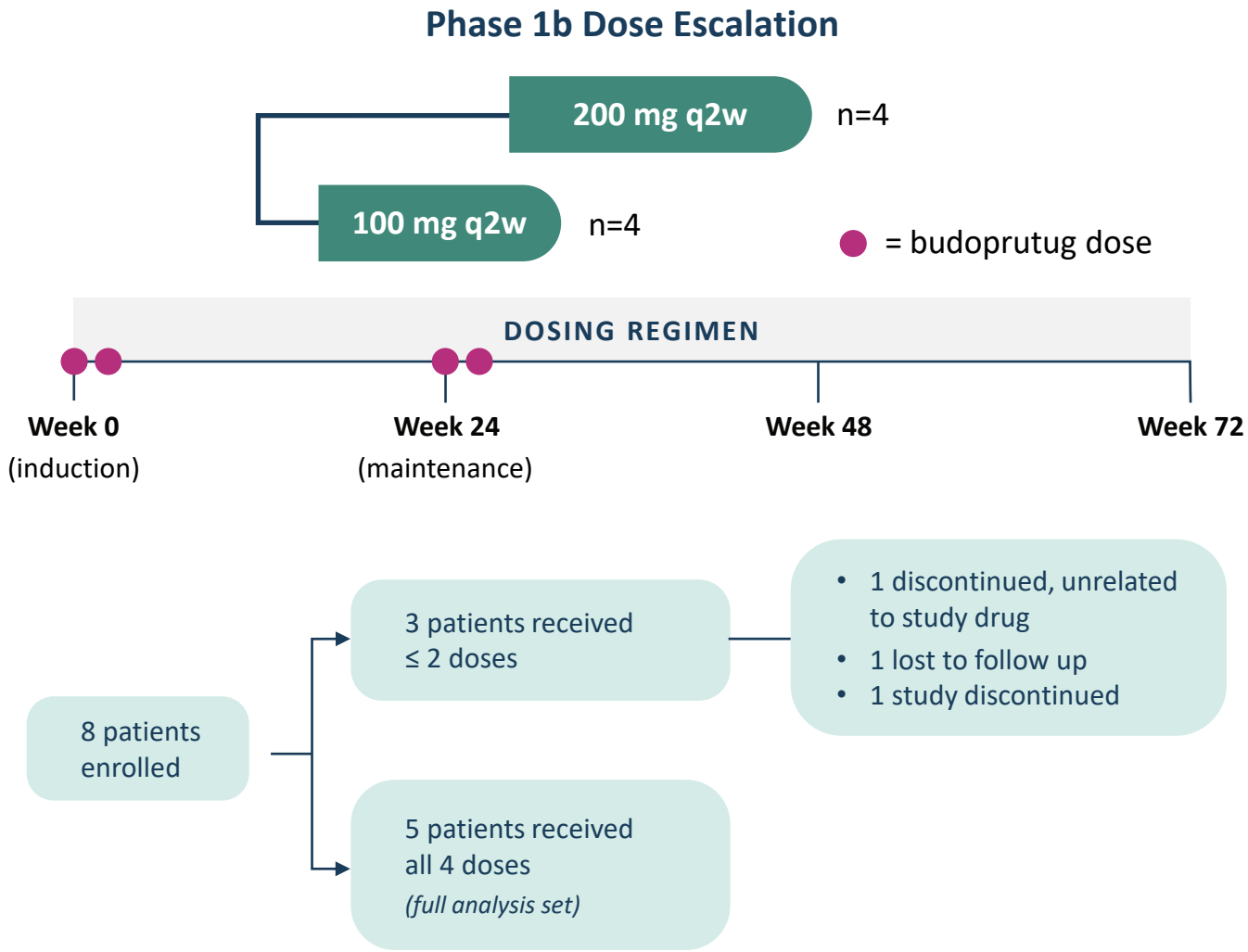
Ronco P 2021; Teisseyre M 2022; KDIGO 2021 guidelines; Ahmad, S.B. 2020; Swaminathan S 2006; Donadio JV 1988; Schieppati A 1993; Shiiki H 2004

Budoprutug Phase 1b Study Design

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

ELIGIBILITY	• UPCR ≥2.0 g/g • B-cell count >LLN (80 cells/µL)
DESIGN	• Dose escalation & expansion • 18-month follow-up
DOSING	2 doses 14 days apart • 100 mg • 200 mg
ENDPOINTS	• Safety, tolerability & PK • PD markers (B-cells, PLA2R) • Proteinuria response

