Corporate Presentation

JULY 2026

CLIMBBIO

Forward Looking Statements

This presentation contains "forward-looking statements" 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 initiating and conducting Climb Bio's Phase 1b/2a clinical trial of budoprutug in systemic lupus erythematosus in China; the anticipated timelines for announcing data from Climb Bio's ongoing and planned clinical trials and Beijing Mabworks Biotech Co., Ltd.'s ("Mabworks") trial of CLYM116; the anticipated timelines for enrolling patients in Climb Bio's ongoing and planned clinical trials; plans for the development strategy for budoprutug and CLYM116; potential commercial opportunity for budoprutug in primary membranous nephropathy, immune thrombocytopenia, and systemic lupus erythematosus and for CLYM116 in IgA nephropathy; the sufficiency of Climb Bio's cash resources for the period anticipated; and other statements containing the words "anticipate," "believe," "continue," "could," "expect," "intend," "may," "plan," "potential," "should," "target," "would," "will," 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.

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In addition, Climb Bio has not conducted any head-to-head clinical studies comparing its product candidates to any third-party products or candidates, whether investigational or approved. Information regarding other drug products in this presentation is meant to provide context for illustrative purposes only. Because there are no head-to-head studies, no conclusions should be made based on cross-study comparisons. Recipients are cautioned not to place undue reliance on these forward-looking statements which speak only as of the date such statements are made and should not be construed as statements of fact.

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; Fast Track and Orphan Drug Designation granted for pMN
- **CLYM116 -** anti-APRIL mAb in development for IgAN

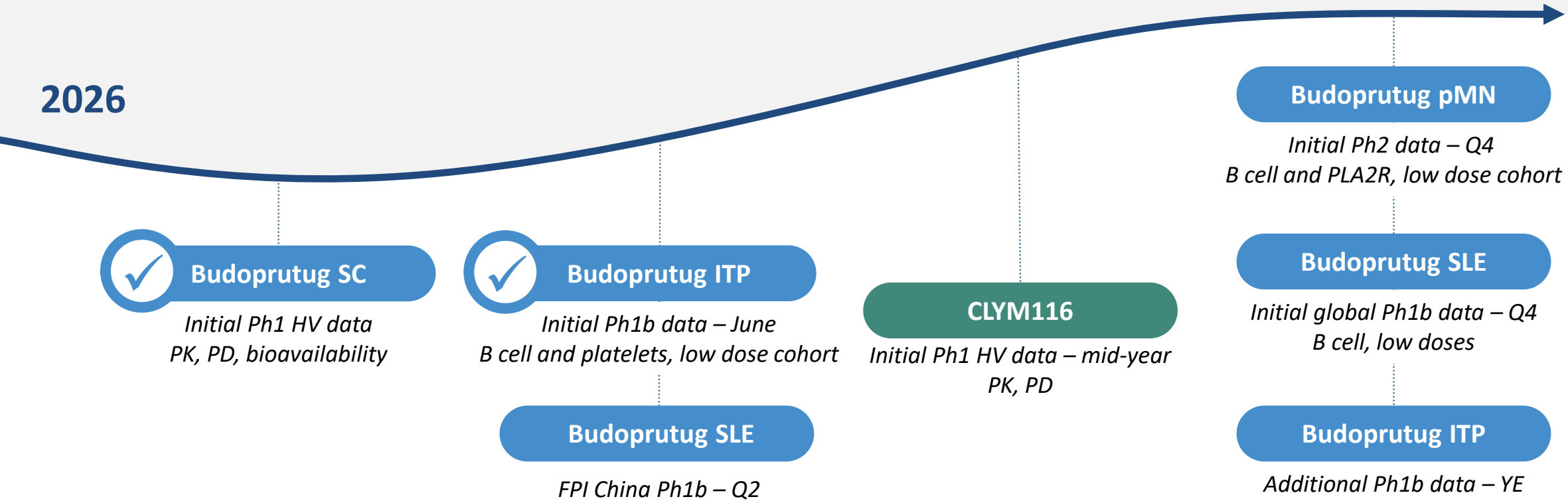
Well-resourced to advance clinical programs through meaningful value-driving milestones, with **runway anticipated into 2028***

*Excluding the proceeds received from the April 2026 private placement

pMN = primary membranous nephropathy, ITP = immune thrombocytopenia, SLE = systemic lupus erythematosus, IgAN = IgA nephropathy

Climb Bio: Data-Rich 2026

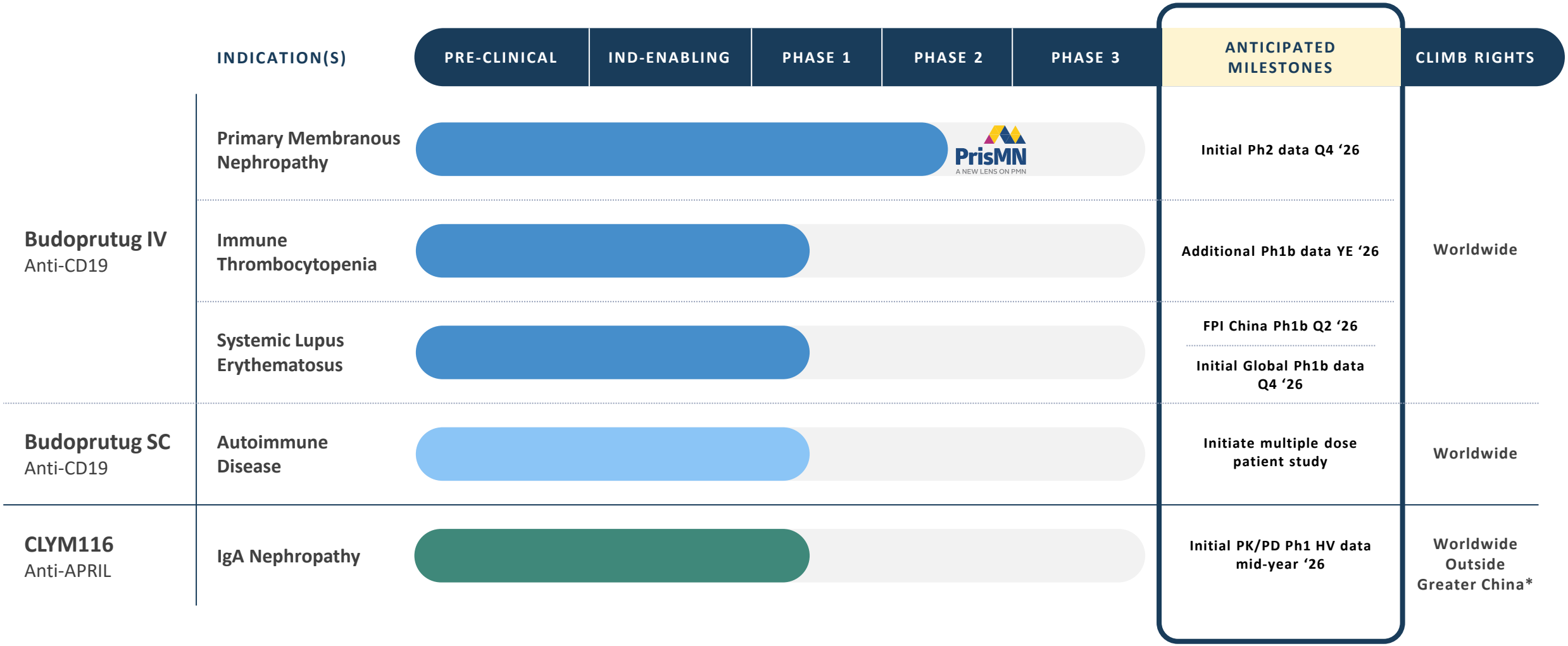
Continuing the ascent with initial readouts anticipated from all ongoing trials



