Corporate Presentation

MARCH 2026

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; expectations regarding the therapeutic benefits, clinical potential and clinical development of budoprutug and CLYM116; the anticipated timelines for reporting initial data from Climb Bio’s ongoing and planned clinical trials of budoprutug and CLYM116; the anticipated timeline for initiating Climb Bio’s parallel Phase 1b clinical trial of budoprutug in patients with systemic lupus erythematosus in China and the projected enrollment of patients with systemic lupus erythematosus that have lupus nephritis; the anticipated benefits of Climb Bio’s technology transfer and exclusive license agreement with Beijing Mabworks Biotech Co., Ltd. (“Mabworks”); 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 technology transfer and exclusive license agreement with 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 or nonclinical studies; competing successfully with other companies that are seeking to develop treatments for primary membranous nephropathy, immune thrombocytopenia, systemic lupus erythematosus, IgA nephropathy and other immune-mediated diseases; maintaining or protecting intellectual property rights related to budoprutug, CLYM116 and/or its other product candidates; the outcome of any legal proceedings or other disputes; 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.

OUR MISSION

Deliver high impact, disease-modifying medicines for individuals living with immune-mediated diseases

CLIMBBIO

Scaling New Heights in the Development of Transformative Immune Medicines

Delivering Clinical Results and Advancing Development

Corporate Highlights

Developing **differentiated,** monoclonal antibody (mAb) therapeutics for **immune-mediated diseases**, including those affecting **kidney health**, with expansive commercial opportunities

Leveraging **clinically validated** B cell targets, **proven mAb modality**, and indications with **well-defined** endpoints and **established** regulatory pathways

Anticipating a **data-rich 2026** with **multiple clinical readouts** across both clinical-stage programs

- **Budoprutug -** anti-CD19 mAb in development for pMN, ITP, and SLE
- **CLYM116 -** anti-APRIL mAb in development for IgAN

Well-resourced to advance clinical programs through meaningful value-driving milestones, with **runway anticipated into 2028**

CLIMBBIO

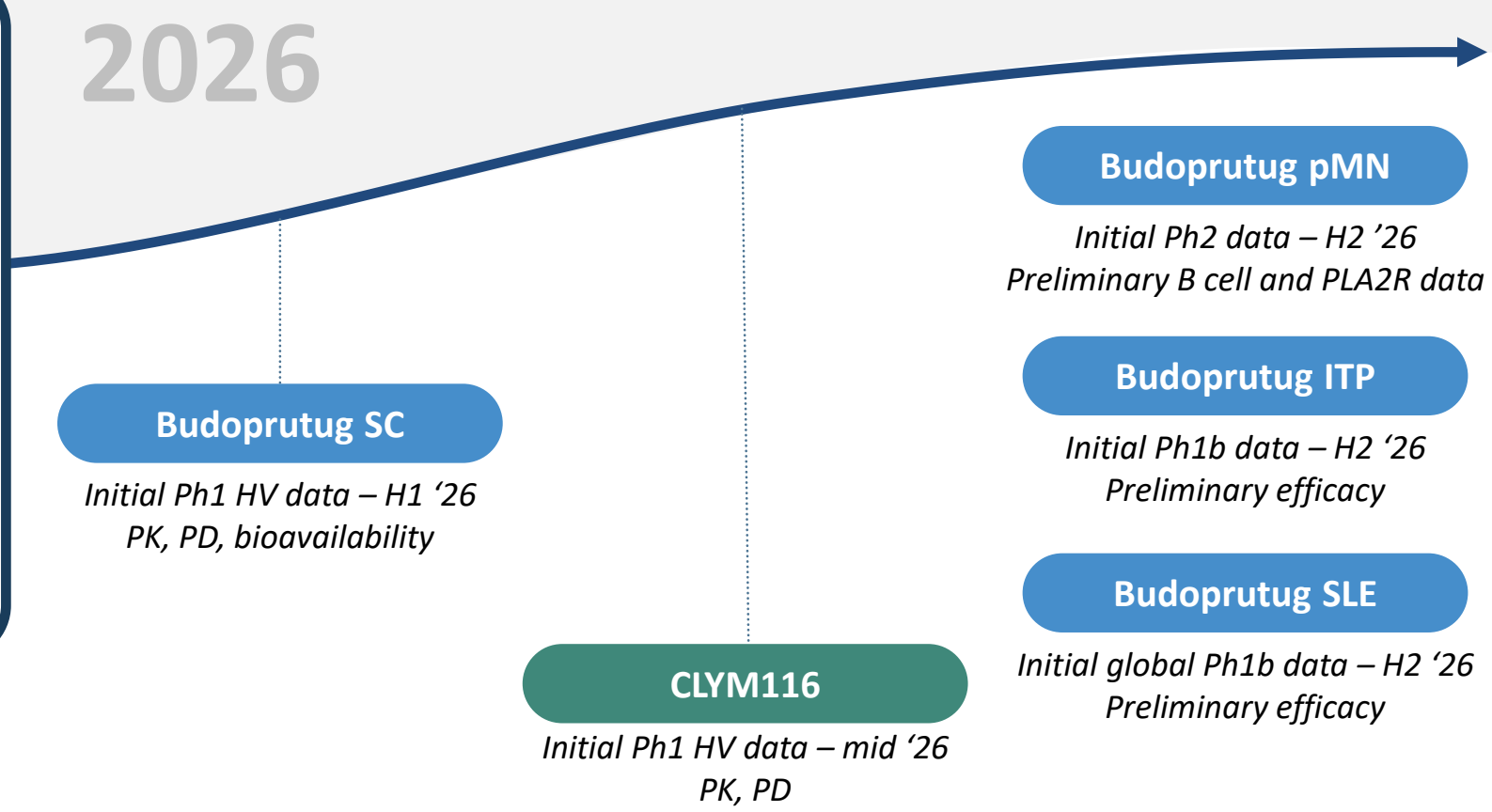
pMN = primary membranous nephropathy; ITP = Immune Thrombocytopenia; SLE = Systemic Lupus Erythematosus; IgAN = IgA Nephropathy

Climb Bio is Poised for a Data-Rich 2026

Five clinical studies underway, with initial readouts anticipated from all ongoing trials

Key 2025 Achievements

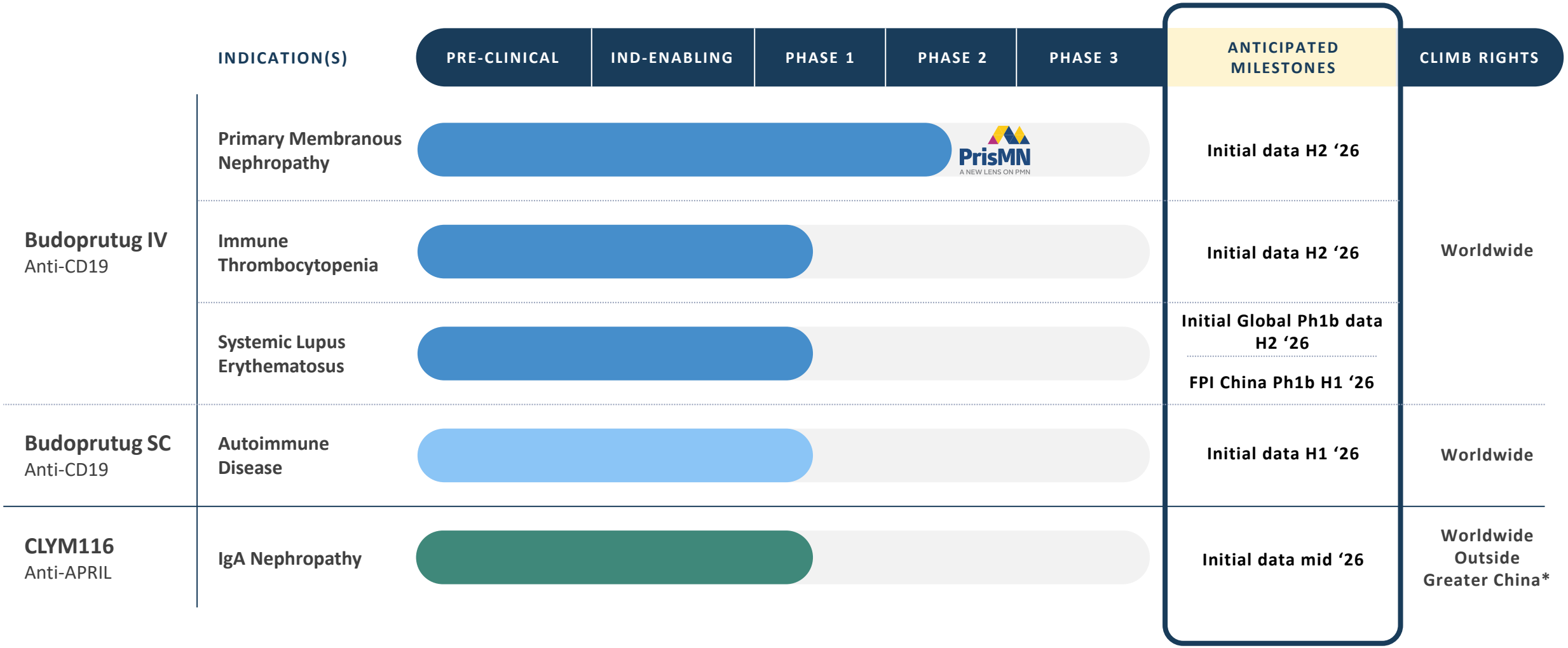
- ✓ CLYM116 in-licensing and Phase 1 initiation
- ✓ Budoprutug ITP, SLE, and pMN PrisMN trial initiations
- ✓ Budoprutug SC Phase 1 initiation
- ✓ Clearance of budoprutug SLE IND in China



ITP = Immune Thrombocytopenia; SLE = Systemic Lupus Erythematosus; pMN = primary membranous nephropathy; SC = subcutaneous; IND = investigational new drug; HV = healthy volunteers; PK = pharmacokinetics, PD = pharmacodynamics

Pipeline of Highly Differentiated mAbs

Anticipating initial readouts from all ongoing trials in 2026



Budoprutug SC and CLYM116 Phase 1 trials conducted in healthy volunteers.

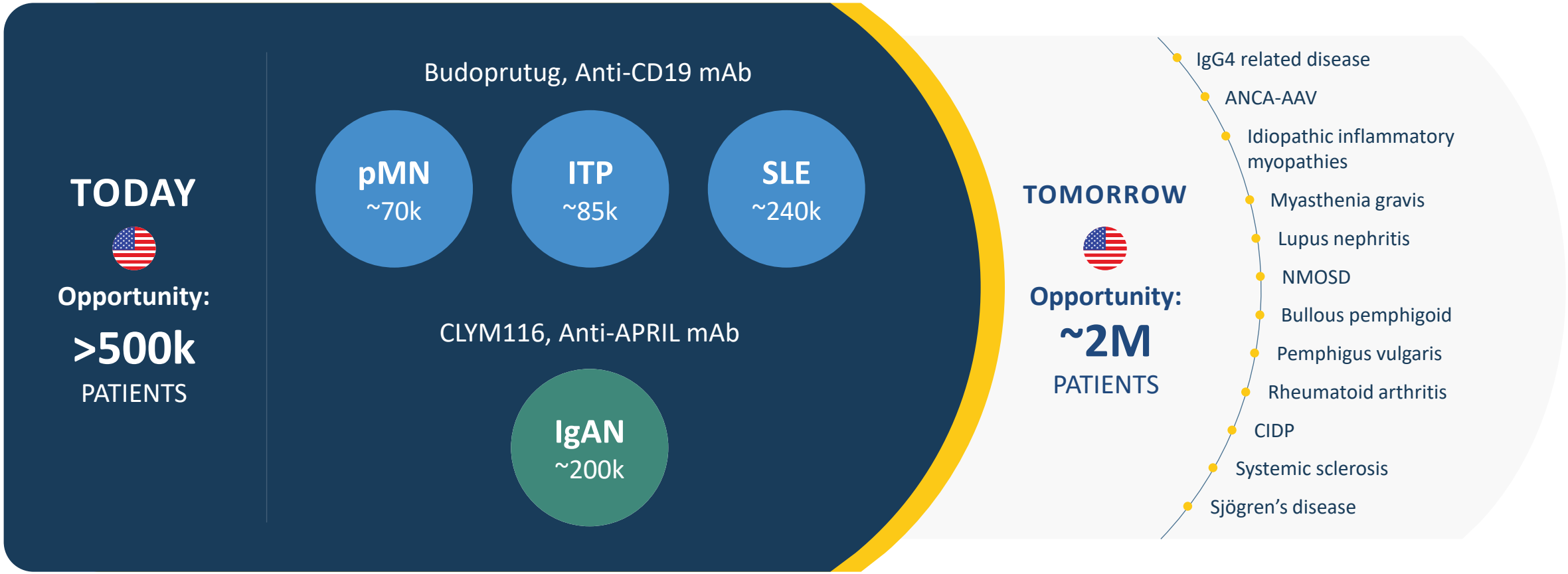
*Greater China defined as mainland China, Hong Kong, Macau, and Taiwan; Partner: Beijing Mabworks Biotech Co., Ltd.

APRIL = a proliferation-inducing ligand, IV = intravenous, mAbs = monoclonal antibodies, SC = subcutaneous

CLIMBBIO

Pursuing Expansive Market Opportunities

Addressing the needs of patients living with B cell-mediated diseases



ANCA-AAV = antineutrophil cytoplasmic antibody-associated vasculitis, CIDP = Chronic inflammatory demyelinating polyradiculoneuropathy, IgAN = IgA Nephropathy, ITP = Immune Thrombocytopenia, NMOSD = Neuromyelitis optica spectrum disorder, pMN = Primary Membranous Nephropathy, SLE = Systemic Lupus Erythematosus, mAb = monoclonal Ab. Prevalence sources: B cell-mediated disease (internal research), IgAN (Stoneman JAMA 2026), ITP (internal research), pMN (internal research), SLE (Izmirly Arthritis Rheumatol 2021).