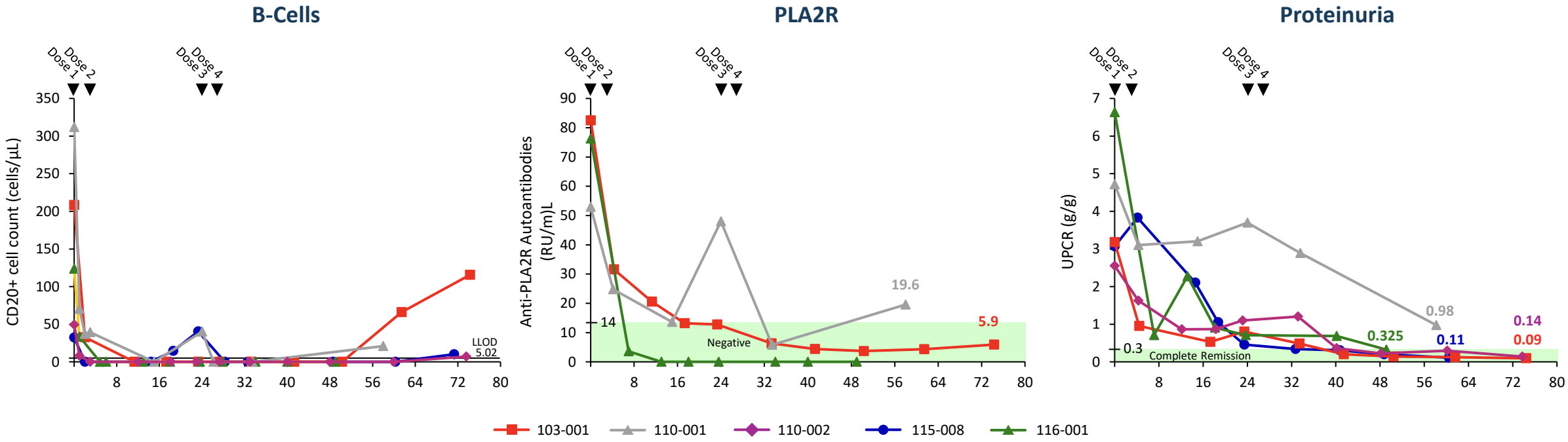
PARAMETER	BASELINE (MEAN)
B-cells	145 cells/µL
PLA2R	71 RU/mL
UPCR	4.03 g/g



LLN = lower limit of normal, PD = pharmacodynamic, PK = pharmacokinetic, PLA2R = phospholipase A2 receptor, pMN=primary membranous nephropathy, UPCR = urine protein creatinine ratio

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)

Note: Interim data presented at ASN 2022

MN = Membranous Nephropathy, PLA2R = phospholipase A2 receptor, UPCR = urine protein creatinine ratio

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

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AE = adverse event, SAE = serious adverse event

Budoprutug: Primarily Single Organ IgG1 - 3

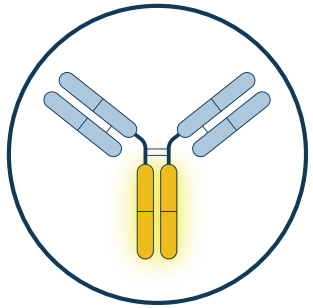
Immune Thrombocytopenia

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



Primarily Single Organ IgG1 - 3

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Multi-organ, systemic diseases with heterogenous patient populations

Lead indication
Systemic Lupus Erythematosus

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Improve efficacy while balancing safety, tolerability, and convenience

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Immune Thrombocytopenia (ITP): Indication Overview

Pathophysiology	• 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	• 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	• 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	• 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