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

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, HV = healthy volunteers

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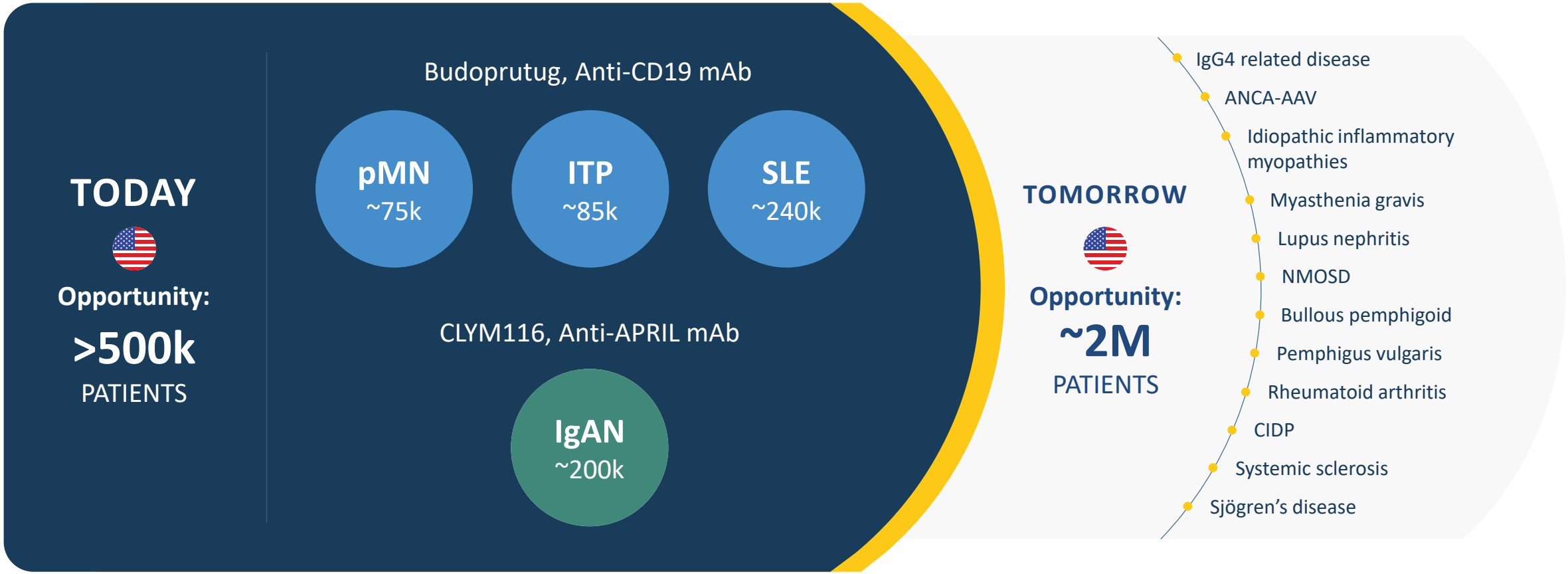
Pursuing Expansive Market Opportunities

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 (Feudjo-Tepie 2008, U.S. Census Estimates 2020-2025), pMN (McGrogan Nephrol Dial Transplant 2011, ~1 per 100,000; U.S. Census Estimates 2020-2025, assumes 28-year duration of pMN), SLE (Izmirly Arthritis Rheumatol 2021, U.S. Census Estimates 2020-2025).

Climb Bio Team Poised to Deliver for Patients

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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 may confer **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

- **Limited competition** in CD19 mAb space
- Administration profile intended to support **outpatient and community use**
- Potential to address **multiple B-cell mediated diseases**
- **Strong IP** protection extending to 2045+

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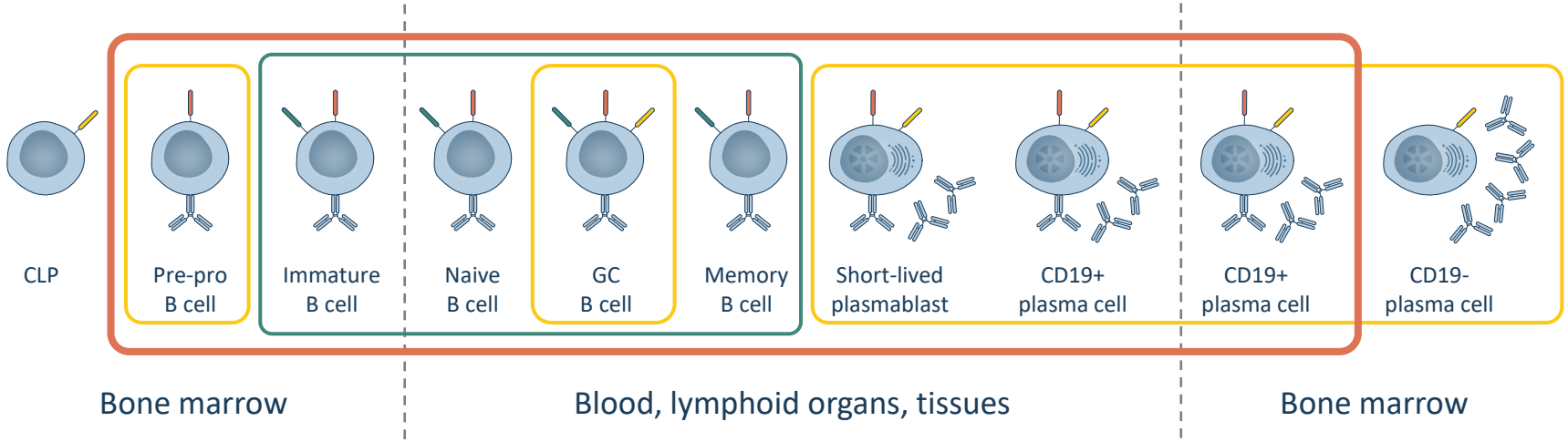
CRS = cytokine release syndrome, ICANS = immune effector cell associated neurotoxicity syndrome, IV = intravenous, mAb = monoclonal antibody, pM = picomolar, SC = subcutaneous

Cortazar ASN 2024

CD19 is Emerging as a Preferred Pan-B-Cell Target

Broad B-cell expression profile with potential for achieving deeper and more durable B-cell depletion while preserving protective antibody responses mediated by long-lived plasma cells

CD19 plays a mechanistic role across all stages of B-cell development, providing potential for profound and durable depletion of B cells and pathogenic autoantibodies[1]



- Naked mAbs targeting CD20 have been shown to deplete B cells in tissue[2]
- Deeper depletion may be achieved by targeting CD19

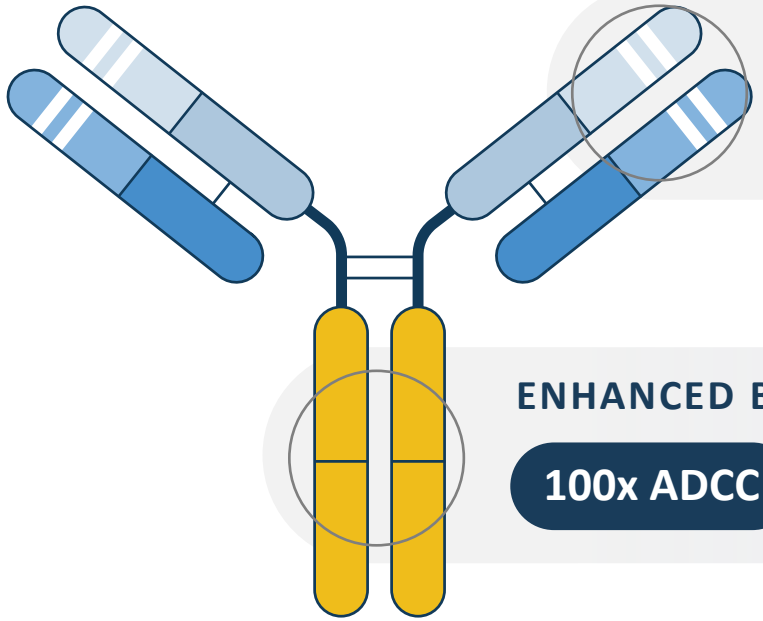
CLP = common lymphoid progenitor, GC = germinal center, mAbs = monoclonal antibodies