Climb Bio Team Poised to Deliver for Patients

Aoife Brennan, MB, ChB
President and CEO

Perrin Wilson, PhD
CBO

Susan Altschuller, PhD, MBA
CFO

Edgar Charles, MD
CMO

Cindy J. Driscoll, MBA
SVP, Finance

Ashley Jones
SVP, People & Workforce Strategy

Adam Villa, MBA, MS
SVP, Tech Ops

Chandra Adams, JD
VP, General Counsel

BOARD OF DIRECTORS

Douglas E. Williams, PhD
Independent, Chairman

Aoife Brennan, MB, ChB

Bo Cumbo
CEO, Solid Biosciences

Kim Drapkin, CPA
Independent

Judith Dunn, PhD
President, Head of R&D, Vima Therapeutics

Andrew Levin, MD, PhD
Managing Director, RA Capital

Stephen Thomas, PhD
CEO, Sera Medicines

Budoprutug

Anti-CD19

Budoprutug: A Differentiated Anti-CD19 Approach

Durable B cell depletion, long dosing interval, with potential for IV and SC formulations

Potential Differentiating Benefits

1

Rapid, deep and **durable B cell depletion** and favorable tolerability demonstrated in a **pilot clinical study**

2

mAb modality confers **distinct advantages**

- **Well-established manufacturing** and supply chains, favorable cost-of-goods and scalability
- **Minimal off-target** effects, with a **low risk of CRS and ICANS**; no lymphodepleting chemotherapy preconditioning requirement
- Ability to **dose in the community setting**; **no in-hospital** or special unit administration required

3

Long dosing interval, with potential to **formulate for both IV and SC** administration

Clear Opportunity

- **Only one approved** CD19 mAb – positioned for rare diseases
- **Substantial potential** for budoprutug in **broader disease** areas where a **larger** patient subset can benefit
- **Strong IP** protection extending to 2045+

CRS = cytokine release syndrome, ICANS = immune effector cell associated neurotoxicity syndrome, IV = intravenous, mAb = monoclonal antibody, pM = picomolar, SC = subcutaneous

Cortazar ASN 2024

CD19: Attractive and Validated Target for B Cell-Mediated Disease

CD19 expression profile provides potential efficacy and safety advantages relative to other targets

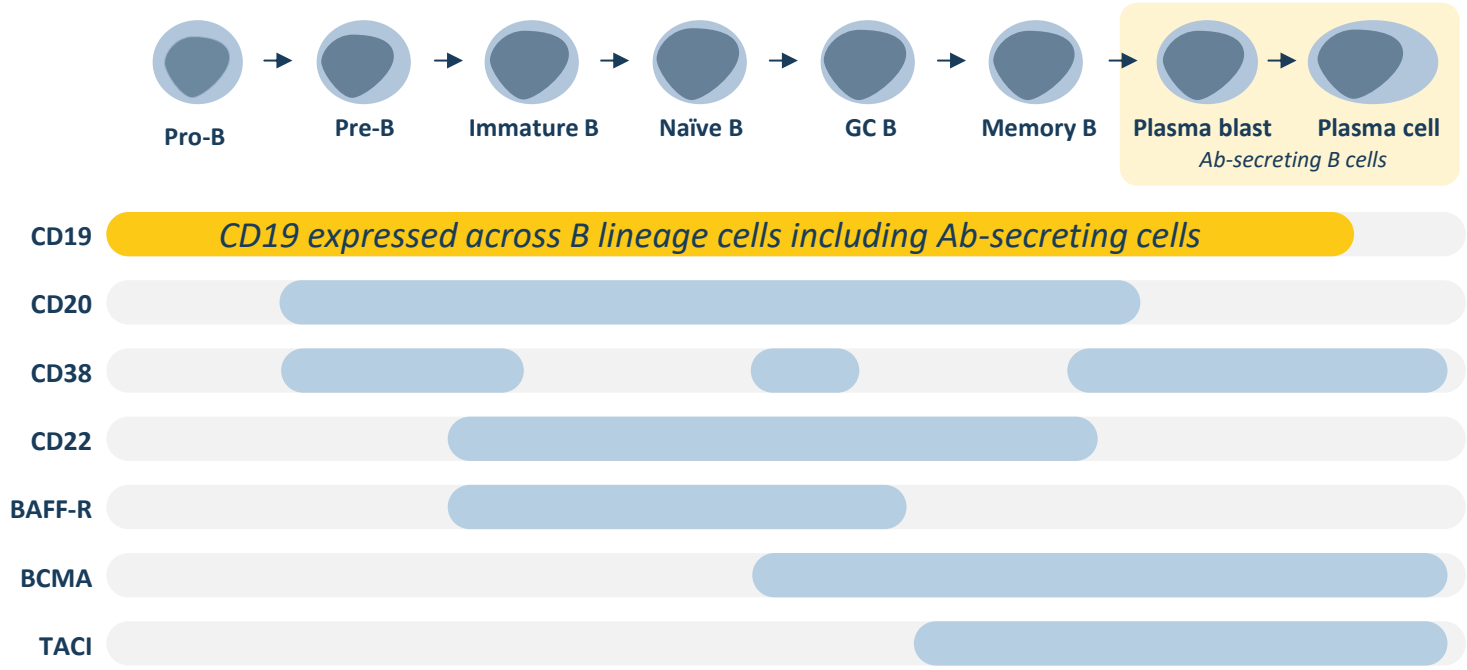
Clinically Proven Approach

CD19 CAR T Therapies

- Have demonstrated **potential for remission** in SLE patients

CD19 mAb Therapy (inebilizumab)

- Approved for NMOSD, IgG4-RD, and gMG
- **Demonstrated benefit** in rituximab relapse and where rituximab has failed



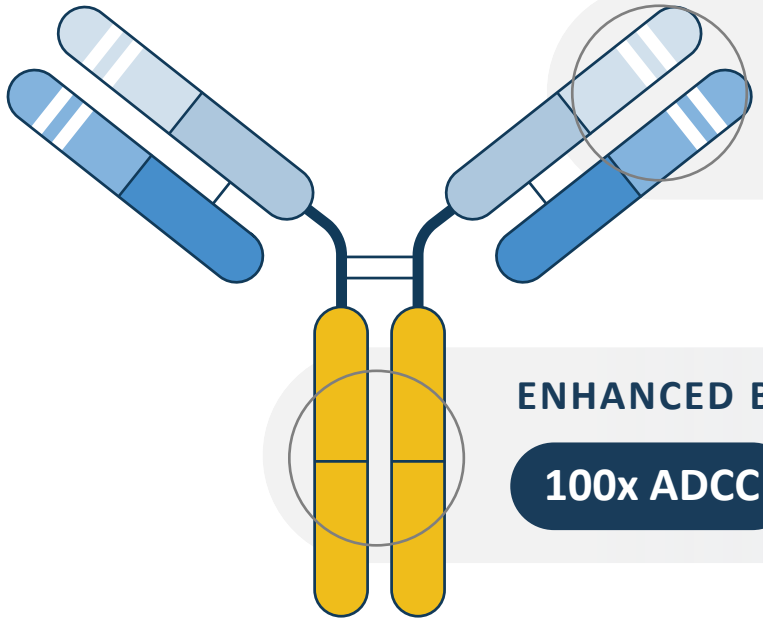
*Broad CD19 expression profile enables **comprehensive, rapid and durable depletion** of pathogenic B cells, while **preserving protective antibody responses** mediated by long-lived plasma cells*

Ab = antibody, BLA = biologics license application, CAR = chimeric antigen receptor, IgG4-RD = immunoglobulin G4-related disease, mAb = monoclonal antibody, gMG = generalized myasthenia gravis, NMOSD = neuromyelitis optica spectrum disorder, SLE = systemic lupus erythematosus.
Zhang Front Immunol 2023, Müller NEJM 2024, Novak NEJM 2025, Osborne Front Neurol 2024, inebilizumab prescribing information

Budoprutug is a Highly Potent, Fc-Enhanced Anti-CD19 mAb

Designed for optimal biological activity, with potential for both IV and subcutaneous administration

KEY FEATURES



STRONG TARGET BINDING

18 pM binding affinity to CD19 counters low antigen density

ENHANCED B CELL DEPLETION

100x ADCC precisely-tuned, low-fucosylated Fc region increases potency vs wild-type Fc

SUBCUTANEOUS DOSING POTENTIAL

≥175 mg/mL

High concentration formulation with low viscosity

ADCC = antibody-dependent cellular cytotoxicity, mAb = monoclonal antibody, pM = picomolar

Developing Budoprutug Across Multiple Diseases

Pursuing development in lead indications with high unmet need and clear B-cell driven pathology

Primary Membranous Nephropathy (pMN)	Immune Thrombocytopenia (ITP)	Systemic Lupus Erythematosus (SLE)
Progressive IgG4-mediated renal disease with clear pathophysiology support targeting of CD19-expressing B cells, with demonstrated clinical proof of concept	Chronic disorder, affecting a single organ (blood cells), for which B-cell depletion has demonstrated clinical proof of concept	Chronic, multi-organ autoimmune condition with early clinical validation for CD19 via other modalities; subset of patients have kidney involvement (lupus nephritis)
Phase 1b completed **Phase 2 ongoing**	**Phase 1b/2a ongoing**	**Global Phase 1b ongoing** **China Phase 1b in start-up**
Initial data expected H2 2026	*Initial data expected H2 2026*	*Initial data from global study expected H2 2026*

CLIMBBIO

Primary Membranous Nephropathy (pMN)

Progressive renal disease

- **Autoantibody mediated** disease characterized by **proteinuria**, **nephrotic syndrome** and progressive **loss of renal function**
- Untreated, ~30% progress to **end-stage renal disease** within 10 years and another 30% develop chronic kidney disease

No approved therapies

- **Rituximab used off-label** and considered **first-line**; 20-40% patients are refractory
- **Clear unmet need** for disease-modifying therapies that deliver **complete remission** of proteinuria

Clear path to approval

- Early demonstration of efficacy and approval possible based on **a validated biomarker** indicative of kidney damage: **proteinuria**

SIGNIFICANT OPPORTUNITY

~70,000 patients
in the US alone

THE CLIMB SOLUTION

Budoprutug demonstrated compelling proof-of-concept in a pilot Phase 1b clinical study in pMN

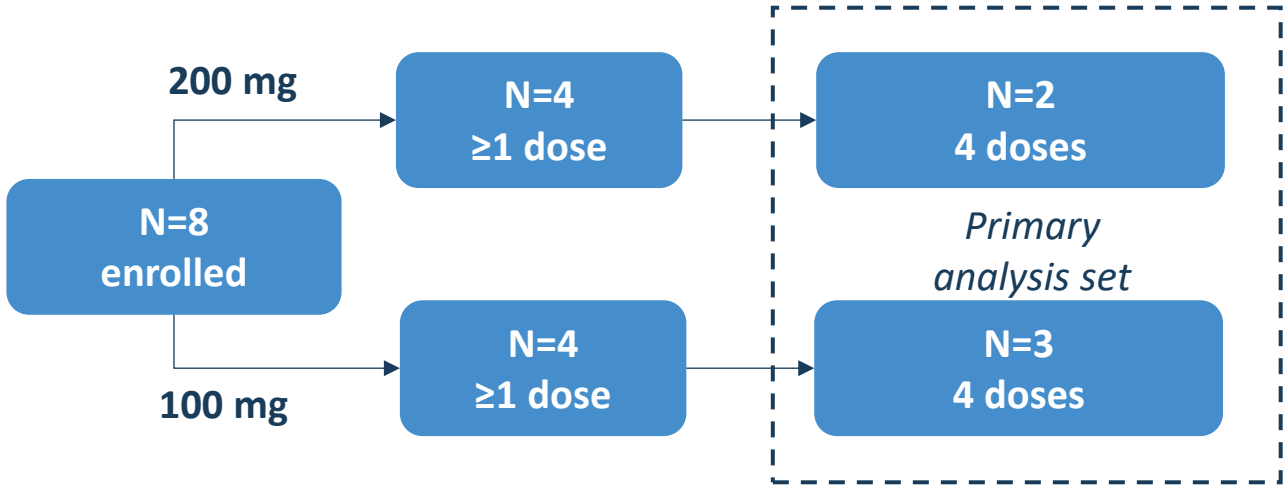
pMN = primary membranous nephropathy; US Prevalence: internal research; Couser CJASN 2017, Ronco J Clin Med 2021, Teisseyre Front Immunol 2022, KDIGO 2021 guidelines

Completed Phase 1b Study Established Proof of Concept in pMN

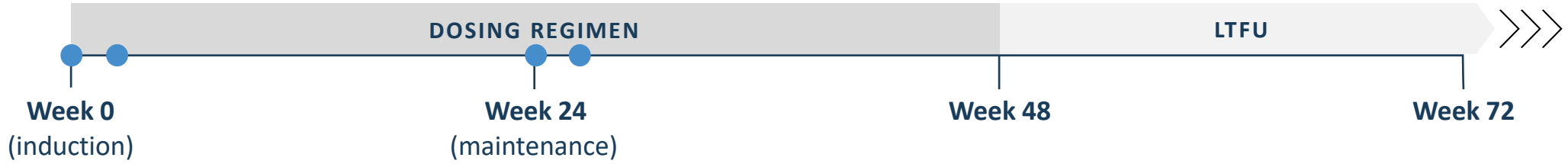
Budoprutug evaluated in an open-label, dose escalation study in adult patients with pMN (NCT04652570)

Patients had **moderate to severe disease** at baseline

- B cells: 145 ± 116 cells/µL
- PLA2R: 71 ± 16 RU/mL
- UPCR: 4.03 ± 1.66 g/g



● = Budoprutug IV infusion (2 doses, 14 days apart)

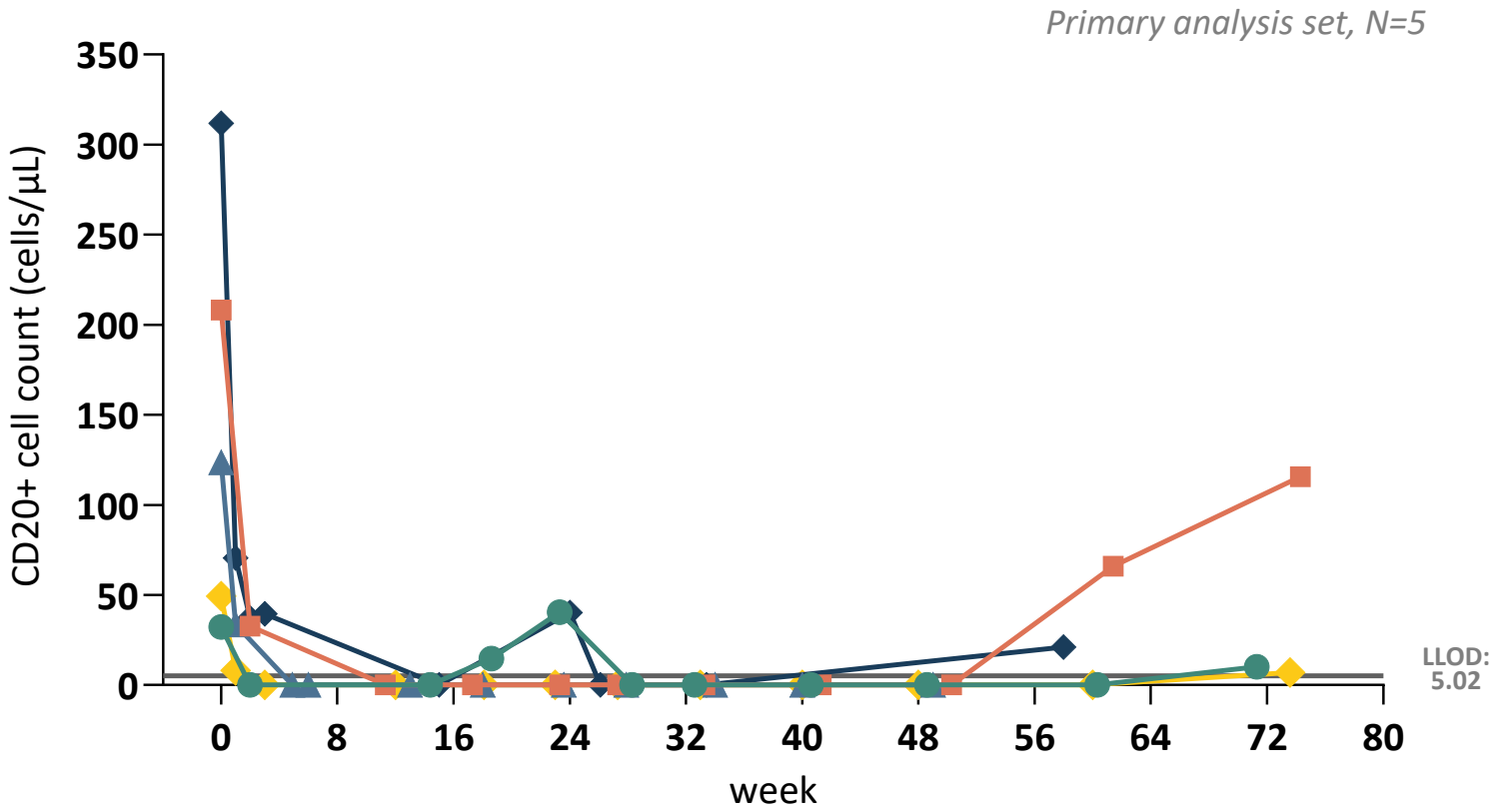


IV = intravenous, LTFU = long term follow-up, PLA2R = phospholipase A2 receptor, pMN = primary membranous nephropathy, UPCR = urine protein creatinine ratio