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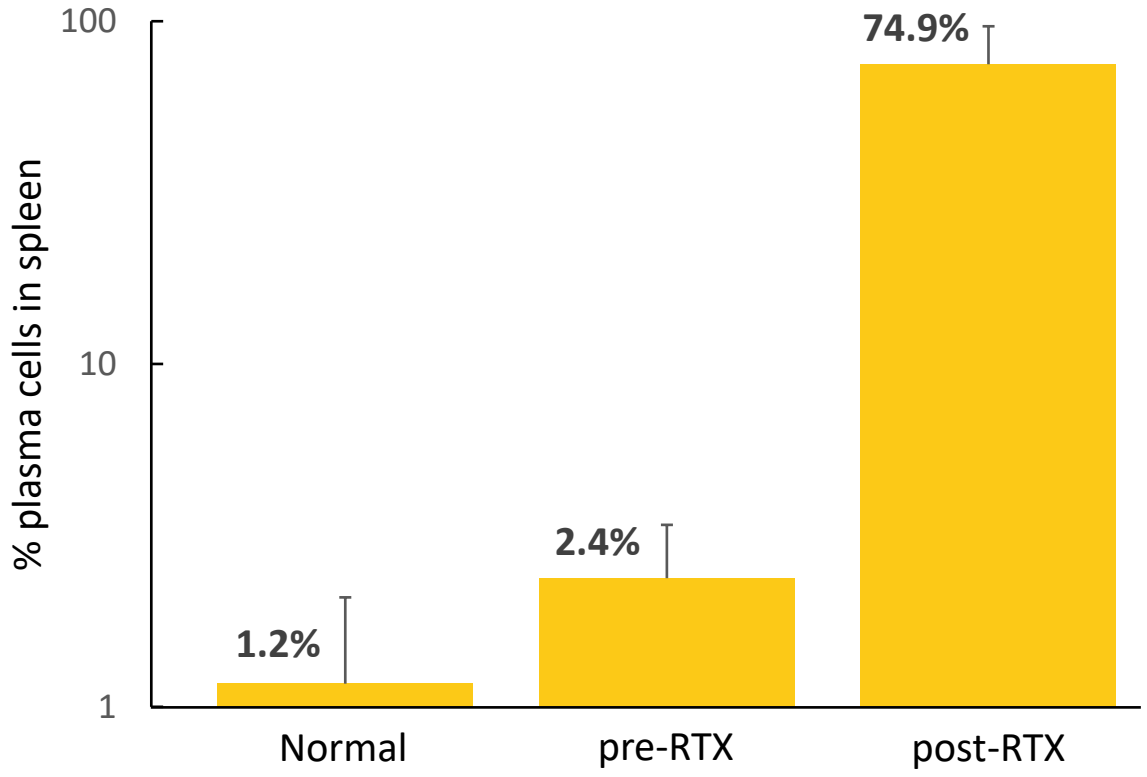
Statpearl ; Weycker D 2020; Moulis G 2021; Moulis G 2014; Jefferies Analyst Report For Rigel Pharmaceuticals from April 2,2024; DelveInsights Press Release for ITP report on Jan 11 2024; Gugenheim Securities Alpine report April 9, 2024