[1] Adapted from Suan J Immunol 2025, [2] Tur Annals Rheum Dis 2024

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 renal disease characterized by proteinuria, nephrotic syndrome and progressive loss of renal function.[5]	Chronic bleeding disorder characterized by the destruction of platelets.[6]	Chronic autoimmune condition with severe disease manifestations that can affect virtually any organ system.[7]
~75,000 patients[1,2]	**~85,000 patients**[2,3]	**~240,000 patients**[2,4]
No approved therapies	Poor QoL, with majority of previously treated patients failing to achieve durable platelet response[6]	Majority of patients fail to achieve disease control with existing treatments[7]
BUDOPRUTUG OPPORTUNITY		
Potential for long-term disease remission based on initial clinical data; Fast Track Designation granted	Potential to achieve durable response and disease remission in the previously treated population	Potential for broad B-cell targeting and disease suppression with the safety and convenience of a mAb

Ongoing Studies Designed to Answer Key Clinical Questions

Ability to achieve deep B-cell depletion • Optimal dose in renal and non-renal indications • Potential for long-term disease control

mAb = monoclonal antibody, QoL = quality of life

[1] McGrogan Nephrol Dial Transplant 2011, [2] U.S. Census Estimates 2020-2025, [3] Feudjo-Tepie 2008, [4] Izmirly Arthritis Rheumatol 2021, [5] Ronco JCM 2021, [6] Gafter-Gvili Eur J Int Med 2023, [7] Marinho Front Immunol 2023

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Primary Membranous Nephropathy

Seferiana, living with pMN

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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**, but **only 35%** of patients achieve a **complete remission (CR)** by year 2
- **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

~75,000 patients
in the US alone

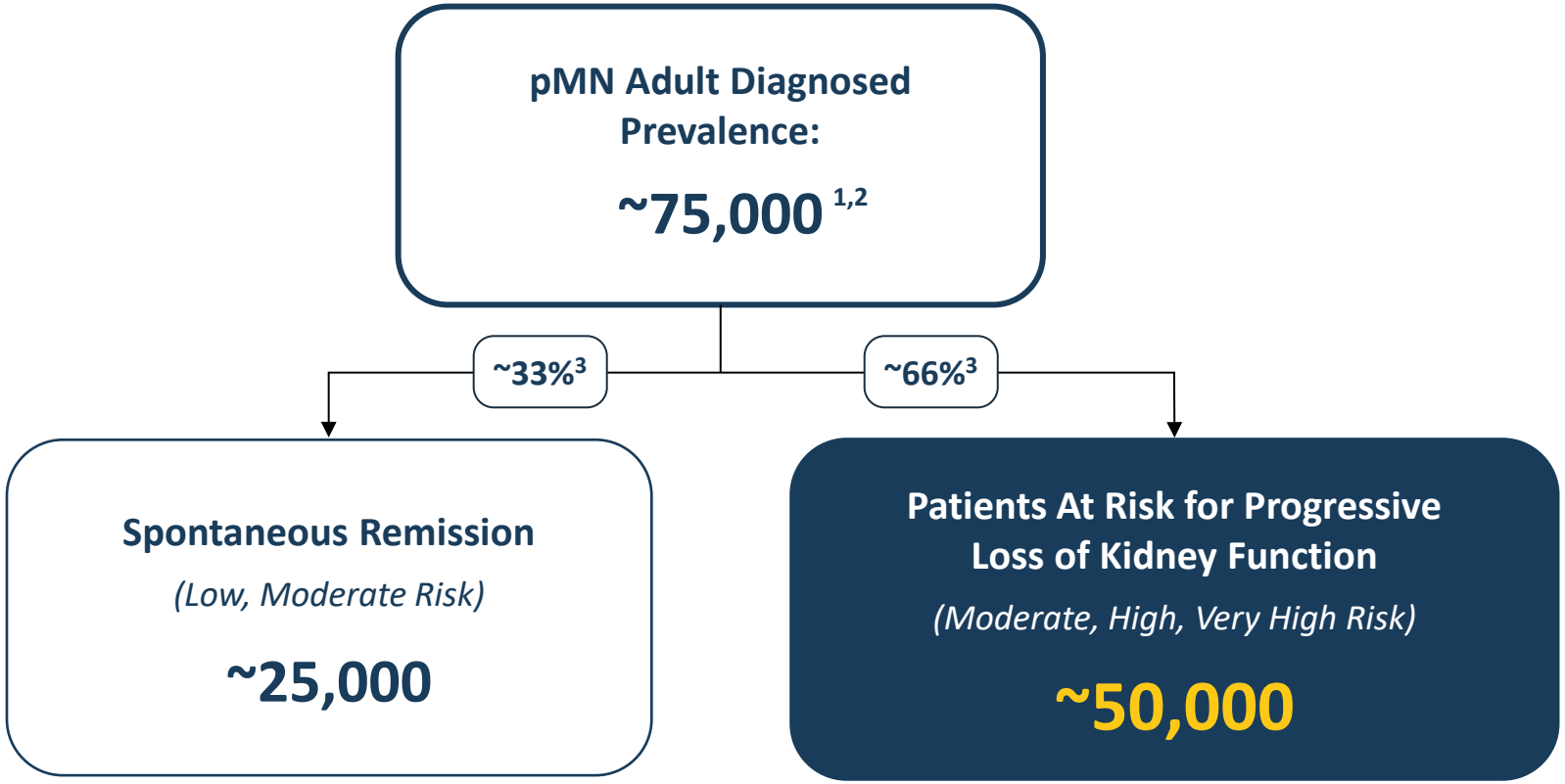
THE CLIMB SOLUTION

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

pMN = primary membranous nephropathy; Couser CJASN 2017, Ronco J Clin Med 2021, Teisseyre Front Immunol 2022, KDIGO 2021 guidelines
US Prevalence: McGrogan Nephrol Dial Transplant 2011, ~1 per 100,000; U.S. Census Estimates 2020-2025, assumes 28-year duration of pMN

Large Addressable Patient Population in pMN

Majority of patients who do not achieve spontaneous remission will require treatment to prevent risk of progressive loss of kidney function



KDIGO Recommendation[4]

Immunosuppressive therapy should be considered in patients:

- with elevated anti-PLA2R and proteinuria >3.5 g/d at diagnosis
- for those who fail to reduce proteinuria <3.5 g after 6 months of supportive care

PLA2R = phospholipase A2 receptor, pMN = primary membranous nephropathy

[1] McGrogan Nephrol Dial Transplant 2011; ~1 per 100,000, [2] U.S. Census Estimates 2020-2025; assumes 28-year duration of pMN, [3] Crouser CJASN 2017, [4] KDIGO 2021 Guideline for the Management of Glomerular Diseases