Cortazar ASN 2024; Baseline characteristics for primary analysis set (N=5), PLA2R from patients with positive anti-PLA2R antibodies at baseline (N=3)

Budoprutug Administration Resulted in Durable B Cell Depletion

Rapid and complete circulating B cell depletion observed in 100% of patients at doses of 100-200mg



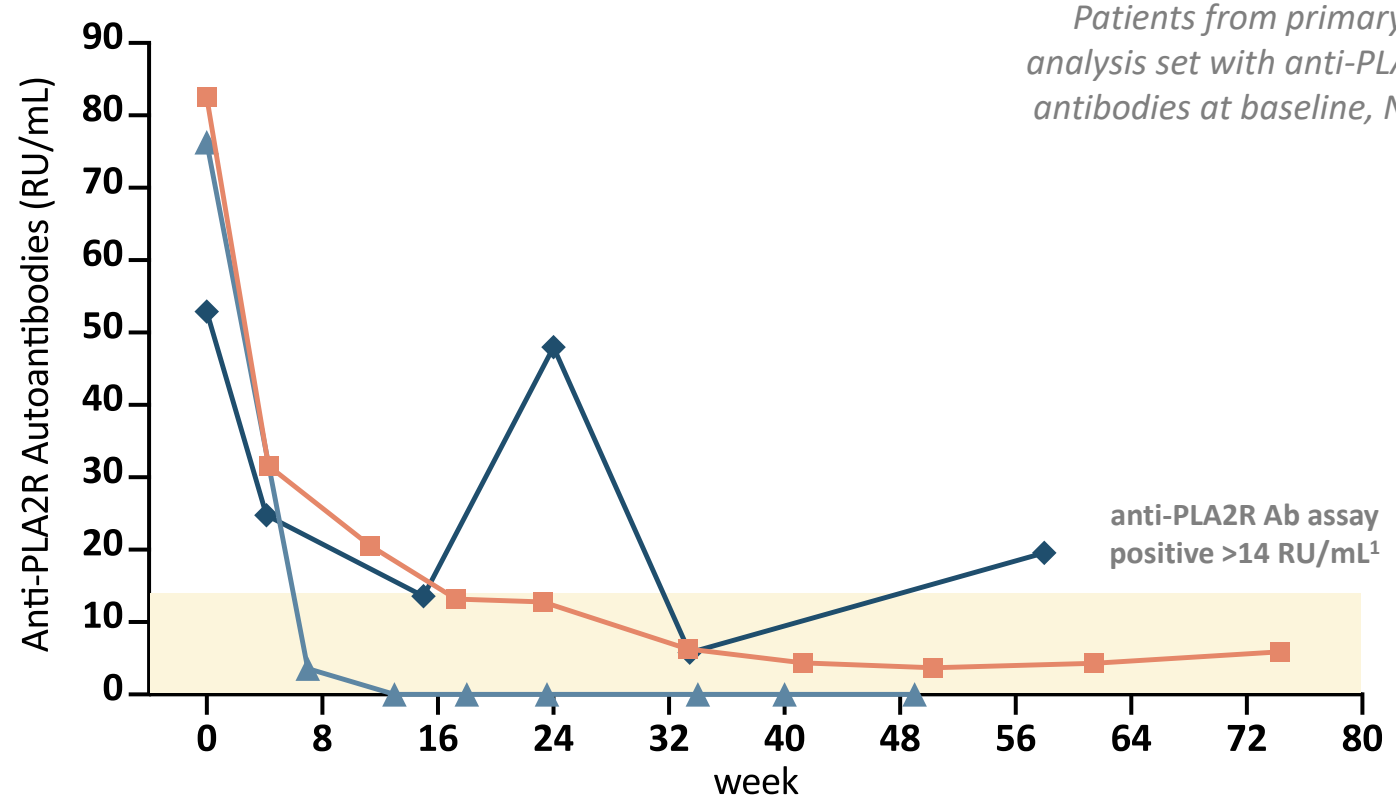
Complete circulating B cell depletion (CD20+ count < 5 cells/µL) achieved in all **5/5 patients**

- **Rapid depletion**: LLOD reached as soon as 2 weeks after first dose
- **Sustained reductions** out to 1 year + after two dose cycles

LLOD = lower limit of detection, pMN = primary membranous nephropathy
Cortazar ASN 2024; Phase 1b pMN: budoprutug administered in two IV infusions, administered 14 days apart, at Week 0 and Week 24

Budoprutug Administration Resulted in Serologic Remission

Anti-PLA2R antibody negativity achieved in 100% (3/3) evaluable patients



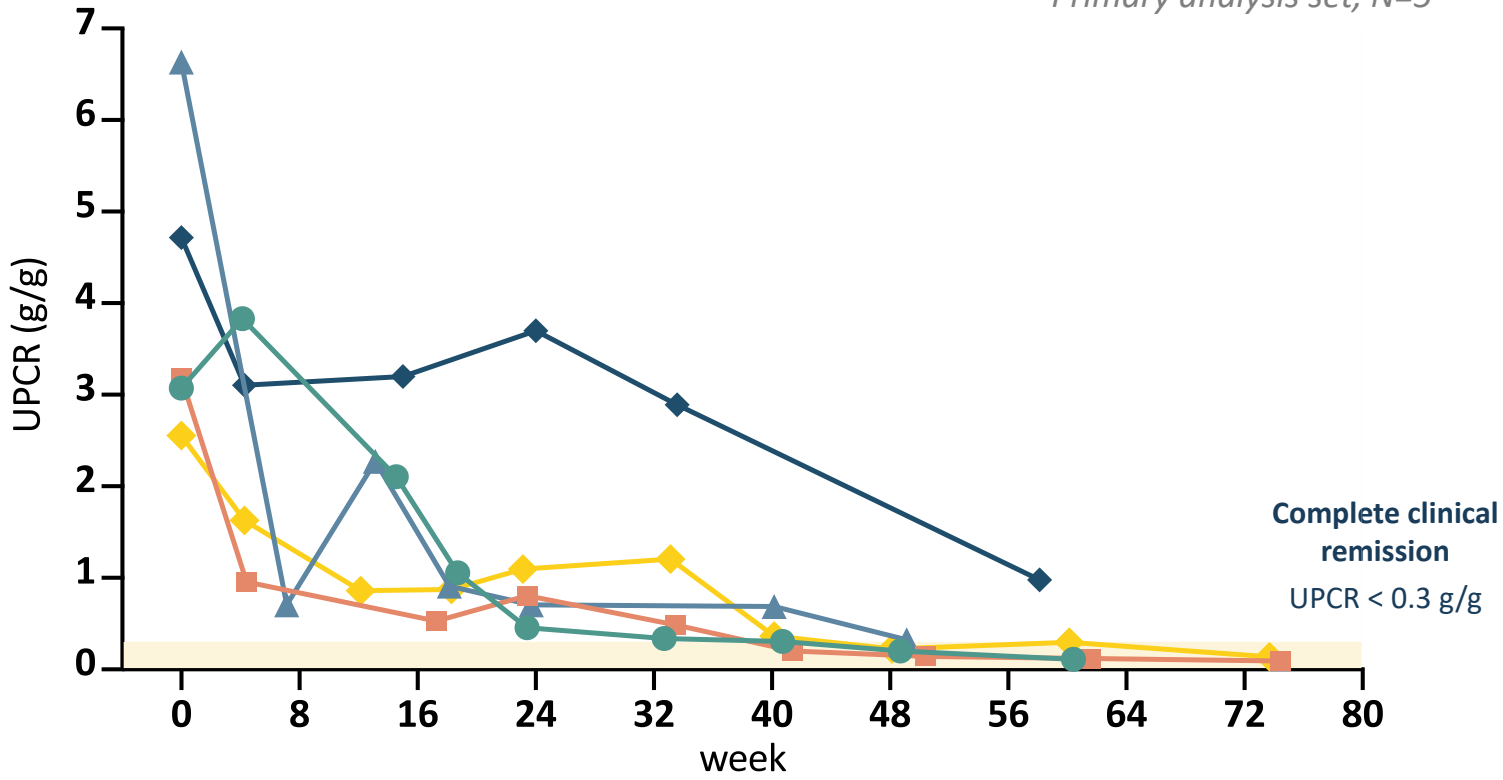
PLA2R antibodies are the primary disease driver in pMN and a key biomarker of clinical response[2]

- ~75% of pMN is mediated by anti-PLA2R autoantibodies
- Disappearance of anti-PLA2R antibodies precedes clinical remission

Ab = antibody, LLOD = lower limit of detection, PLA2R = phospholipase A2 receptor, pMN = primary membranous nephropathy
Cortazar, ASN 2024 ; Phase 1b pMN: budoprutug administered in two IV infusions, administered 14 days apart, at Week 0 and Week 24. 1. Stefan 2022, 2. Ronco J Clin Med 2021

Budoprutug Administration Led to Clinical Remission (Proteinuria)

All patients achieved complete or partial clinical remission by Week 48, as measured by UPCR



All patients achieved complete (3/5) or partial (2/5) clinical remission by Week 48

- 2 patients with partial clinical remission achieved serologic remission, suggesting further progress to full clinical remission may be possible

Long-term control of proteinuria for up to 3 years after initial dosing was observed in 4 patients who received up to 4 doses of budoprutug

- In 3 of these patients, no further immunosuppressive treatment was required

Proteinuria (UPCR) is a key measure of kidney function and is the primary outcome measure in pMN

pMN = primary membranous nephropathy, UPCR = urine protein creatinine ratio

Cortazar ASN 2024, Cortazar ASN 2025; Phase 1b pMN: budoprutug administered in two IV infusions, administered 14 days apart, at Week 0 and Week 24

Safety

Budoprutug was generally well tolerated in the completed Phase 1b pMN trial at doses of up to 200mg, supporting exploration of higher doses in ongoing and future studies

8 Patients received at least one injection of budoprutug and were included in the safety analysis population in the completed Phase 1b pMN trial

- There were no deaths on study
- There were 3 SAEs, none 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

AE = adverse event, SAE = serious adverse event
Cortazar ASN 2024

Budoprutug Has a Highly Competitive Profile in pMN

High serologic remission (anti-PLA2R negativity) and complete clinical remission rates support a potentially strong and differentiated clinical profile

	Budoprutug **(based on completed pMN Ph1b)**	Rituximab	Obinutuzimab	Povetacicept	Felzartamab
Target	CD19	CD20	CD20	BAFF/APRIL	CD38
Serologic remission (anti-PLA2R negativity)	✓ **100%** (3/3)[1]	64–95%[2] (titer decrease)	90-92%[6-7]	100% (4/4)[9]	23%[10]
Complete or partial clinical remission	✓ **100%** (5/5)[1]	60% (39/65)[2]	85% (50/59) to 95% (20/21)[6-7]	100% (5/5)[9]	35% (9/26)[10]
Complete remission	✓ **60%** (3/5)[1] *UPCR ≤0.3 g/g*	14-41%[3-5] *UPCR ≤0.3 g/g*	29-38%[6-8] *UPCR ≤0.3 g/g*	40% (2/5)[9] *UPCR <0.5 g/g*	n/a[10]
Dosing	✓ 2 IV doses administered 14 days apart, then q6m	2 x 1000 mg IV doses administered 7 days apart, then q6m	2 x 1000 mg IV doses administered 14 days apart, then q6m	80 mg SC every 4 weeks	9 IV doses over 6 months

Table above reflects cross-trial comparisons and not data from head-to-head studies; differences exist between trial designs and participant characteristics and caution should be exercised when comparing data across trials.

Note: To date, there are no FDA-approved therapies for pMN

IV = intravenous, PLA2R = phospholipase A2 receptor, pMN = primary membranous nephropathy, q6m = every 6 months; Serologic remission defined as <14 RU/mL, complete remission defined as UPCR ≤0.3 g/g for budoprutug, rituximab, obinutuzimab and UPCR <0.5 g/g for povetacicept, partial remission defined as UPCR between <3.5 g/g and ≥50% reduction from baseline.

1. Cortazar ASN 2024, 2. Fervenza NEJM 2019 - immunological remission not reported, 3. Fervenza Kid Int 2008, 4. Fervenza CJASN 2010, 5. Roccatello Autoimmun Rev 2016, 6. Hu CJASN 2024, 7. Su KI Reports 2024, 8. Lin Kid Med 2024, 9. Madan KI Reports 2025, 10. Rovin KI Reports 2024

PrisMN: Budoprutug Phase 2 pMN Study Enrolling

Biomarker endpoint provides potential to rapidly identify dose to carry forward into Phase 3 (NCT07096843); initial data anticipated in H2 2026

PrisMN
A NEW LENS ON PMN

Open-label, dose ranging study

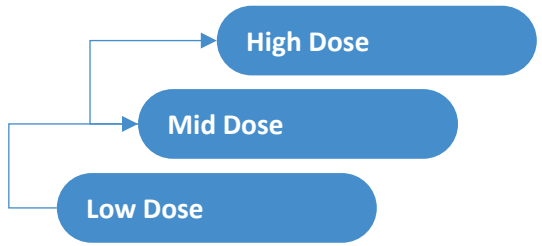
Population
- 18-70 years of age
- UPCR ≥ 2.0 g/g; designed to ensure adequate enrollment of patients with UPCR > 5.0 g/g
- PLA2R antibody positive

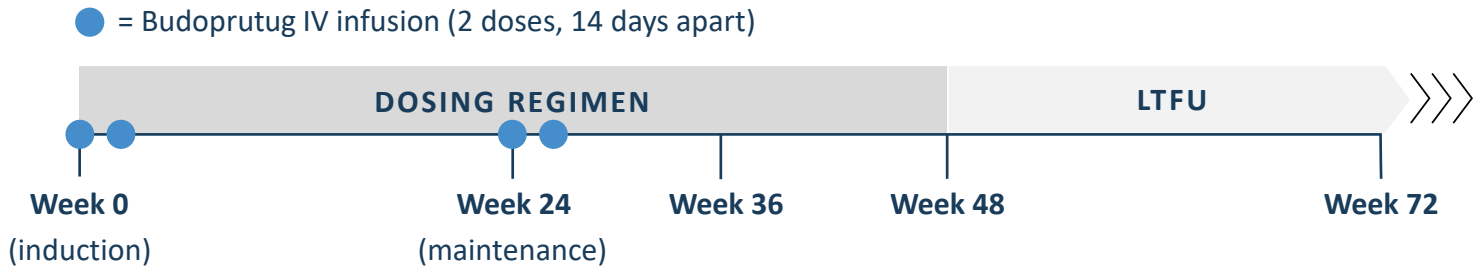
Primary Objective
- Safety and tolerability