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

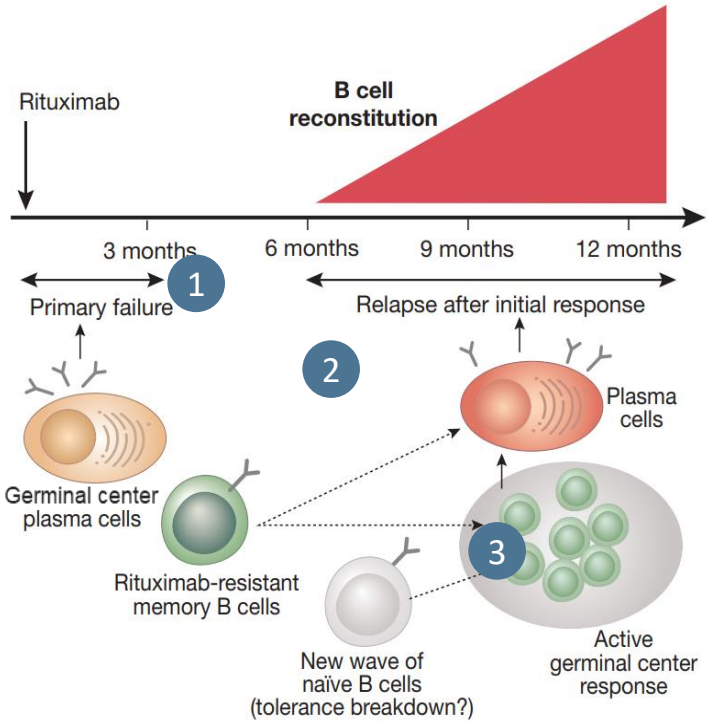


Primary failure

1. Pre-existing $CD20^-$ PCs

Relapse after initial response

2. Pre-existing $CD20^-$ B-cells
3. *De novo* $CD20^-$ B-cells



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Mahevas et al. 2013

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/µL
- B-cells > 40 /µL

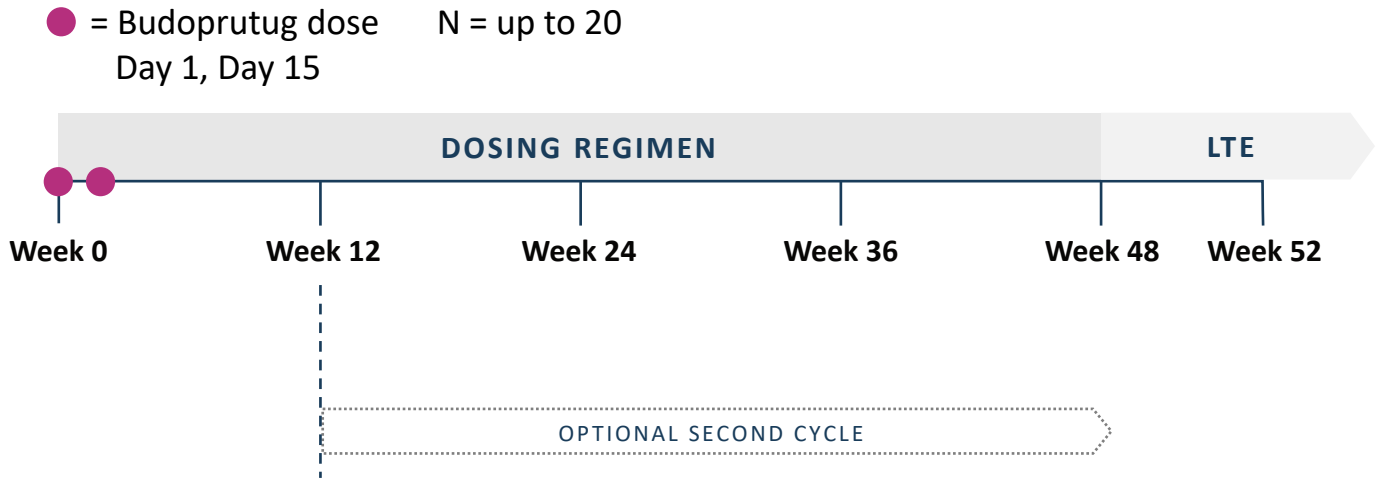
PRIMARY OBJECTIVE

- 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



*Subject to investigational new drug application clearance

Budoprutug: Complex Systemic

Systemic Lupus Erythematosus

Primarily IgG4-Mediated

Clear pathophysiology supporting targeting of CD19-expressing B-cells

Lead indication
Primary Membranous Nephropathy

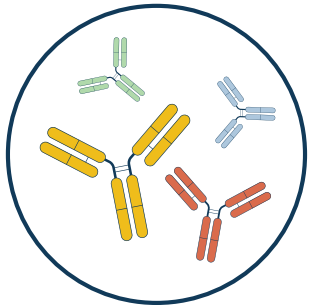
Opportunity to Differentiate
Potential for “immune reset”, improved efficacy

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

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Systemic Lupus Erythematosus (SLE): Indication Overview

Pathophysiology	• **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	• **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	• **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	• **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	• **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) Week 52 SRI-4 27.2% (vs. 22.7%)
Obinutuzumab	Roche	CD20	IV	Phase 3 LN (completed; results pending)	Phase 2 (vs placebo, n=125) Week 52 CRR 35% (vs 23%) Week 104 CRR 41% (vs 23%)
Belimumab	GSK	BAFF	SC	SLE and LN (marketed)	Phase 3 (vs. placebo, n=836) Week 52 SRI-4 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

Arnold 2022; Furie 2022; Scherlinger 2019; Benlysta FDA label

BAFF = B-cell activating factor, CCR = complete renal response, IV = intravenous, LN = lupus nephritis, SC = subcutaneous, SLE = systemic lupus erythematosus; SRI = systemic lupus erythematosus responder index