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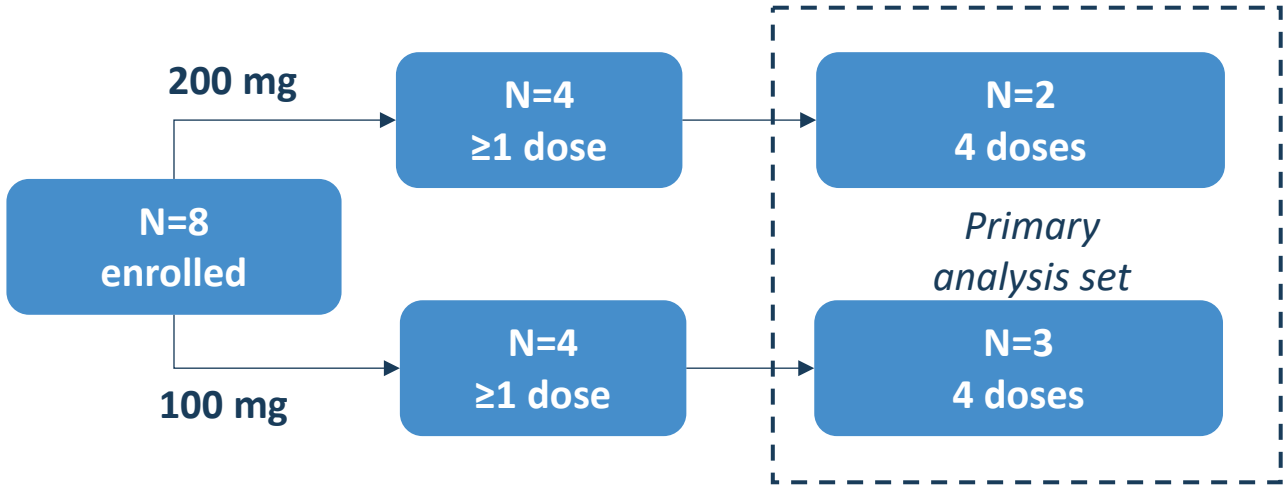
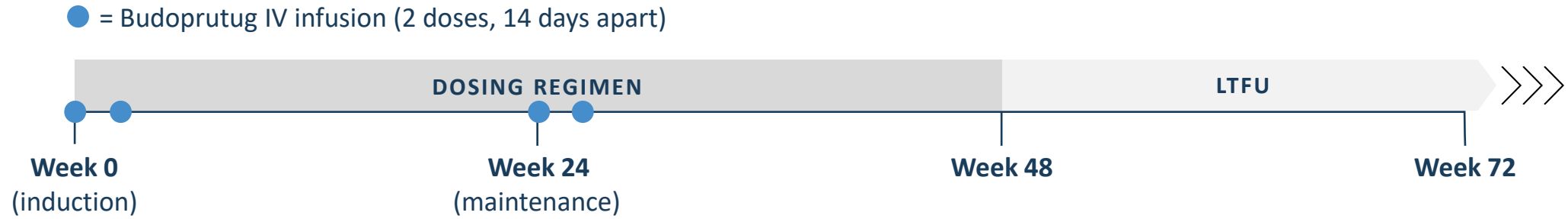
Completed Phase 1b Study Established Proof-of-Concept in pMN

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

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



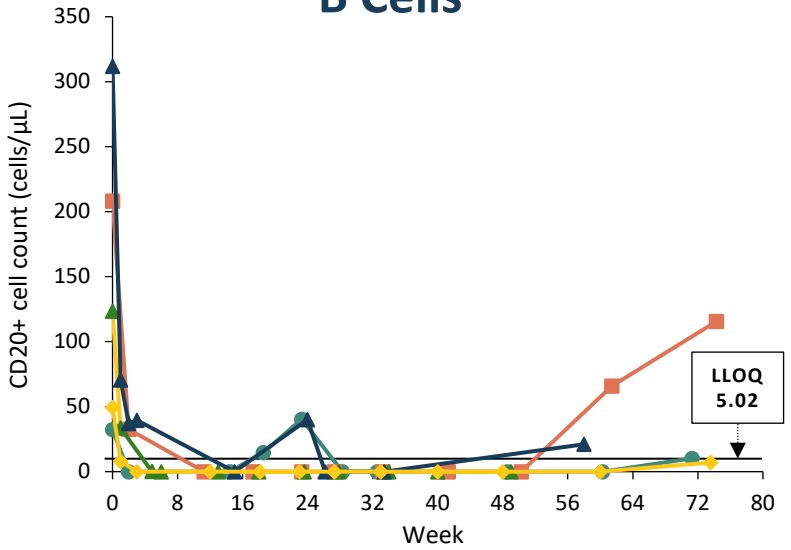


NCT04652570; IV = intravenous, LTFU = long term follow-up, PLA2R = phospholipase A2 receptor, pMN = primary membranous nephropathy, UPCR = urine protein creatinine ratio
Cortazar ASN 2024; Baseline characteristics for primary analysis set (N=5), PLA2R from patients with positive anti-PLA2R antibodies at baseline (N=3)

Clinical Proof-of-Concept Demonstrated in Pilot Study in pMN

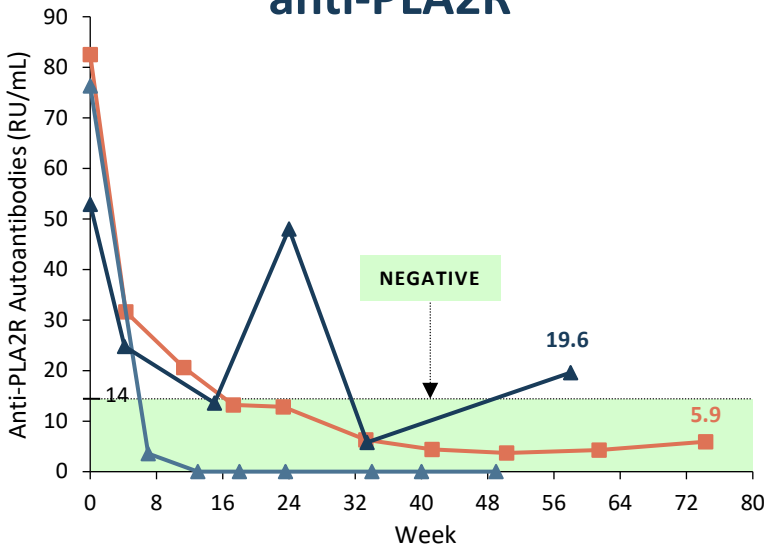
Budoprutug administration resulted in durable B-cell depletion, serologic remission, and clinical remission (as measured by proteinuria)

B Cells[1]



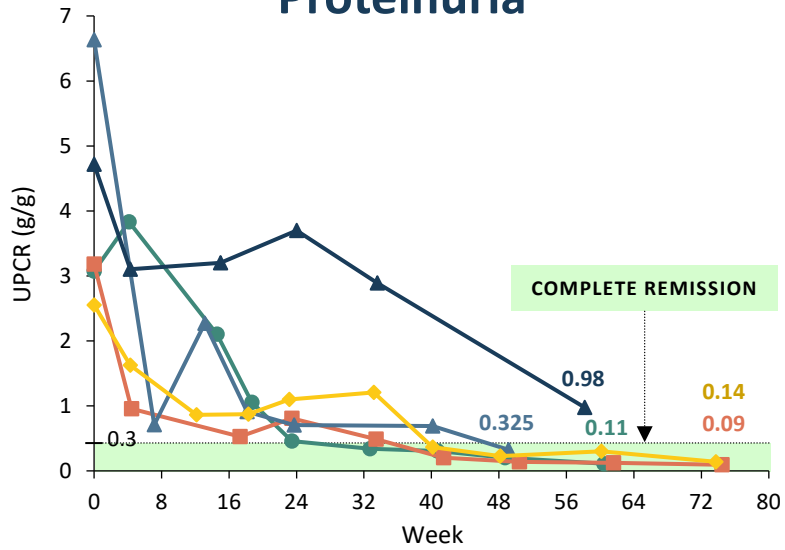
- **Rapid and complete** circulating **B cell depletion** observed in **100% (5/5) patients** at doses of 100-200 mg
- **Sustained reductions** out to 1 year + after two dose cycles

anti-PLA2R[1]



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

Proteinuria[1]



- All patients achieved **complete (3/5) or partial (2/5) clinical remission** by Week 48
- **Long-term control** up to 3 years after initial dosing observed in 4 patients who received up to 4 doses[2]

LLOQ = lower limit of quantification, PLA2R = phospholipase A2 receptor, pMN = primary membranous nephropathy, UPCR = urine protein creatinine ratio

[1] Cortazar ASN 2024, [2] Cortazar ASN 2025; Phase 1b pMN: budoprutug (100-200 mg) administered in two IV infusions, 14 days apart, at Wk 0 and Wk 24

Safety

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

8 patients received at least one infusion 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 ; Phase 1b pMN: budoprutug (100-200 mg) administered in two IV infusions, 14 days apart, at Wk 0 and Wk 24