Secondary Objectives
- Preliminary PK and PK/PD
- PD markers (B cells, anti-PLA2R, total Ig)
- Preliminary efficacy: complete and partial remission at week 48 (UPCR and eGFR)

DOSE ESCALATION

15 patients per cohort, enrolled sequentially





CLIMBBIO

eGFR = estimated glomerular filtration rate , Ig = immunoglobulin, IV = intravenous, LTFU = long term follow-up, PD = pharmacodynamic, PK = pharmacokinetic, PLA2R = phospholipase A2 receptor, pMN = primary membranous nephropathy, UPCR = urine protein creatinine ratio

Immune Thrombocytopenia (ITP)

Chronic bleeding disorder

- **Autoantibody mediated** disease characterized by the **destruction of platelets**
- **Results in bruising, bleeding episodes, hemorrhage** and fatigue

Limited treatment options

- **~80% fail first line** glucocorticoids; **~40% progress** on second line TPO-RAs
- **Rituximab** has demonstrated the potential to achieve long-term disease remission, but **only ~20%** maintain remission
- **Surgical removal of the spleen** may be considered for chronic cases or when therapeutic interventions fail

Defined endpoint

- **Platelet response** can demonstrate early proof-of-concept and served as primary endpoint measure for approvals in ITP

SIGNIFICANT OPPORTUNITY

~85,000 patients
in the US alone

THE CLIMB SOLUTION

Budoprutug has the potential to achieve disease remission in this high unmet need population

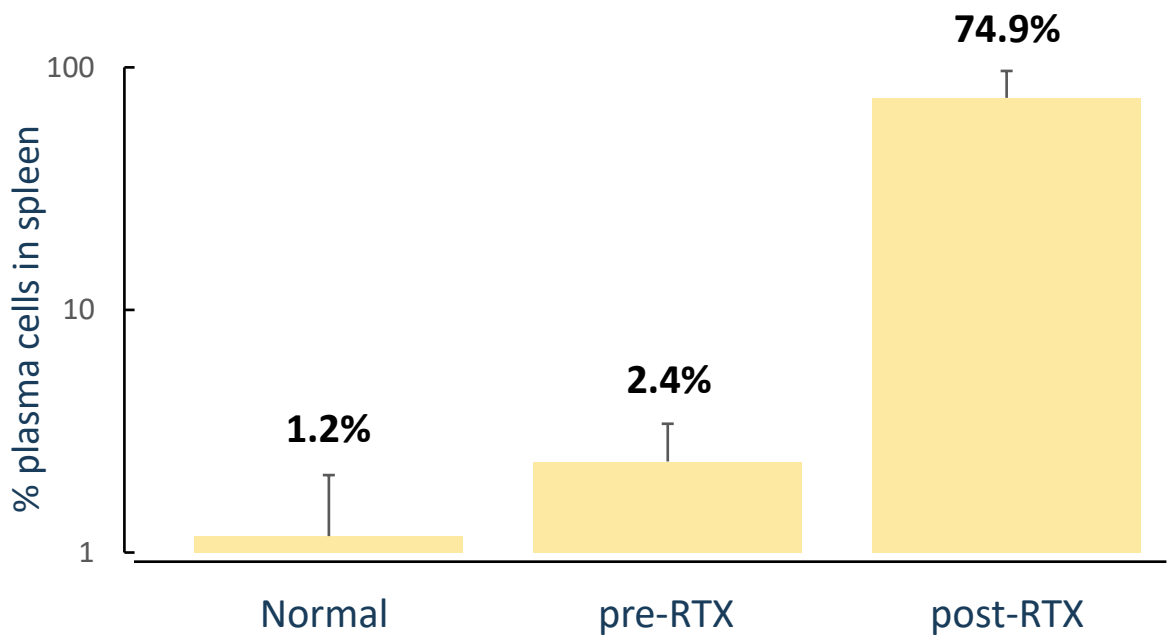
BTK = Bruton's tyrosine kinase, pMN = primary membranous nephropathy;
US Prevalence: Internal Research; StatPearls Pietras 2024, Gafter-Gvili A Euro J Int Med 2023, Lucchini Haematologica 2019, Internal Research

Strong Rationale for CD19 Approach in ITP

Potent anti-CD19 approach offers the promise of sustained elimination of anti-platelet antibodies

- B cell targeting via CD20 (rituximab) has demonstrated benefit in ITP, however up to 80% **fail rituximab**, **likely due to** the presence of **CD19+/CD20- B cells**
- **Anti-CD20 mAbs do not eliminate plasmablasts or plasma cells**, which continue to drive anti-platelet antibody production, while **CD19 is expressed on plasmablasts and some plasma cells**

CD19^{+}/CD20^{-} plasma cells expand within B cell niches post anti-CD20 treatment



Broader expression of CD19 across B cell lineage may overcome limitations of anti-CD20 therapies

ITP = Immune Thrombocytopenia, mAb = monoclonal antibody, RTX = rituximab
Mahevas J Clin Invest 2013, Crickx Kidney Intl 2020, Lucchini Haematologica 2019

Budoprutug Phase 1b/2a ITP Trial Enrolling

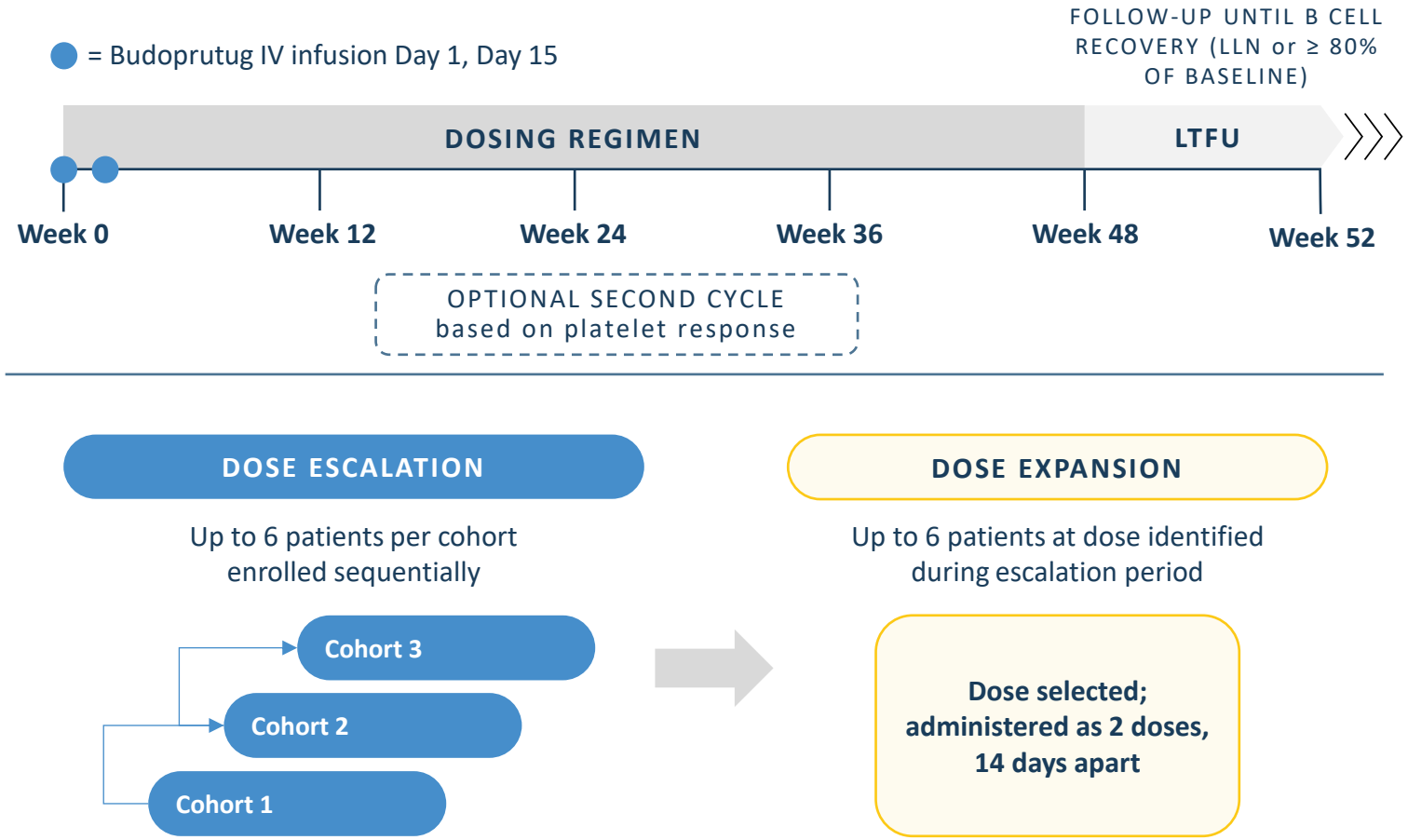
Designed to define dose and regimen, and establish depth and duration of platelet response and B cell depletion (NCT07043946); initial data anticipated in H2 2026

Open label, dose escalation and expansion study

Population
- N ~ 30 patients
- Insufficient response to 1 or more prior therapies
- Platelet count <30,000/µL

Primary Objective
- Safety and tolerability

Secondary Objectives
- Pharmacokinetic profile
- Effects on B cell depletion (pharmacodynamic response)
- Effects on platelet counts (ITP clinical response)



ITP = Immune Thrombocytopenia, LTFU = long term follow-up, LLN = lower limit of normal

Systemic Lupus Erythematosus (SLE)

Chronic autoimmune condition

- **Autoreactive lymphocytes and autoantibodies** mistakenly **attack tissues and organs** throughout the body
- Wide array of autoantibodies cause disease manifestations that can **affect virtually any organ system;** subset of patients have kidney involvement (lupus nephritis)
- **Relapses** may lead to **cumulative damage** and **organ failure**

High unmet need

- Steroids given first-line but are not a long-term solution
- ~50% of patients on approved biologics relapse
- **Off-label rituximab is a mainstay of treatment,** underscoring need for more effective therapies

Established PoC for CD19

- **CD19 CAR T**-cell therapies have demonstrated **potential for remission;** but **modality limits broad utility** and patient access

SIGNIFICANT OPPORTUNITY

~240,000 patients in the US alone

THE CLIMB SOLUTION

Budoprutug offers potential for broad B-cell targeting with the safety and convenience of a mAb

Approved biologics includes anti-BAFF (B-cell activating factor) and anti-interferon therapies.
CAR = chimeric antigen receptor, IFN = interferon, mAb = monoclonal antibody, PoC = proof-of-concept, SLE = systemic lupus erythematosus.
US Prevalence: Izmirly Arthritis Rheumatol 2021, Wang BMC Research Notes 2022, Marinho Front Immunol 2023, Wu J Manag Care Spec Pharm 2023.

Strong Rationale for Anti-CD19 mAb in SLE

CD19 mAb approach could provide optimal profile of disease control, safety and broad patient accessibility

B-cell targeting has promise

Anti-BAFF mAb approved for SLE/LN, anti-CD20 mAb filed for LN, rituximab used off-label

***but* up to 55% of patients still fail to achieve disease control**

Likely reasons for anti-CD20 treatment failure are addressable with CD19 targeting

- Persistence of CD19+ self-reactive B cell subsets
- Continued production of pathogenic autoantibodies by plasmablasts
- Rapid recovery of pathogenic B cell subsets

CD19 CAR Ts demonstrate strong efficacy

8/8 SLE patients treated with anti-CD19 CAR T-cells achieved disease remission by 6 months

***but* have significant risks and access challenges**

mAb approach can overcome key CAR T-cell challenges

- Low risk of CRS and ICANS, no lymphodepletion required
- Long treatment interval with the ability to easily retreat as needed
- Administered in the community setting

BAFF = B-cell activating factor, CAR = chimeric antigen receptor, CRS = cytokine release syndrome; ICANS = immune effector cell associated neurotoxicity syndrome, IFN = interferon, LN = lupus nephritis, mAb = monoclonal antibody, SLE = systemic lupus erythematosus

Furie NEJM 2025, Scherlinger Annals Rheumatic Diseases 2019, belimumab prescribing information, Roche Press release March 5 2025, Müller NEJM 2024

Budoprutug Global SLE Phase 1b Trial Enrolling

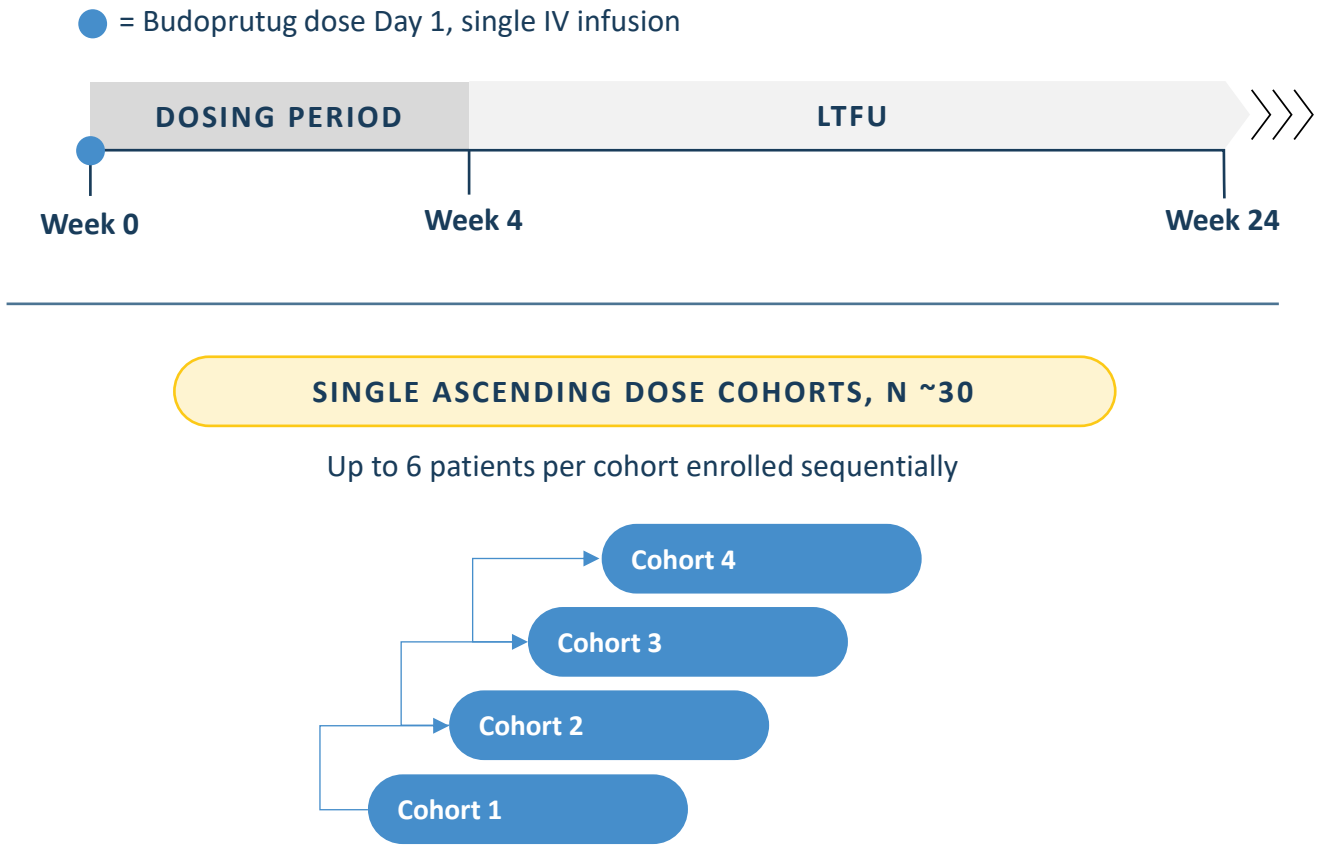
Designed to define optimal dose and assess depth and duration of B cell depletion and autoantibody reduction (NCT07011043); initial data anticipated in H2 2026