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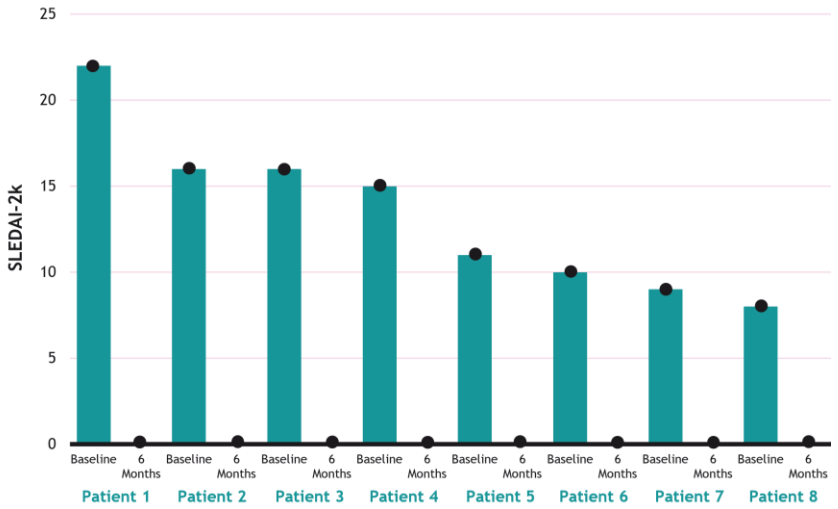
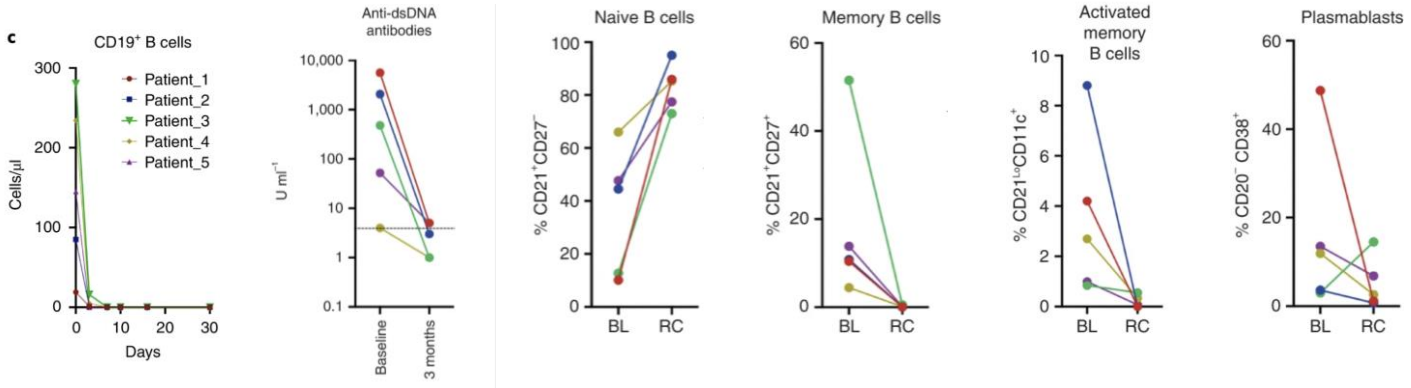
Targeting B-Cells Has Shown Promise in SLE

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

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



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

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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

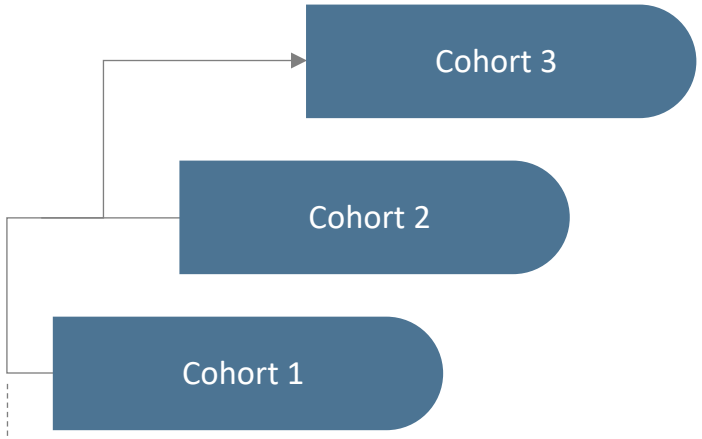
PRIMARY OBJECTIVE

- 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



Subjects with active SLE on stable limited background are eligible for dose escalation phase

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

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SLEDAI = Systemic Lupus Erythematosus Disease Activity Index; ANA = antinuclear antibodies; anti-dsDNA = anti double-stranded DNA; ENA = extractable nuclear antigen

Multiple Milestones Anticipated Over Coming 12 Months

Budoprutug	
Initiation of SLE global clinical trial with first patient in	H1 2025
Initiation of ITP global clinical trial with first patient in †	H1 2025
Additional non-clinical data from subcutaneous program	H1 2025
Advance pMN program to late phase development	2025
Initiate clinical development of subcutaneous program	2025

CLYM116	
Initial preclinical data	H2 2025

~$212.9M*

RUNWAY THROUGH 2027

67.3M*

SHARES OUTSTANDING

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*As of Dec 31, 2024 †Subject to investigational new drug application clearance

ITP = immune thrombocytopenia, m = million, pMN = primary membranous nephropathy, SLE = systemic lupus erythematosus

Building A Leading Immune-Mediated Disease Focused Company

Immune-Mediated Disease Focus
Committed to developing better treatments for patients with immune-mediated disease

Broad Potential
Programs focused on clinically validated biology and have potential for development in multiple indications

Well Resourced
Funded through 2027 enabling delivery of key value inflection points across multiple programs

Experienced Team
Track record of identifying potential best-in-class assets, operational excellence and delivering results

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