Budoprutug Has a Highly Compelling 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[1])	**Rituximab** (MENTOR study[2])	**Obinutuzimab** (MAJESTY Ph3 study[3])	**Povetacicept** (RUBY-3 Ph1/2 study[4])	**Felzartamab** (Phase 1b/2a[5])
Target	CD19	CD20	CD20	BAFF/APRIL	CD38
Serologic remission (anti-PLA2R negativity)	✓ **100%** (3/3) *48wks*	**64-95%** *(titer decrease), 6-24mo*	**70.4 %** (38/54) *104wks*	**100%** (4/4) *48wks*	**23%** (6/26) *6mo*
Complete or partial clinical remission	✓ **100%** (5/5) *48wks*	**60%** (39/65) *24mo*	**50.8%** (36/71) *104wks*	100% (5/5) *48wks*	**35%** (9/26) *12mo*
Complete remission	✓ **60%** (3/5) *UPCR ≤0.3 g/g, 48wks*	**14%, 35%** (9/65, 23/65) *UPCR ≤0.3 g/g; 12mo, 24mo*	**24%, 37%** (17/72, 26/72) *UPCR ≤0.3 g/g; 52 wks, 104wks*	40% (2/5) *UPCR <0.5 g/g; 48wks*	**0%** (0/26) *UPCR <0.5 g/g; 12mo*
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

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IV = intravenous, PLA2R = phospholipase A2 receptor, pMN = primary membranous nephropathy, q6m = every 6 months, SC = subcutaneous; 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 and felzartamab, 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 NEJM 2026, [4] Madan KI Reports 2025, [5] Rovin KI Reports 2024

PrisMN: Budoprutug Phase 2 pMN Study Enrolling

PrisMN
A NEW LENS ON PMN

Biomarker endpoint provides potential to rapidly identify dose to carry forward into Phase 3; initial B cell and PLA2R data from low dose cohort anticipated in Q4 2026

Open-label, dose ranging study

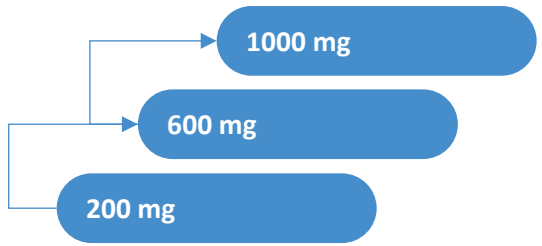
Population
- 18-70 years of age
- UPCR ≥ 2.0 g/g; designed to ensure adequate enrollment of patients with UPCR > 5.0 g/g
- PLA2R antibody positive

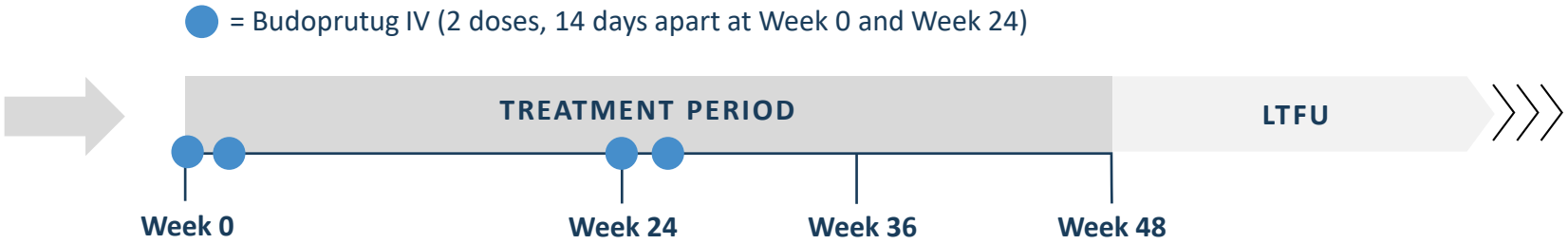
Primary Objective
- Safety and tolerability

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





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NCT07096843; 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

John, living with ITP

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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

- **Guidelines recommend** corticosteroids as 1L treatment, TPO-RAs, rituximab, or splenectomy as 2L, however **many experience treatment failure**
- **Only ~20%** of previously-treated patients achieve **durable platelet response** with available 3L therapies (BTK, SYK inhibitors)

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

ITP = immune thrombocytopenia; 1L = first line; 2L = second line; 3L = third line
US Prevalence: Feudjo-Tepie 2008; U.S. Census Estimates 2020-2025
StatPearls Pietras 2024, Gafter-Gvili A Euro J Int Med 2023, Lucchini Haematologica 2019, Internal Research

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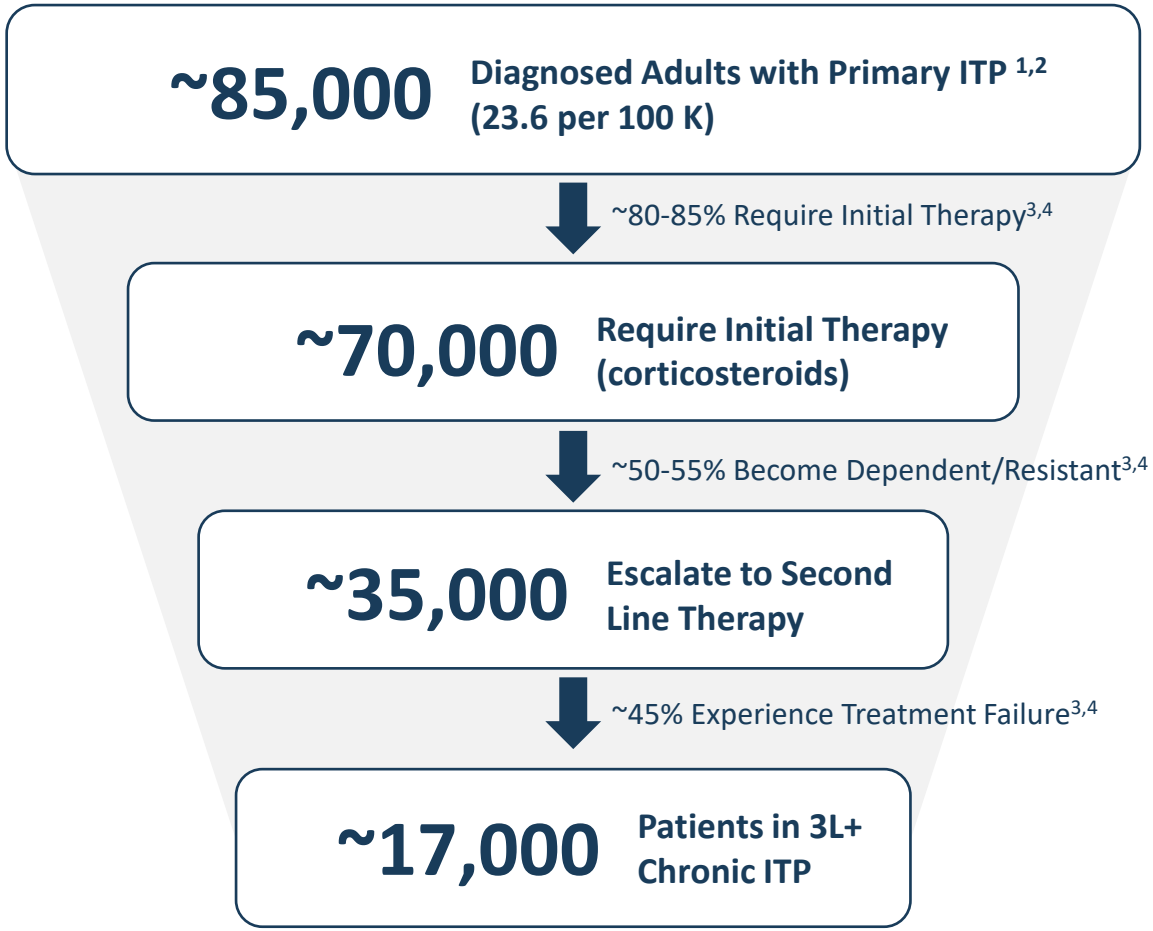
Significant Opportunity for a Disease Modifying Approach in ITP

Chronic ITP patients often cycle through multiple therapies to maintain platelet control



40-50%
of patients require chronic therapy

~20%
progress to 3L+ disease, representing the highest unmet need

Later-line ITP represents a high-value opportunity for disease-modifying therapies