Open-label, dose escalation study

Population
- N ~ 30 patients
- Seropositive SLE with SLEDAI≥8
- Refractory to adequate trials of at least 2 therapies
- Stable protocol-limited background therapy at entry

Primary Objective
- Safety and tolerability

Key Secondary/Exploratory Objectives
- Effects on B cell depletion (PD response), autoantibody levels, and protective antibody levels
- PK and PK/PD profile
- Preliminary signs of clinical activity
- Kinetics of re-population of B cell subsets and antibodies after depletion



IV = intravenous, LTFU = long term follow-up, PD = pharmacodynamic; PK = pharmacokinetic; SLE = Systemic Lupus Erythematosus

Budoprutug China SLE Phase 1b Trial in Start-up

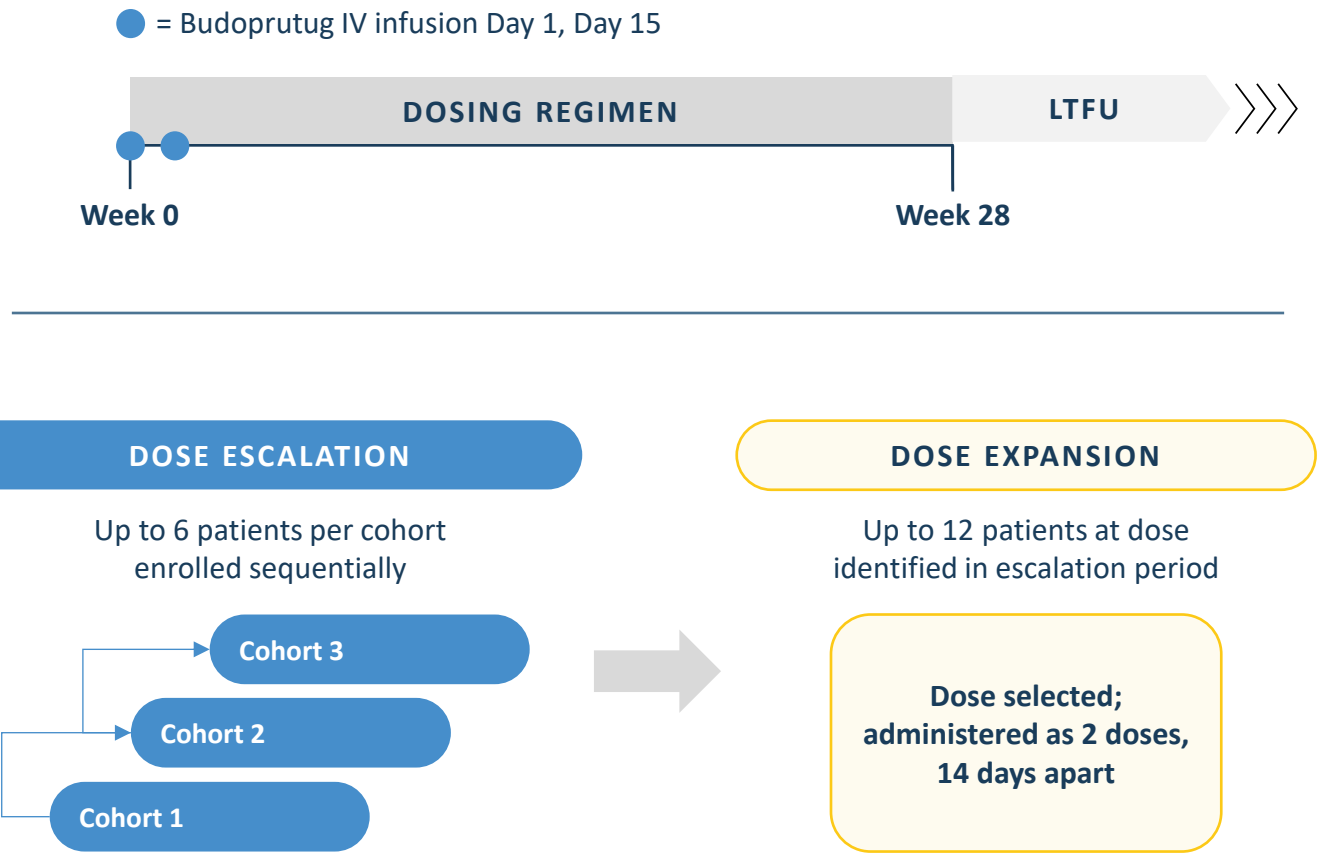
Companion to global study, with potential to evaluate lupus nephritis; Dosing of first patient anticipated H1 2026

Open-label, dose escalation study

Population
- N ~ 30 patients
- Seropositive SLE with SLEDAI≥6
- Refractory to adequate trials of at least 1 therapy
- Stable protocol-limited background therapy at entry

Primary Objective
- Safety and tolerability

Key Secondary/Exploratory Objectives
- Effects on B cell depletion (PD response), autoantibody levels, and protective antibody levels
- PK and PK/PD profile
- Preliminary signs of clinical activity
- Kinetics of re-population of B cell subsets and antibodies after depletion



IV = intravenous, LTFU = long term follow-up, PD = pharmacodynamic; PK = pharmacokinetic; SLE = Systemic Lupus Erythematosus, SLEDAI = Systemic Lupus Erythematosus Disease Activity Index

Budoprutug – Subcutaneous (SC) Formulation

Anti-CD19

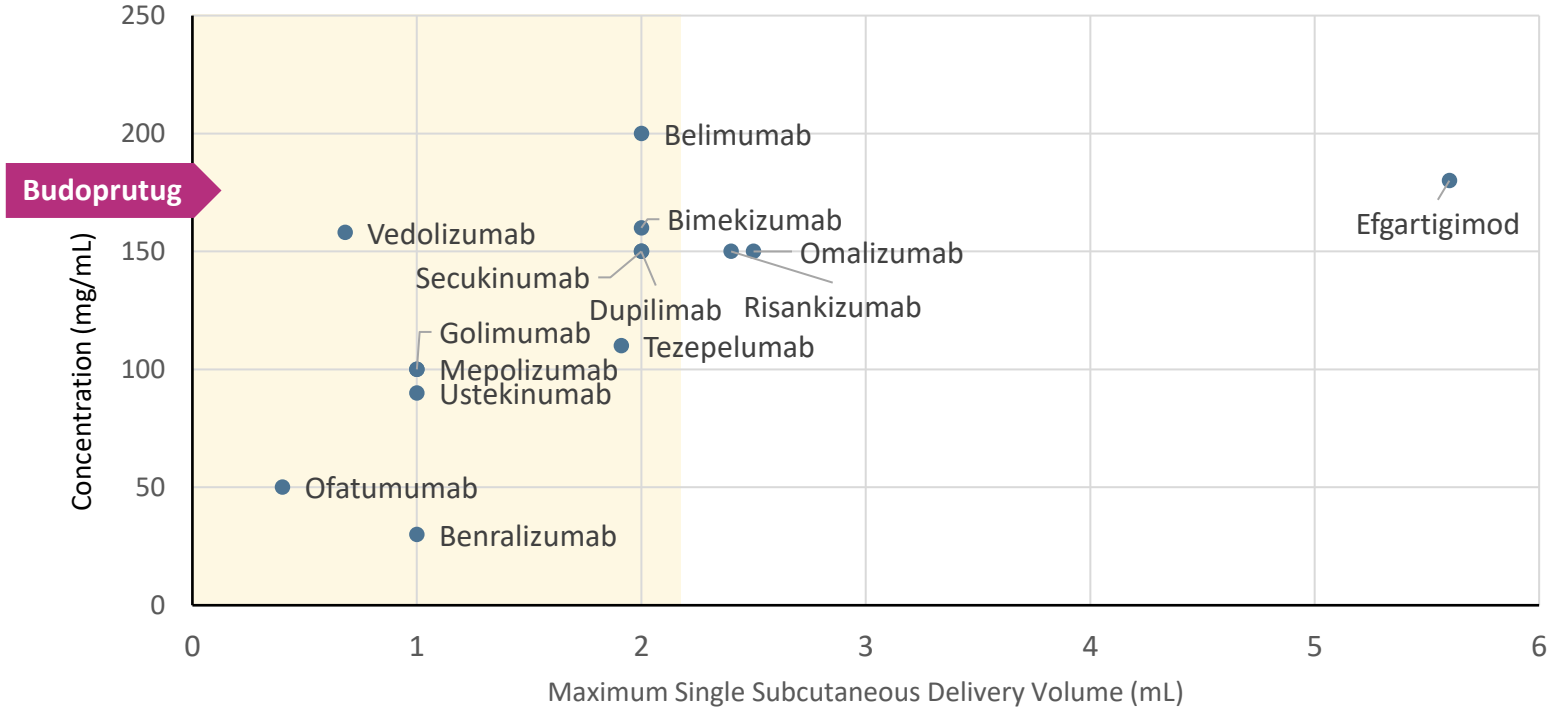
Budoprutug SC Formulation Provides Optionality and Differentiation

Ability to formulate as both IV and SC provides optionality in development and may be a differentiating feature of budoprutug relative to other anti-CD19 mAbs

Potential Value of SC Administration

- Patient and provider flexibility
- Potential for at-home administration, reducing burden on healthcare facilities and improving patient convenience
- Potential to broaden target patient populations or indications

Budoprutug SC concentration and volume targets defined by benchmark immunology mAbs

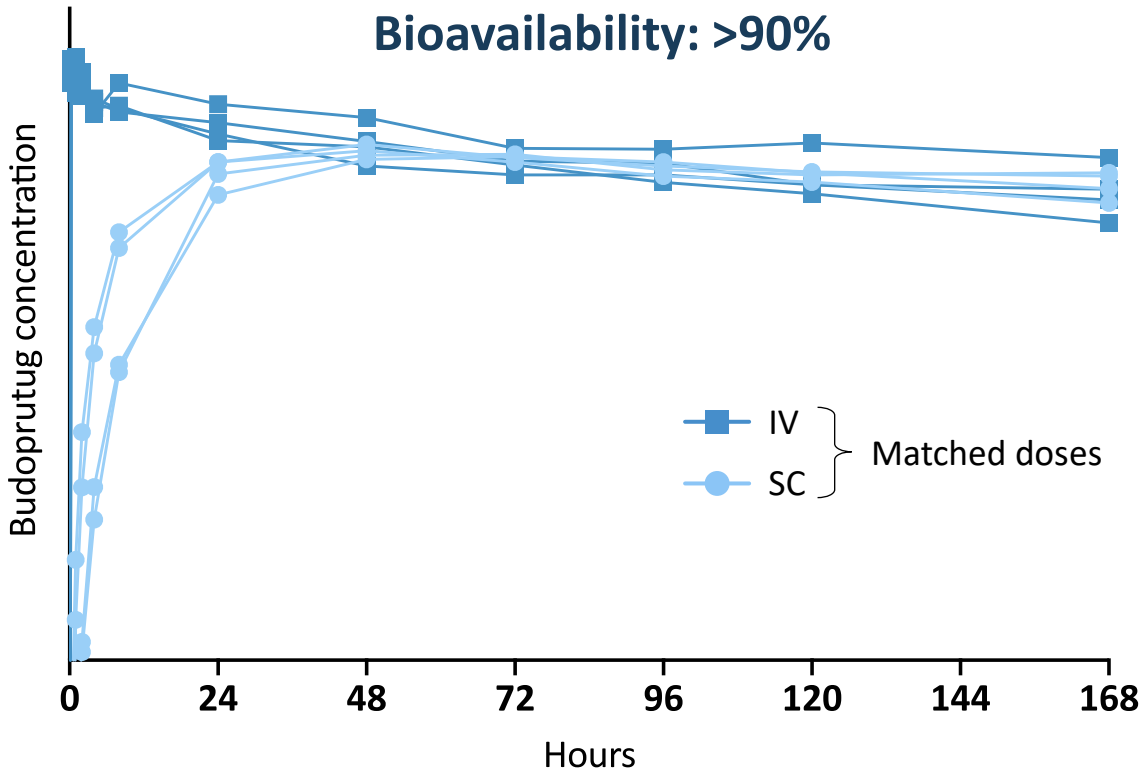


IV = intravenous, mAbs = monoclonal antibodies, SC = subcutaneous

Source: FDA-approved labels. Concentration normalized to mg/mL; where multiple indications or dosing regimens are approved, the highest volume single dose was used

NHP Data Supports Viability of Budoprutug SC Formulation

Budoprutug SC formulation demonstrated high bioavailability and favorable tolerability



Favorable Tolerability Profile

- No local tolerance issues identified on histopathology
- No safety findings
- No observed anti-drug antibodies (ADAs)

High concentration, low viscosity, and high bioavailability provide potential for a high concentration, single SC injection

IV = intravenous, NHP = nonhuman primates, SC = subcutaneous

Note: Each line in the bioavailability figure represents an individual animal (n= 4/arm)