3L = third line, ITP = immune thrombocytopenia

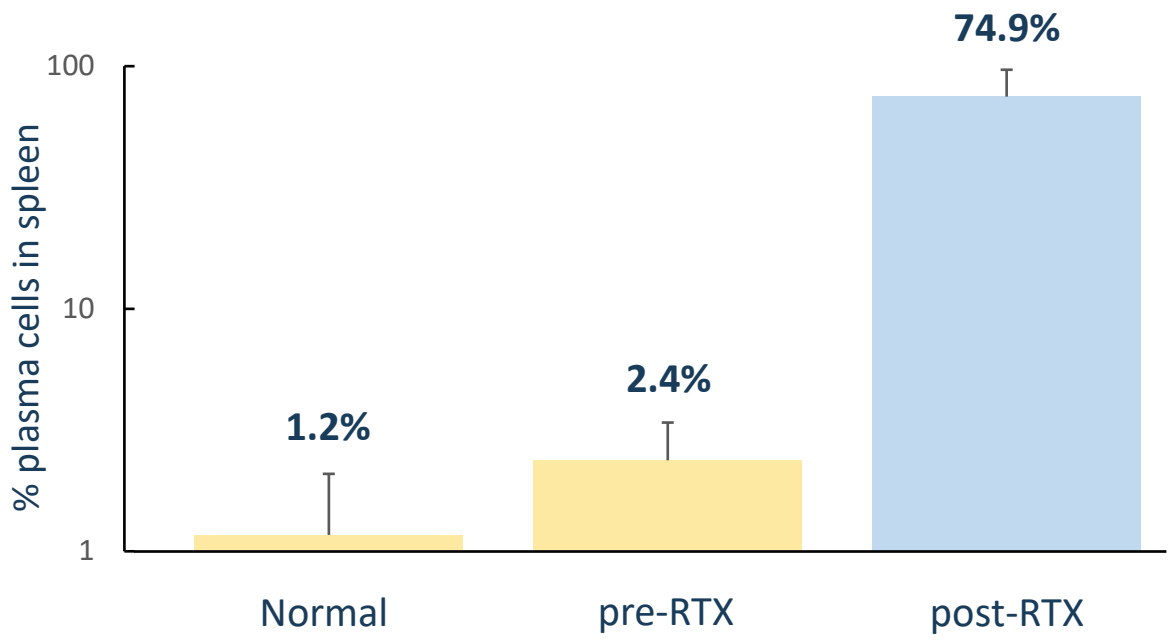
[1] Feudjo-Tepie 2008, [2] U.S. Census Estimates 2020-2025,[3] Moulis Haematol 2025, [4] Climb Market Research 2026

Strong Rationale for CD19 Approach in ITP

Potent anti-CD19 approach offers the promise of sustained elimination of pathogenic B cells

- 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**[1-3]
- **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 certain plasma cells**[2]

$CD19^+/CD20^-$ plasma cells expand within B cell niches post anti-CD20 treatment[1]



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

ITP = immune thrombocytopenia, mAbs = monoclonal antibodies, RTX = rituximab

[1]Adapted from Mahevas J Clin Invest 2013, [2]Crickx Kidney Intl 2020, [3]Lucchini Haematologica 2019

Budoprutug Phase 1b/2a ITP Trial Ongoing

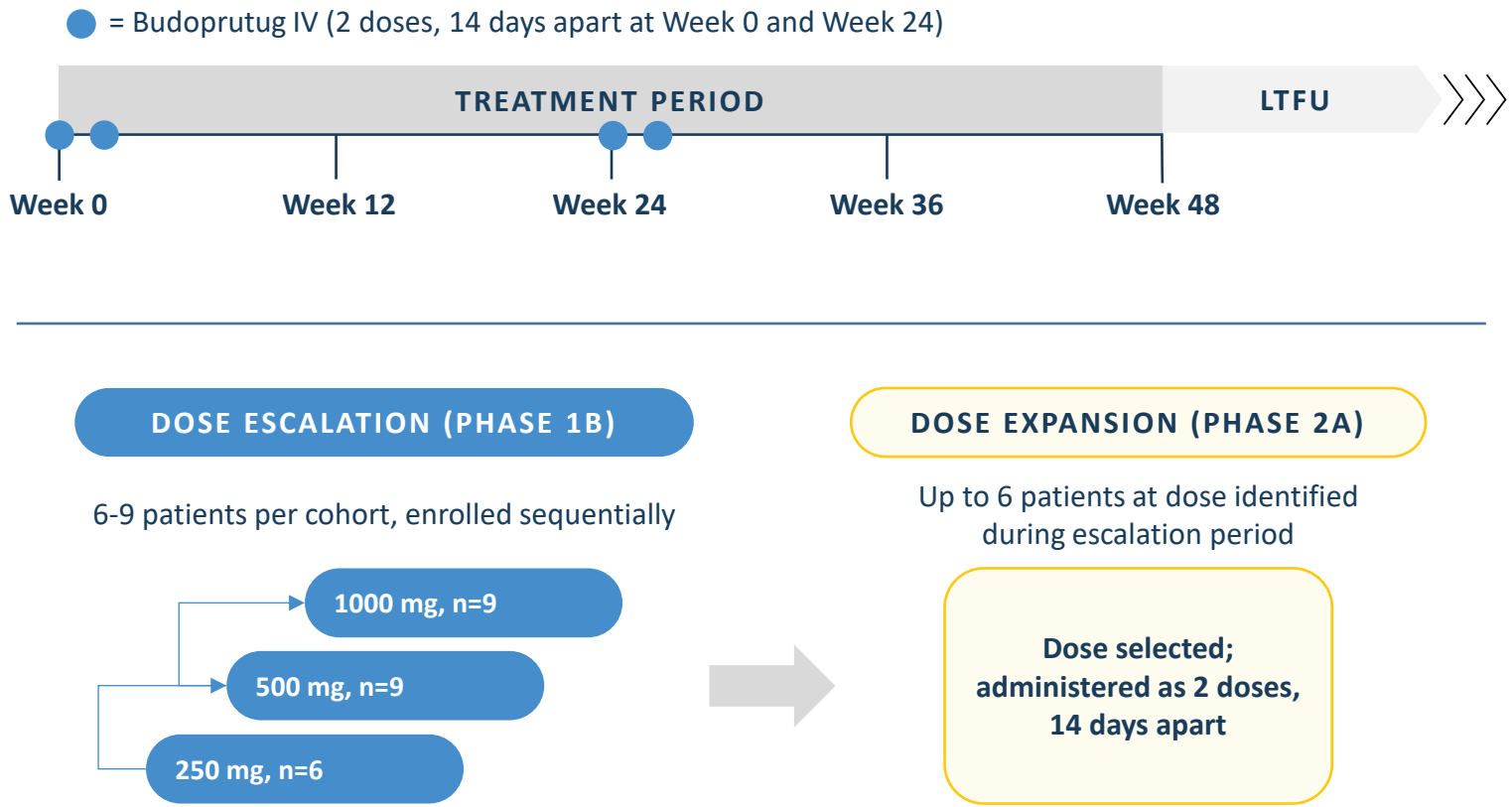
Designed to define dose and regimen, and establish depth and duration of platelet response and B cell depletion

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)



NCT07043946; ITP = immune thrombocytopenia, LTFU = long term follow-up, LLN = lower limit of normal

ITP Phase 1b/2a: Patient Demographics and Initial Safety Data

Budoprutug was generally well tolerated in the 250 mg and 500 mg cohorts which enrolled heavily pretreated patients with persistent or chronic ITP; enrollment of 1000 mg cohort ongoing

	250 mg (N=6)	500 mg (N=9)
Age (median, range)	42 (22-75)	41 (18-58)
Gender (M/F)	2/4	5/4
Time since diagnosis (median, range)	6.5 years (0.5-12)	15 years (0.6-40)
Prior lines of therapy (median, range)	7.5 (4-18)	6 (2-15)
Prior therapy:		
Corticosteroids (n)	6	9
IVIg (n)	2	5
TPO-RA (n)	4	7
Rituximab (n)	4	2
Splenectomy (n)	0	1
Baseline:		
Platelet count x10^3/µL (median, range)	13 (4-29)	7 (2.5-12)
CD19$^+$ B-cells/µL (median, range)	117.5 (87-287)	156 (82-343)

Median of **6 to 7.5** prior lines of therapy • Disease duration ranging from **0.5 to 40 years** • **6 of 15 (40%)** patients had prior rituximab exposure

Safety

Budoprutug was generally well tolerated in the ongoing Phase 1b/2a ITP trial at doses of 250 mg and 500 mg

- ✓ No SAEs or deaths reported
- ✓ All AEs were Grade 1 or Grade 2; majority of AEs belonged to the gastrointestinal system organ class
- ✓ No discontinuations due to AEs
- ✓ No infusion-related reactions
- ✓ No participants had hypogammaglobulinemia

*15 patients received two infusions of budoprutug and were included in the safety analysis population in the initial Phase 1b data cut**

AE = adverse event, ITP = immune thrombocytopenia, SAE = serious adverse event, TPO-RA = thrombopoietin receptor agonist

**Median (range) follow-up for participants in Cohort 1 and 2: 38 weeks (28-44), and 12 weeks (3-20), respectively*

Kazi EHA 2026; Phase 1b/2a ITP: budoprutug administered in two IV infusions, 14 days apart. Data as of June 1, 2026.

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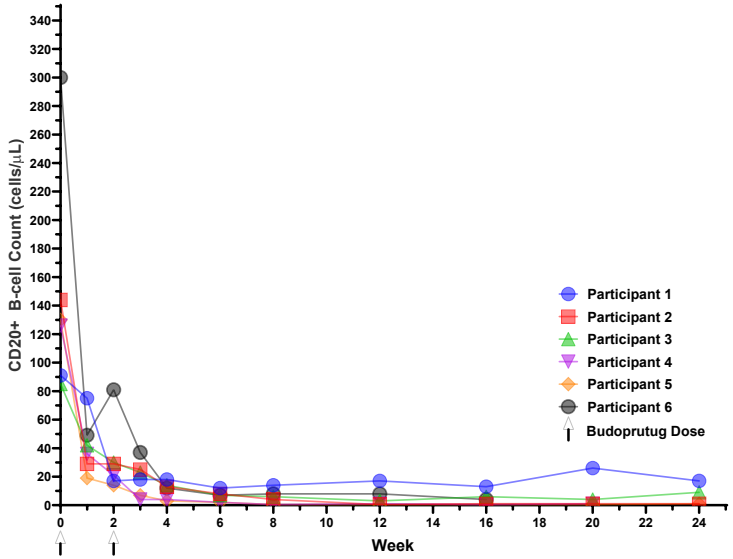
Clinical Proof-of-Concept Demonstrated in ITP Phase 1b Cohort 1

Initial data from ongoing Phase 1b demonstrated durable B-cell depletion and platelet response following administration of budoprutug (low dose, 250 mg)

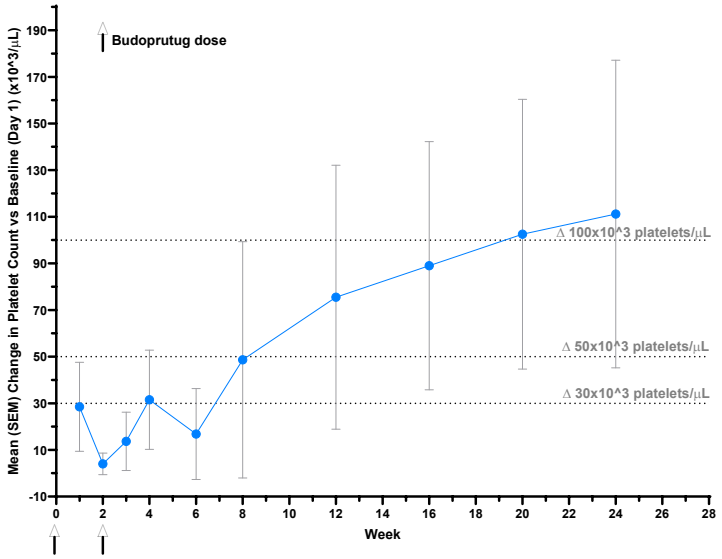
B-Cell Depletion



High sensitivity assay, LLOQ of 0.4 cells/µL

Platelet Response Assessments

At **Week 24**, mean (SEM) increase in platelet count from baseline was **111 (66) x 10^3 platelets/µL**



Participants continue to be followed

- **>90%** mean B-cell depletion at **Week 4**
- **66% (4/6)** patients achieved a **durable platelet response**
- **33% (2/6)** patients achieved a **complete platelet response**
  - **Participants with complete response maintained platelet levels ≥100 x 10^3/µL for ≥24 weeks**; both previously relapsed on rituximab
- **75% (3/4) participants who had prior rituximab responded**, two of whom had durable complete responses

ITP = immune thrombocytopenia; LLOQ = lower limit of quantification; BL = baseline

Durable Response: ≥30 × 10^3/µL and ≥2x BL at 6 months; Complete Response: ≥100 × 10^3/ µL and absence of bleeding; Responses require confirmation on two separate occasions at least 7 days apart through Week 24.

Kazi EHA 2026; Phase 1b/2a ITP: budoprutug administered in two IV infusions, 14 days apart. Data as of June 1, 2026: B-cell and platelet counts presented up to Week 24, last date with data point for all 6 participants.

CLIMBBIO

Systemic Lupus Erythematosus

Marisa, living with SLE

CLIMBBIO

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; U.S. Census Estimates 2020-2025, 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[1], 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

CLIMBBIO

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

[1] belimumab prescribing information, [2] obinutuzumab prescribing information, [3] Scherlinger Annals Rheumatic Diseases 2019, [4] Müller NEJM 2024

Parallel Global and China SLE Studies

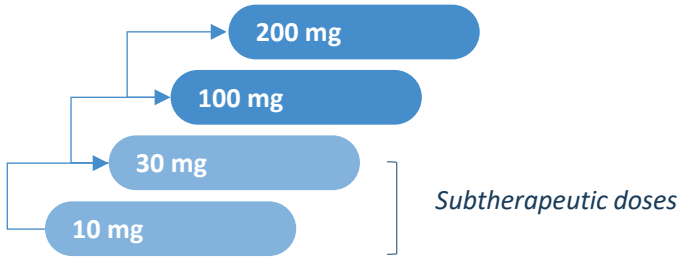
Designed to assess safety, tolerability, B-cell depletion, and kinetics of B-cell subset repopulation

Global Phase 1b Study

Enrolling adults with seropositive SLE; SLEDAI ≥ 8; refractory to adequate trials of at least 2 therapies

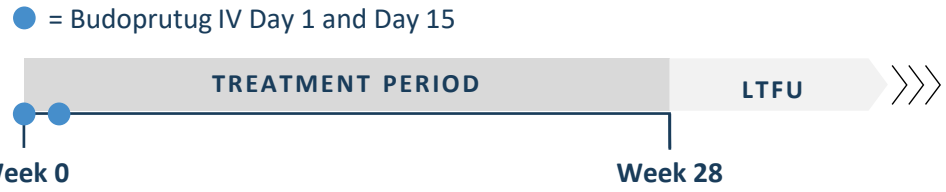
SINGLE ASCENDING DOSE COHORTS, N ~30

Up to 6 patients per cohort enrolled sequentially



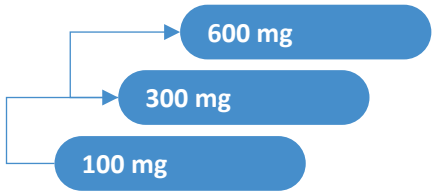
China Phase 1b/2a Study

Enrolling adults with seropositive SLE; SLEDAI ≥ 6; refractory to adequate trial of at least 1 therapy