Budoprutug SC Formulation Phase 1 Trial Dosing Completed

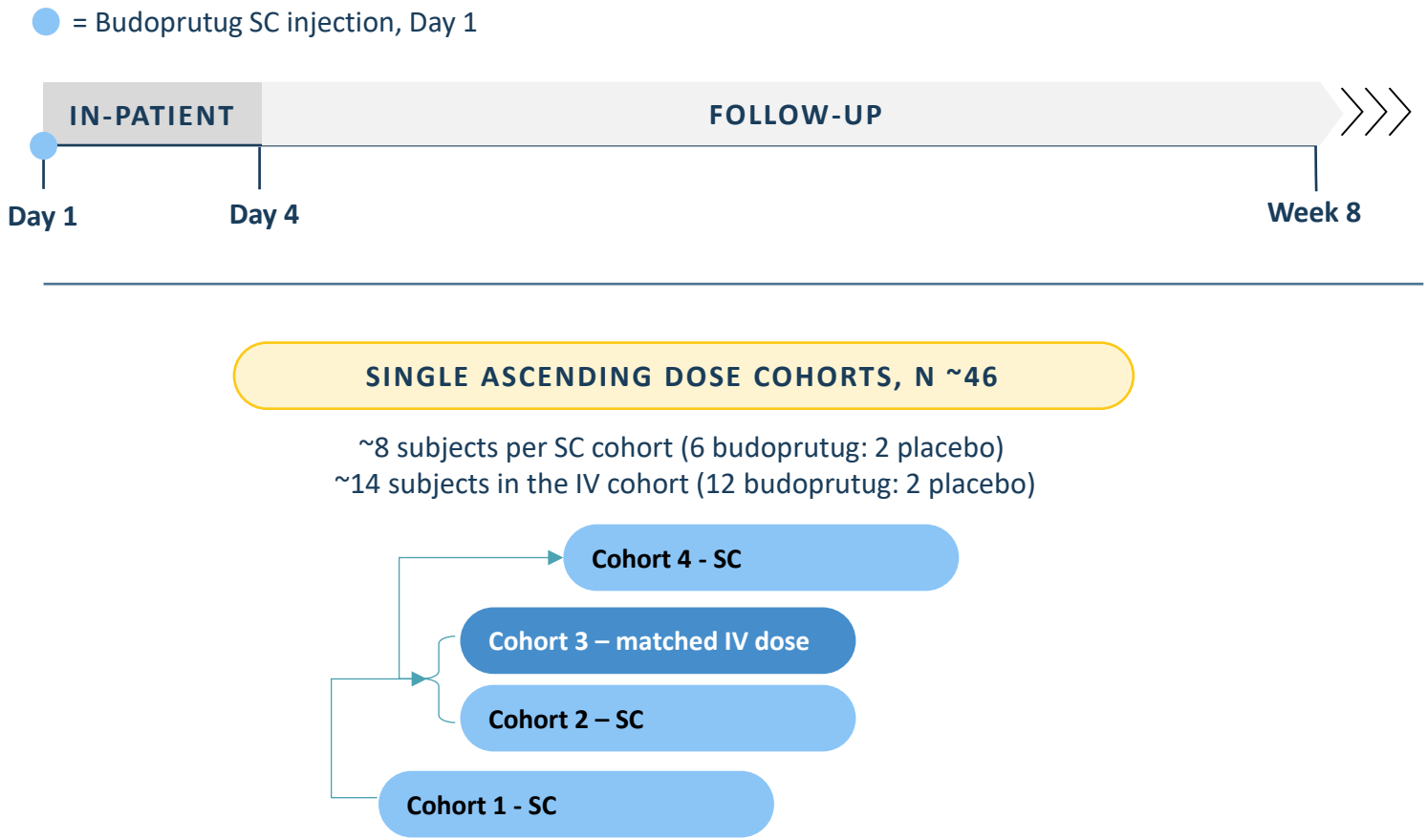
Clinical data anticipated H1 2026 (NCT07090655)

Randomized, double-blind, placebo-controlled, single ascending dose study

Population
- N ~ 46 healthy volunteers

Primary Objective
- Safety and tolerability

Secondary Objectives
- Pharmacokinetic profile
- Effects on B cell depletion (pharmacodynamic response)
- Bioavailability of SC formulation



IV = intravenous, SC = subcutaneous. Follow-up will continue for subjects whose B cell count is not above 50% of baseline or above lower limit of normal by week 8

CLYM116

Anti-APRIL mAb

CLYM116: A Potential Best-in-Class anti-APRIL mAb

Initial clinical data expected mid-2026

- NHP data demonstrated potential for deep and durable IgA suppression, long half-life, and acceptable safety profile

- CLYM116 has the potential to provide:
  - ✓ Improved activity
  - ✓ Less frequent dosing
  - ✓ Favorable safety profile

- Phase 1 clinical trial in healthy volunteers to evaluate safety and PK/PD ongoing, with initial data expected mid-2026
- Mabworks Phase 1/2 trial in healthy volunteers and patients with IgAN in China also ongoing*

*CLYM116 is being developed as MIL116 in Greater China by our partner, Beijing Mabworks Biotech (NCT07375758)

APRIL = a proliferation-inducing ligand, HV = healthy volunteer, mAb = monoclonal antibody, NHP = nonhuman primate

IgA Nephropathy (IgAN)

Progressive, lifelong renal disease

- **Autoantibody mediated** disease caused by deposition of immune complexes in the glomeruli, which leads to proteinuria, kidney injury and loss of kidney function
- Diagnosed **early in life** (typically, ages 15-40)[1]; patients at **risk for renal failure** as disease progresses
- **Lifelong disease** – patients likely to require chronic therapy

Growing market

- Treatment goals are to **normalize proteinuria and preserve kidney function** (stabilize eGFR)[2]
- KDIGO 2025 Guideline updates likely to increase diagnosis rates, expand patient population requiring treatment, and increase proportion of patients receiving potential disease-modifying therapy
- Market expected to reach **~ $10-20B annually**[3-5]

Rapid and defined path to approval

- Accelerated approval based on **reduction in proteinuria** with full approval based on **stabilization of eGFR**
- **Biomarkers (APRIL, IgA)** enable rapid assessment of clinical profile during early development

SIGNIFICANT OPPORTUNITY

Most common primary glomerular disease worldwide, **~200,000** patients in the US alone

THE CLIMB SOLUTION

CLYM116 is a potential best-in-class anti-APRIL mAb, designed for improved activity and less frequent dosing

APRIL = a proliferation-inducing ligand, eGFR = estimated glomerular filtration rate, mAb = monoclonal antibody

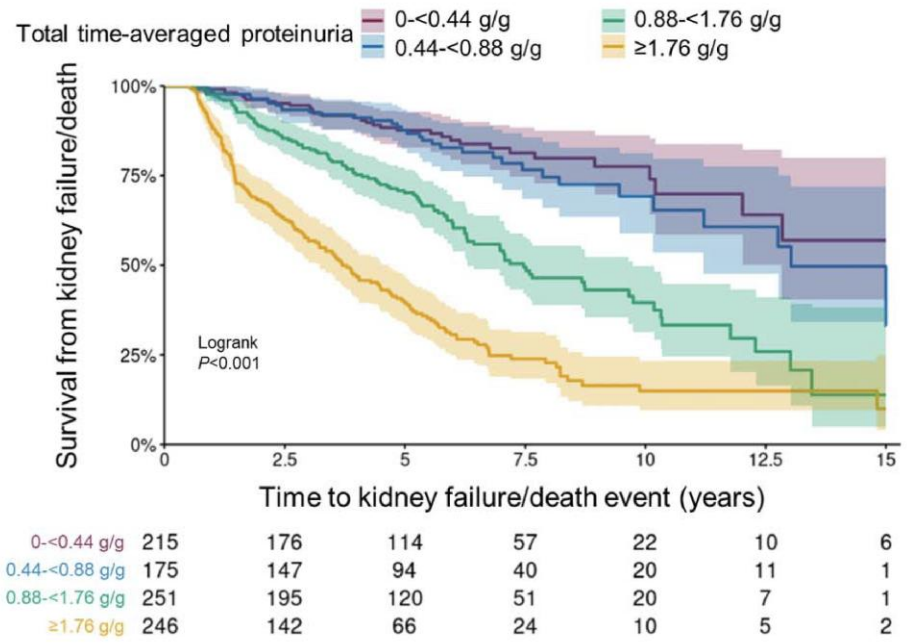
US Prevalence: Stoneman JAMA 2026; [1] Schena Semin Nephrol 2018, [2] KIDGO 2025 Clinical Practice Guideline for the Management of IgAN, [3] Cantor IgAN Report Mar 2025, [4] Goldman Sachs IgAN Report Aug 2025, [5] Oppenheimer IgAN Report Aug 2025.

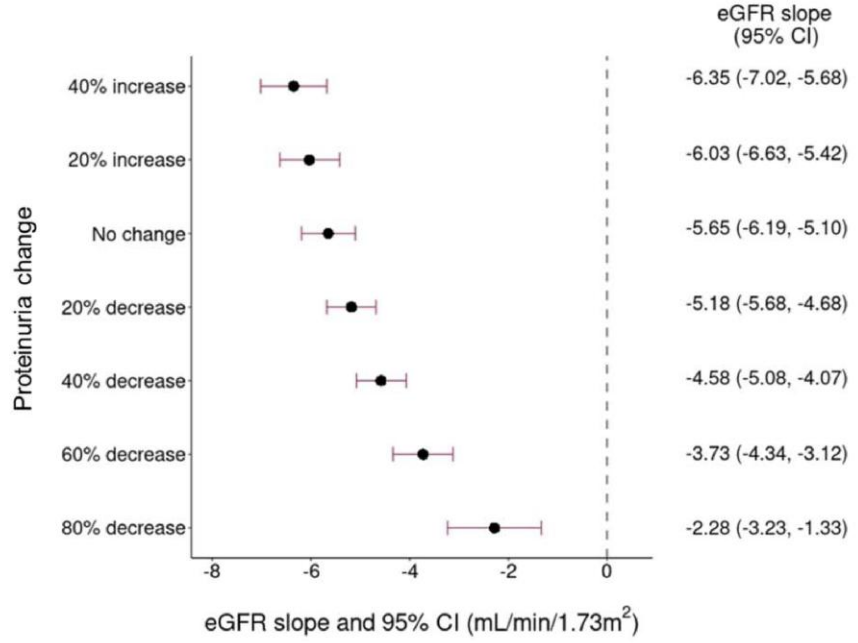
CLIMBBIO

Proteinuria is an Established Surrogate Endpoint for IgAN

Prior approvals provide precedent regarding study design and registrational endpoints, including proteinuria at 9 months for accelerated approval and stabilization of eGFR at 2 years for full approval

Proteinuria is associated with eGFR decline and kidney failure/death (UK RaDaR study)[4]





- Magnitude of proteinuria predicts development of kidney failure/death
- Proteinuria improvement (6-12 month) correlates with long-term (6-30 month) kidney function improvement

CLIMBBIO

eGFR = estimated glomerular filtration rate, IgAN = IgA nephropathy, RaDaR = UK National Registry of Rare Kidney Diseases

Sources: Inker Am J Kidney Dis 2021, Inker Am J Kidney Dis 2016, Thompson CJASN 2019, FILSPARI Drugs@FDA, TARPEYO Drugs@FDA. UK RaDaR: Pitcher CJASN 2023.

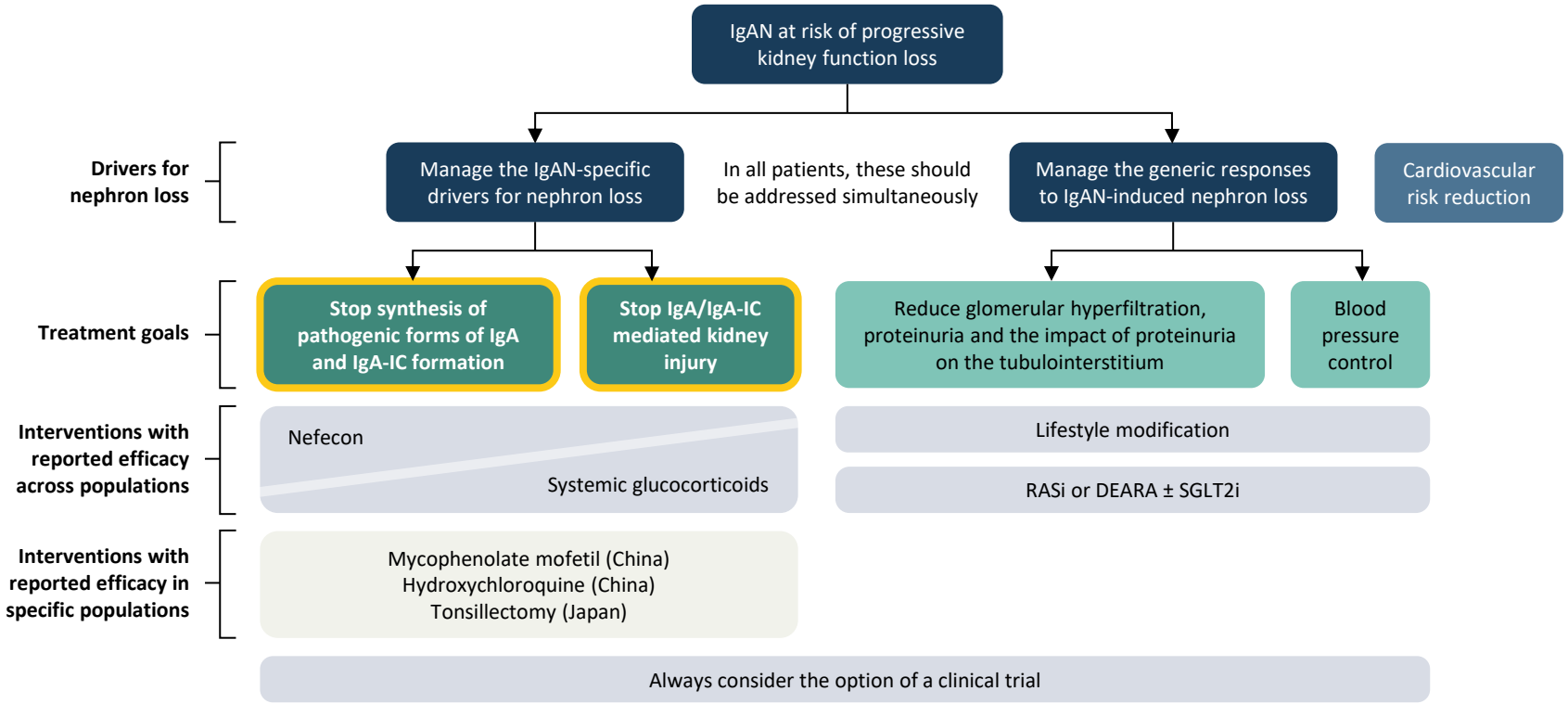
KDIGO 2025 Guideline Highlights Role for IgA Reduction in IgAN

KDIGO updates reinforce need for disease modifying treatments that halt the production of pathogenic IgA, positioning anti-APRIL therapies as a core pillar in the treatment of IgAN

KDIGO 2025 Guidelines recommend important changes:

- Lower threshold for biopsy (**proteinuria >=0.5 g/day**) to enable earlier diagnosis
- **Treatment initiation** in patients with **proteinuria >=0.5 g/day**
- Revised **treatment goal**: proteinuria maintained at <0.5 g/day, preferably <0.3 g/day
- Use of multiple treatment strategies simultaneously

Initiation of treatment with therapies that both prevent/reduce pathogenic IgA and immune complex formation and manage disease-induced nephron loss

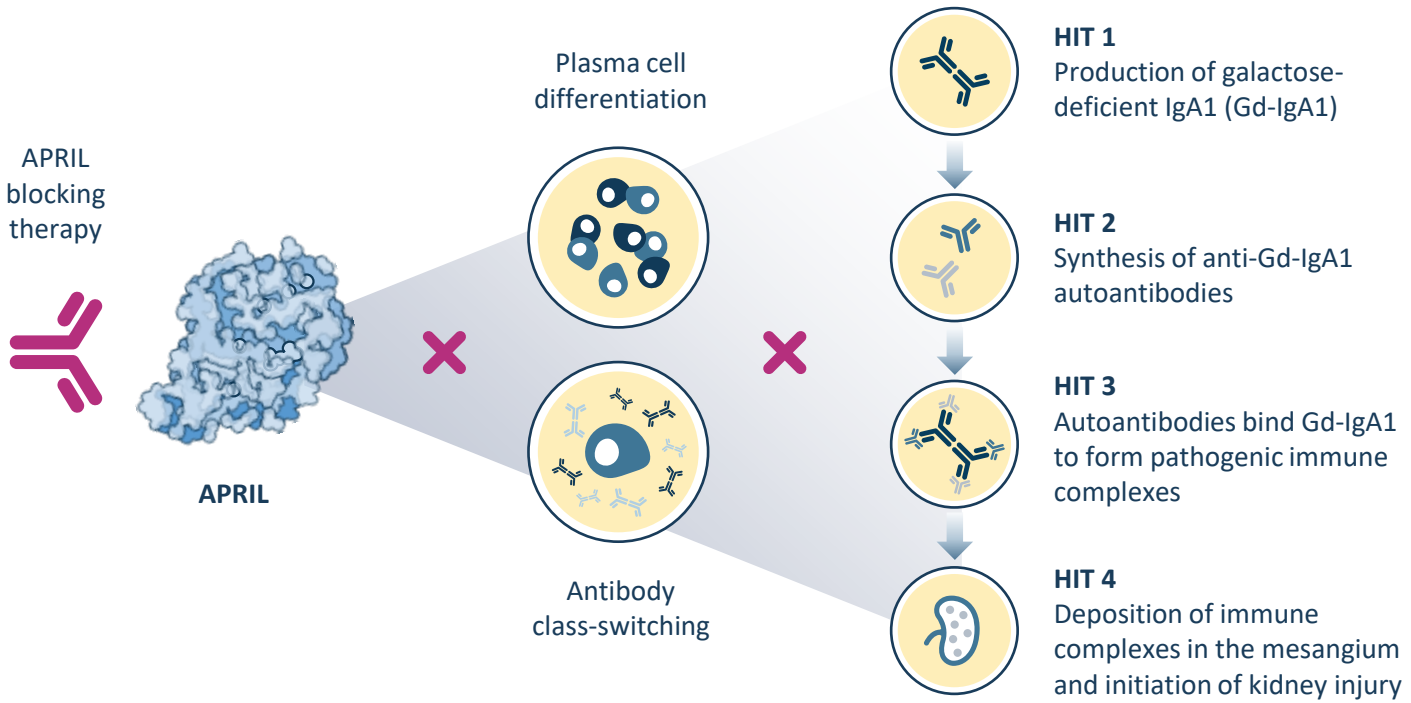


APRIL = a proliferation-inducing ligand, IgAN = IgA nephropathy , KDIGO = Kidney Disease Improving Global Outcomes

KIDGO 2025 Clinical Practice Guideline for the Management of IgAN

APRIL is a Potentially Disease Modifying Approach in IgAN

In IgAN, APRIL inhibition has been demonstrated to prevent the production of pathogenic IgA and the consequent immune complex formation that leads to kidney damage



Clinical endpoints in IgAN assess key measures of disease activity

IgA and **Gd-IgA1** serve as pharmacodynamic biomarkers in early clinical studies[1-3]

Proteinuria reductions and **eGFR** stabilization reflect potential to preserve kidney function and support accelerated and full approval, respectively[4,5]

APRIL = a proliferation-inducing ligand, eGFR = estimated glomerular filtration rate, IgAN = IgA nephropathy
Adapted from Mathur J Clin Med 2023. [1] Mathur KI Reports 2022, [2] Davies Clin Transl Sci 2024, [3] Kooienga Kid Intl 2025, [4] Thompson CJASN 2019, [5] Pitcher CJASN 2023

Selective APRIL Inhibition Has Been Clinically Validated in IgAN

Anti-APRIL mAb, sibeprenlimab, demonstrated numerically better proteinuria (UPCR) reductions as compared to anti-BAFF/APRIL antagonist, atacicept, in Phase 3 IgAN studies

	Sibeprenlimab[1] (anti-APRIL mAb)	**Atacicept[2]** (TACI-IgG Fc)
Dose	400 mg SC, Q4W	150 mg SC, QW
N	320*	203**
UPCR change at 9 months	**-50.2%** vs. +2.1% for placebo	-46% vs. +7% for placebo
UPCR reduction at 9 months (placebo-adjusted)	**51.2%** p<0.0001	**42%** p<0.0001

- **Dual BAFF/APRIL inhibition does not appear to provide an efficacy benefit** beyond APRIL inhibition alone in IgAN
- APRIL only approach avoids potential immunosuppression associated with BAFF inhibition

Table above reflects cross-trial comparisons and not data from head-to-head studies; differences exist between trial designs and participant characteristics and caution should be exercised when comparing data across trials.

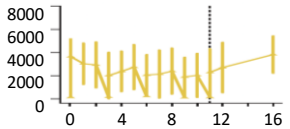
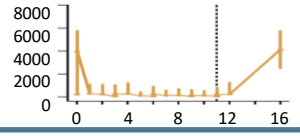
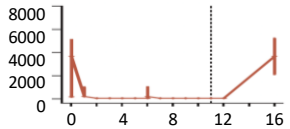
*Interim analysis of 320 patients, total of 530 enrolled; **Interim analysis of 203 patients, total of 431 enrolled; [1] Perkovic ERA 2025, 40 week endpoint, [2]Vera Corporate Presentation, June 2025, 36 week endpoint. APRIL = a proliferation-inducing ligand, BAFF = B-cell activating factor, IgAN = IgA nephropathy, mAb = monoclonal antibody, QW = once weekly, Q4W = once every four weeks, SC = subcutaneous, UPCR = urine protein creatinine ratio

CLIMBBIO

Next Gen Anti-APRILs Have Potential to Deliver Improved Profiles

Opportunity for next generation anti-APRIL agents to demonstrate improved efficacy, less frequent dosing, and reduced immunogenicity

Clinical data[1] suggest that sibeprenlimab recommended dosage[2] may not completely suppress APRIL or provide optimal proteinuria control

Dose (IV, Q4W)	APRIL levels over 16 wks	Change in UPCR at 12 mo	Clinical Remission % patients with UPCR < 0.3 g/d at 12 mo
2 mg/kg		-47.2%	7.9%
4 mg/kg		-58.8%	12.2%
8 mg/kg		**-62.0%**	**26.3%**

Sibeprenlimab recommended dosage (400 mg SC) roughly equivalent to 4 mg/kg IV[3]

Immunogenicity was observed in the Phase 3 sibeprenlimab study, which resulted in an impact on drug exposure and proteinuria reductions [2]

34% evaluable patients developed ADA

In patients who developed ADA:

- Drug exposure was ~40% lower
- UPCR reductions at Month 9 were lower (41.6% vs. 52.7%)

OPPORTUNITY FOR NEXT GENERATION APPROACH

Improved efficacy: more robust proteinuria reductions through deeper APRIL suppression, getting more patients to clinical remission

Less frequent dosing: reduced injection frequency through more prolonged APRIL suppression

Favorable safety profile, with **reduced immunogenicity:** supporting chronic administration

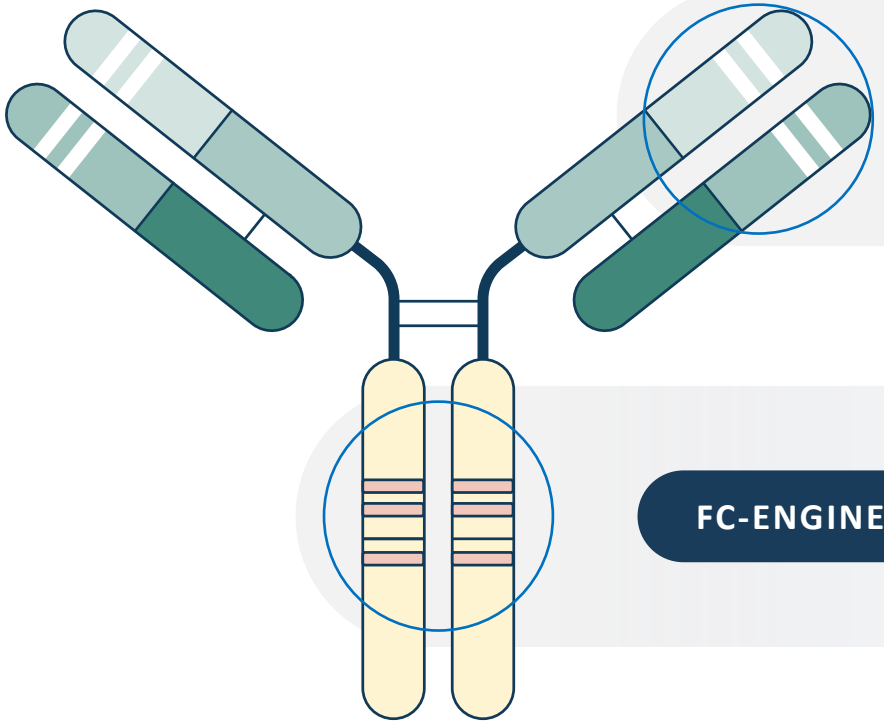
CLIMBBIO

[1]Chart adapted from Mathur NEJM 2024 (Ph2 study results), [2] Sibeprenlimab prescribing information, [3] Equivalency based on average 80 kg weight and 75% bioavailability (Zhang Clin Pharm Drug Dev 2023). APRIL = a proliferation-inducing ligand, IV = intravenous, mo = months, Q4W = once every four weeks, SC = subcutaneous, UPCR = urine protein creatinine ratio, wks = weeks

CLYM116 Is The Only Known “Sweeper” Anti-APRIL In Development

Potential best-in-class anti-APRIL mAb, designed for improved activity, less frequent dosing, and favorable safety profile

KEY FEATURES



HIGH AFFINITY

pH-dependent binding to APRIL

FC-ENGINEERED

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

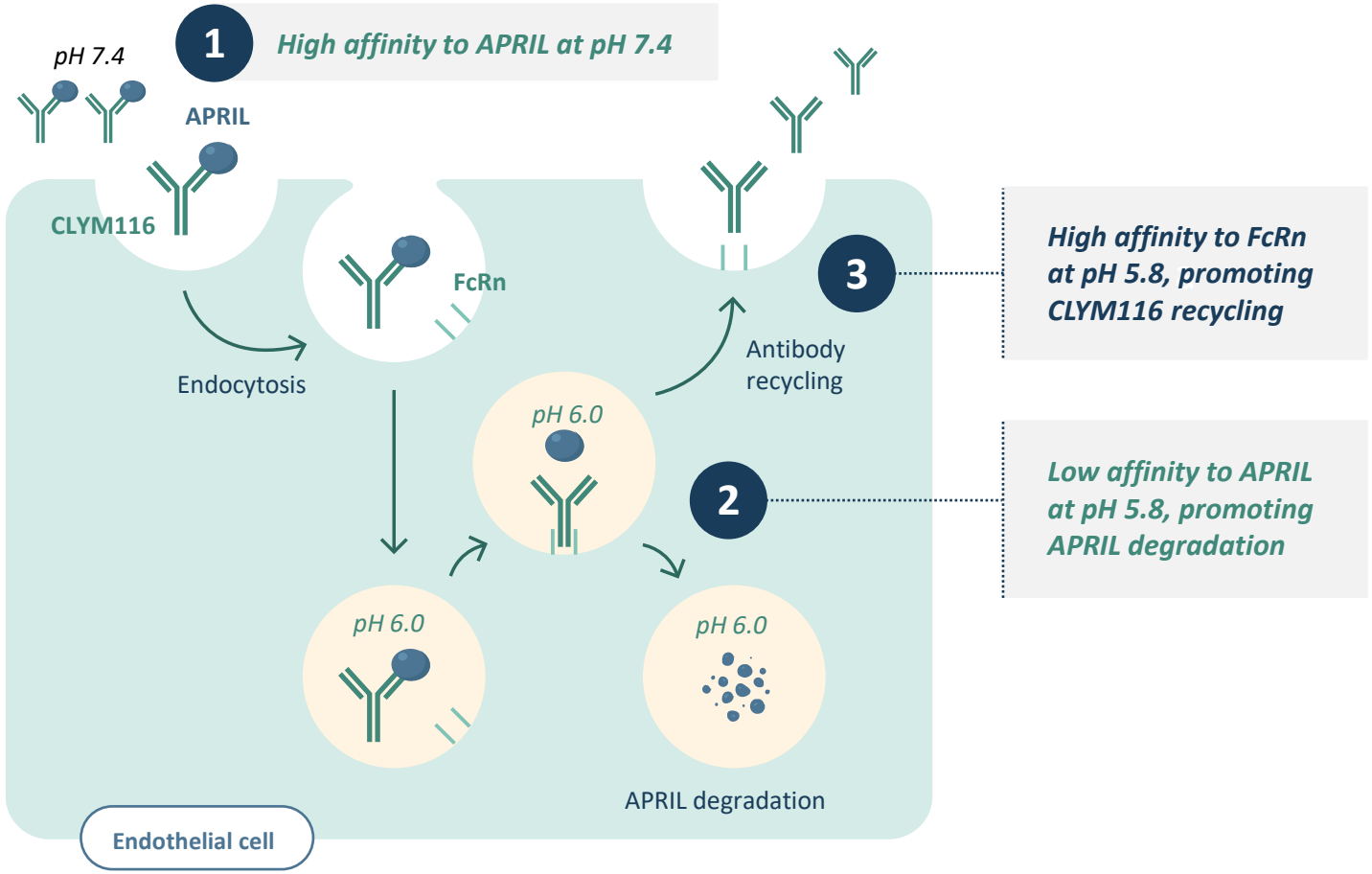
SWEEPER MECHANISM

Facilitates recycling of CLYM116 and elimination of APRIL

APRIL = a proliferation-inducing ligand, mAb = monoclonal antibody

CLYM116 “Sweeper” MoA Provides Potential for Clinical Benefits

CLYM116’s recycling degrader ‘sweeper’ mechanism of action provides potential for improved activity and less frequent dosing vs. first generation approaches or half-life extension alone



pH-dependent binding to APRIL provides potential for enhanced APRIL elimination through both:

1. **potent blocking of APRIL** binding to its receptors ***and***
2. **promotion of APRIL degradation** in the lysosome

Efficient antibody recycling 3 reduces clearance of CLYM116, resulting in potentially longer half-life

APRIL = a proliferation-inducing ligand, MoA = mechanism of action

CLYM116 *In Vitro* and *In Vivo* Data Support Sweeper Mechanism

Preclinical data demonstrate the potential for CLYM116 to provide a differentiated activity profile

CLYM116 demonstrated:

- ✓ **Potent, pH-dependent binding of APRIL** in an *in vitro* binding assay, as compared to first-generation anti-APRIL mAbs (sibeprenlimab and zigakibart) which did not demonstrate this profile
- ✓ **Fewer high molecular weight (HMW) complexes** vs sibeprenlimab in an HPLC (high performance liquid chromatography) analysis; HMW complexes may increase risk of immunogenicity
- ✓ **More effective APRIL depletion and clearance** as compared to first-generation anti-APRIL mAbs (sibeprenlimab and zigakibart) in APRIL degradation assay in a C57BL/6 mouse model
- ✓ **More efficient antibody recycling** as compared to first-generation anti-APRIL mAbs (sibeprenlimab and zigakibart) in an antibody exposure humanized FcRN transgenic mouse model

APRIL = a proliferation-inducing ligand, mAbs = monoclonal antibodies; Beeck, ASN 2025

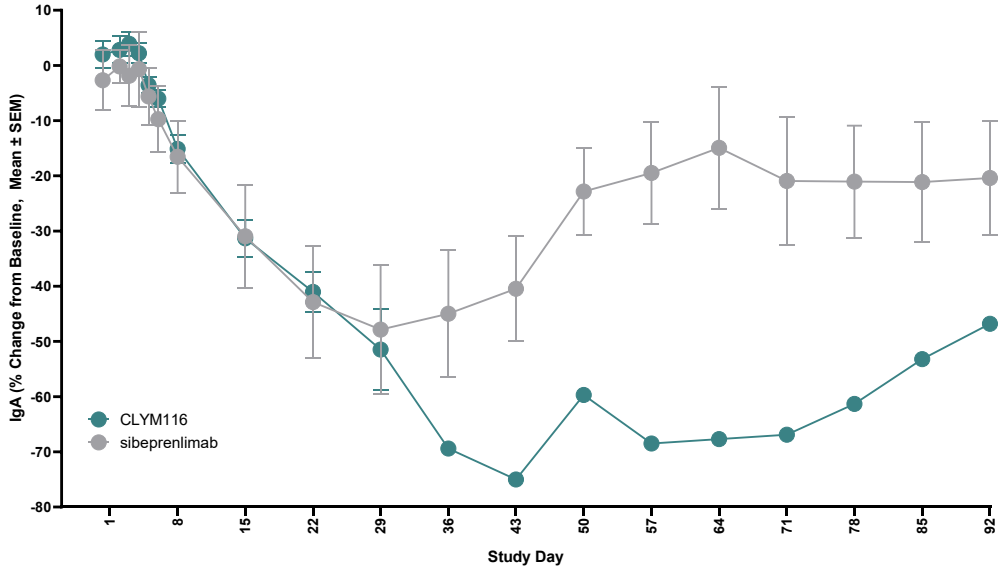
CLYM116 Showed a Differentiated PK/PD Profile in NHPs

CLYM116 demonstrated high bioavailability, a ~2-3 fold longer half-life vs. sibeprenlimab and deep and durable suppression of IgA after a single SC dose in NHPs

In a head-to-head study in NHPs, CLYM116 SC demonstrated:

- **~85% bioavailability** and **favorable tolerability**, with a formulation designed to support **potential for convenient, at-home dosing**
- **~2-3x longer half-life** across doses as compared to sibeprenlimab, supporting **potential for improved exposure and less frequent dosing in humans**
- **Deeper and more prolonged IgA reduction** compared to sibeprenlimab after a single subcutaneous administration at equivalent doses (6 mg/kg), supporting **potential to demonstrate a differentiated activity profile**

CLYM116 demonstrated >70% maximal reduction in IgA after a single 6 mg/kg SC dose, with >50% reduction in IgA maintained out to 3 months



Confirmed ADA+ animals were excluded from the analysis

ADA = anti-drug antibody, NHPs = nonhuman primates, SC = subcutaneous
Data from a head-to-head study in nonhuman primates; single subcutaneous administration. Sibeprenlimab analog generated from publicly available sequence.
Beeck ASN 2025

CLYM116 Has The Potential For A Best-in-Class Profile in IgAN

CLYM116 development ongoing, NHP data support potential for a differentiated profile

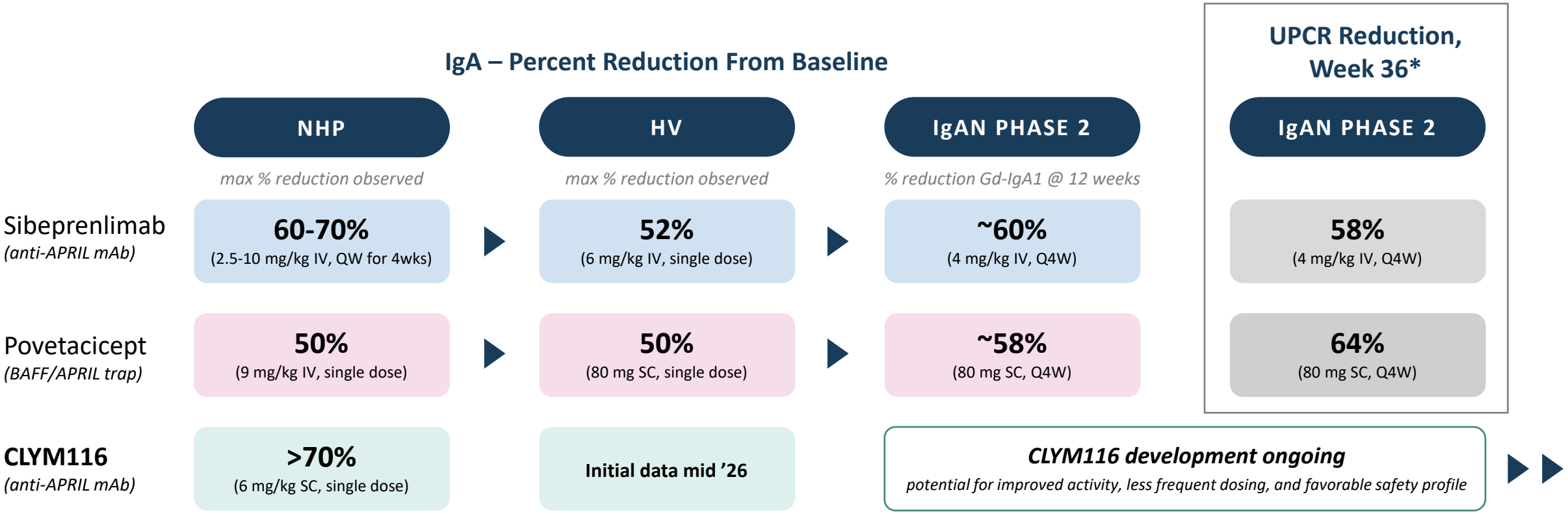


Table above reflects cross-study and cross-trial comparisons and not data from head-to-head studies; differences exist between trial designs and participant characteristics and caution should be exercised when comparing data across trials.

*Primary endpoint for accelerated approval.

Sibeprenlimab: Myette Kid Intl 2019, Mathur Kid Intl Reports 2022, Mathur NEJM 2024. Povetacicept: Evans Arthritis & Rheumatology 2023, Davies Clin Transl Sci 2024, Tumlin WCN 2024.

APRIL = a proliferation-inducing ligand, BAFF = B-cell activating factor, HV = healthy volunteers, IgAN = IgA nephropathy, IV = intravenous, mAb = monoclonal antibody, NHPs = nonhuman primates, QW = once weekly, Q4W = once every 4 weeks, SC = subcutaneous, UPCR = urine protein creatinine ratio, wks = weeks.

CLIMBBIO

CLYM116 Phase 1 Study In Healthy Volunteers Enrolling

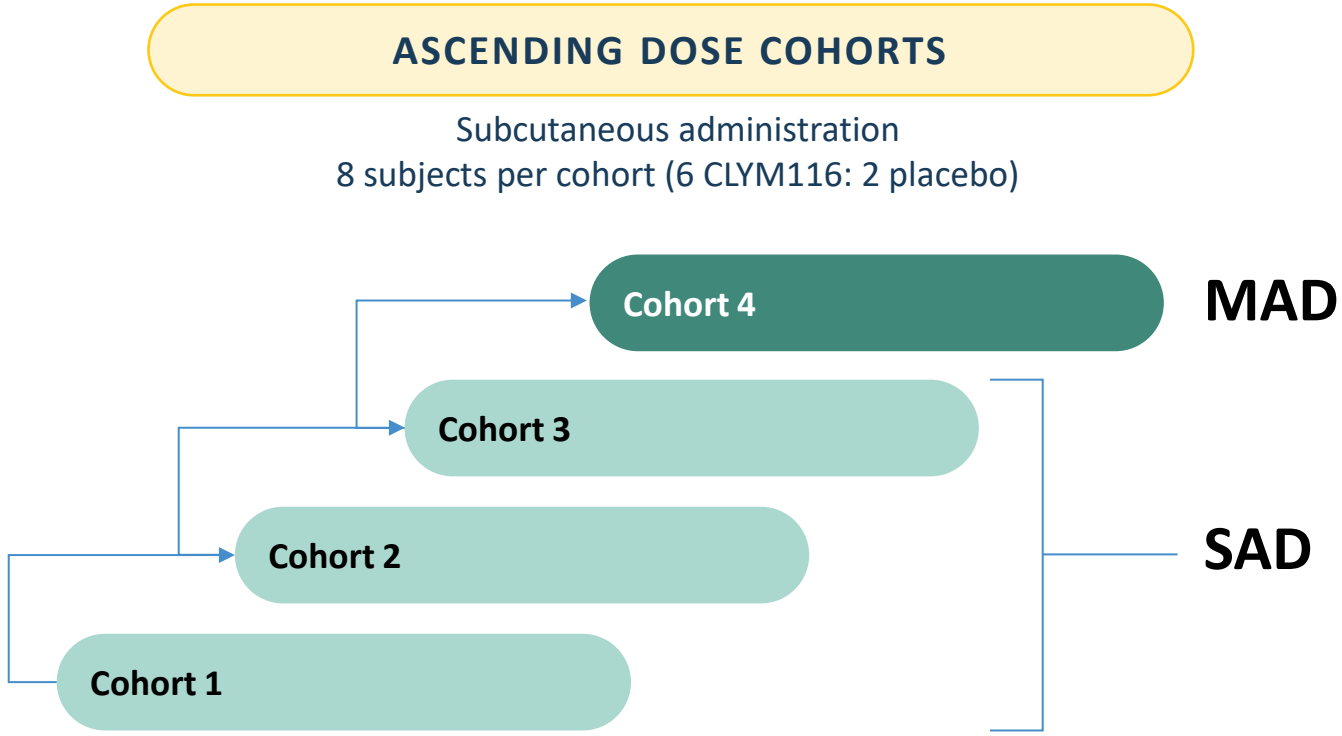
Pharmacodynamic biomarker data (APRIL, IgA) expected to guide dose and dose frequency for studies in IgAN patients; initial data expected mid-2026

Randomized, double-blind, placebo-controlled, ascending dose study

Population
- Healthy volunteers

Primary Objective
- Safety and tolerability

Secondary Objectives
- Pharmacokinetic profile
- Effect on immunoglobulins (IgA, IgM, and IgG) and APRIL levels (pharmacodynamic response)



(additional cohorts may be included as study progresses)

APRIL = a proliferation-inducing ligand, IgAN = IgA nephropathy, MAD = multiple ascending dose, SAD = single ascending dose

Looking ahead

Climb Bio is Poised for a Data-Rich 2026

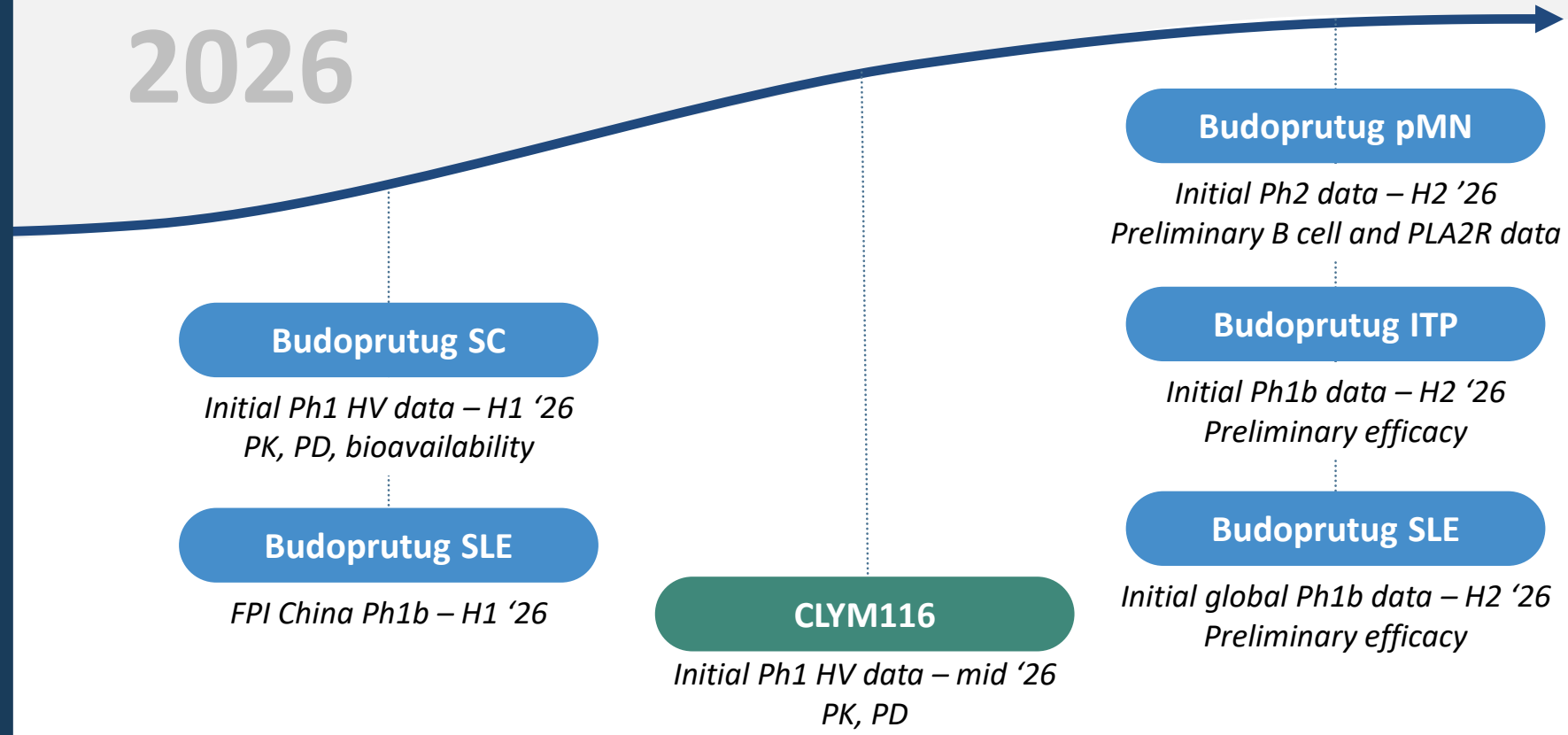
Continuing the ascent with initial readouts anticipated from all ongoing trials

$160.7M*

RUNWAY INTO 2028

68.2M†

FULLY DILUTED SHARES OUTSTANDING



*As of December 31, 2025

†47.8 million common shares outstanding; on December 11, 2025, RA Capital converted 20.4 million common shares to pre-funded warrants

ITP = Immune Thrombocytopenia; SLE = Systemic Lupus Erythematosus; pMN = primary membranous nephropathy; SC = subcutaneous; HV = healthy volunteers; PK = pharmacokinetics, PD = pharmacodynamics; FPI = first patient in

CLIMBBIO

Climb Bio is Well Positioned for Success

Multiple clinical readouts within cash runway

Developing **differentiated,** monoclonal antibody (mAb) therapeutics for **immune-mediated diseases**, including those affecting **kidney health**, with expansive commercial opportunities

Leveraging **clinically validated** B cell targets, **proven mAb modality**, and indications with **well-defined** endpoints and **established** regulatory pathways

Anticipating a **data-rich 2026** with **multiple clinical readouts** across both clinical-stage programs

- **Budoprutug -** anti-CD19 mAb in development for pMN, ITP, and SLE
- **CLYM116 -** anti-APRIL mAb in development for IgAN

Well-resourced to advance clinical programs through meaningful value-driving milestones, with **runway anticipated into 2028**

CLIMBBIO

pMN = primary membranous nephropathy; ITP = Immune Thrombocytopenia; SLE = Systemic Lupus Erythematosus; IgAN = IgA Nephropathy