DOSE ESCALATION, N ~18

Up to 6 patients per cohort enrolled sequentially



DOSE EXPANSION, N ~12

Up to 12 patients at dose identified in escalation period

Dose selected; administered as 2 doses, 14 days apart

Initial data from low dose cohorts in the Global Study anticipated in Q4 2026, with FPI in China Study on track for Q2 2026

Global study: NCT07011043 China study: NCT07564596

FPI = first patient in, IV = intravenous, LTFU = long term follow-up, SLE = systemic lupus erythematosus, SLEDAI = SLE disease activity index

Budoprutug – Subcutaneous (SC) Formulation

Anti-CD19

Budoprutug SC Formulation Provides Optionality and Differentiation

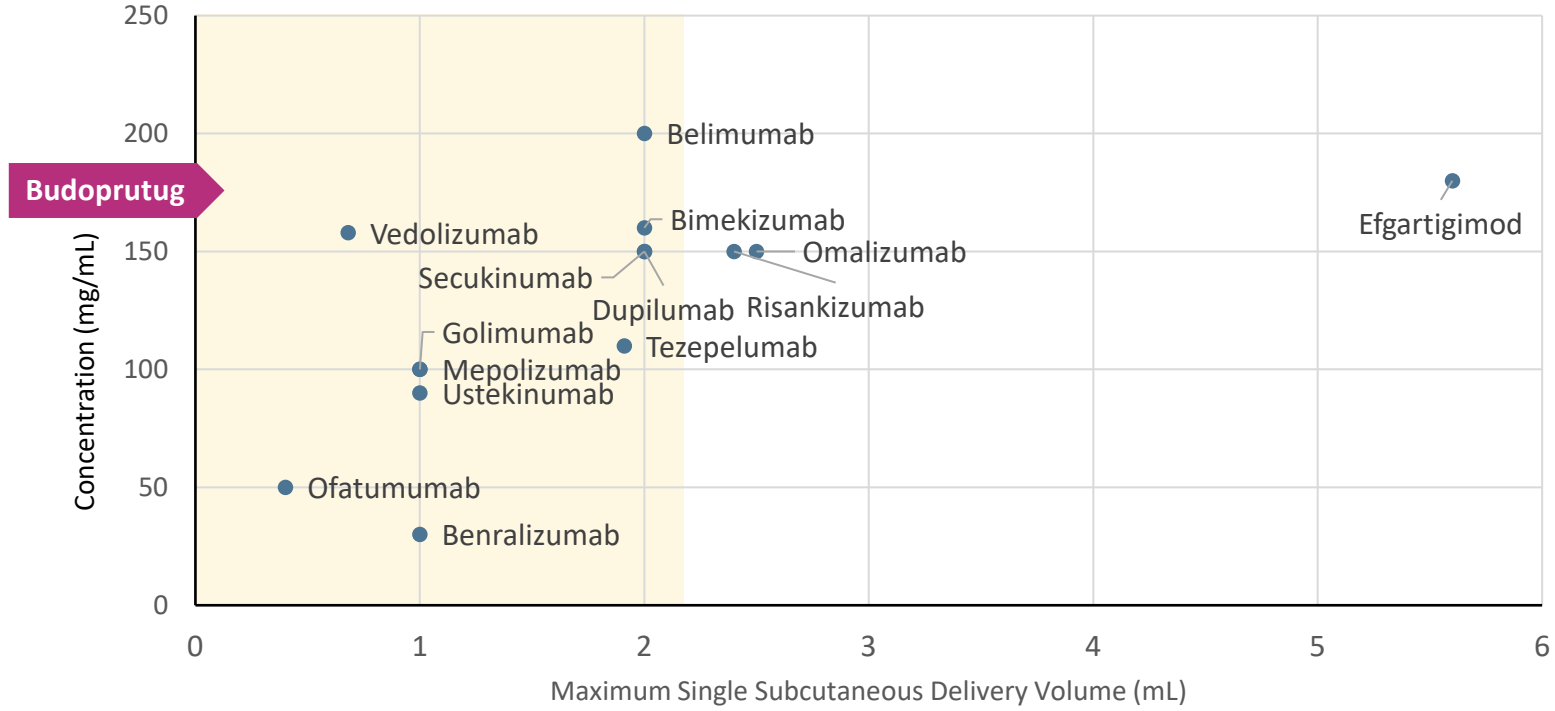
Ability to formulate as both IV and SC provides optionality in development and may be a differentiating feature of budoprutug relative to other anti-CD19 mAbs

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

Potential to deliver 350 mg of budoprutug in a single, 2 mL SC injection

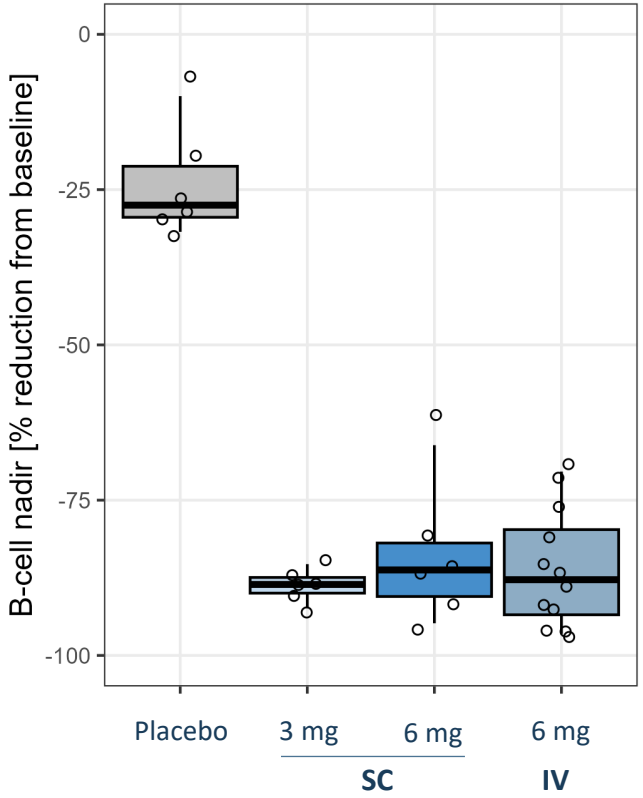


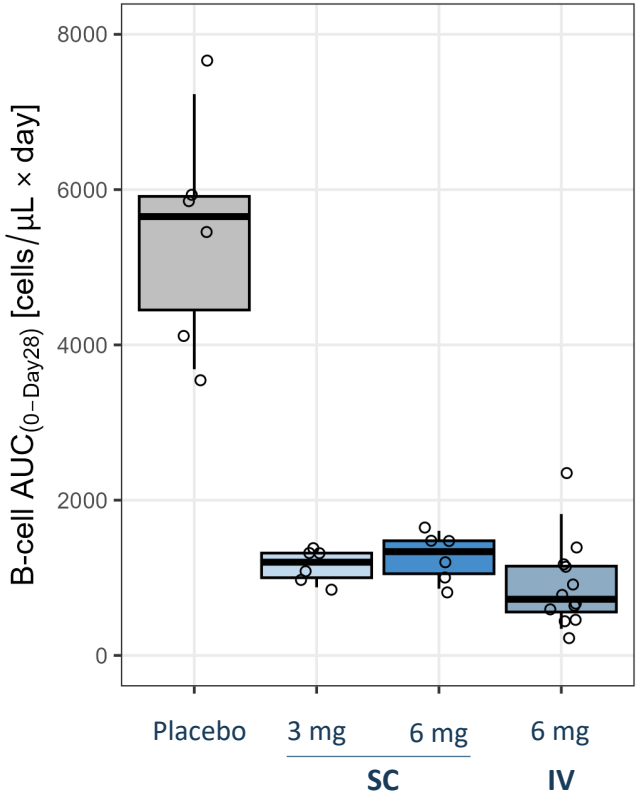
IV = intravenous, mAbs = monoclonal antibodies, SC = subcutaneous

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

Robust B-cell Depletion Observed with Budoprutug SC in HV

Budoprutug SC administration demonstrated ~80% B-cell depletion; similar magnitude and time course of depletion between SC and IV



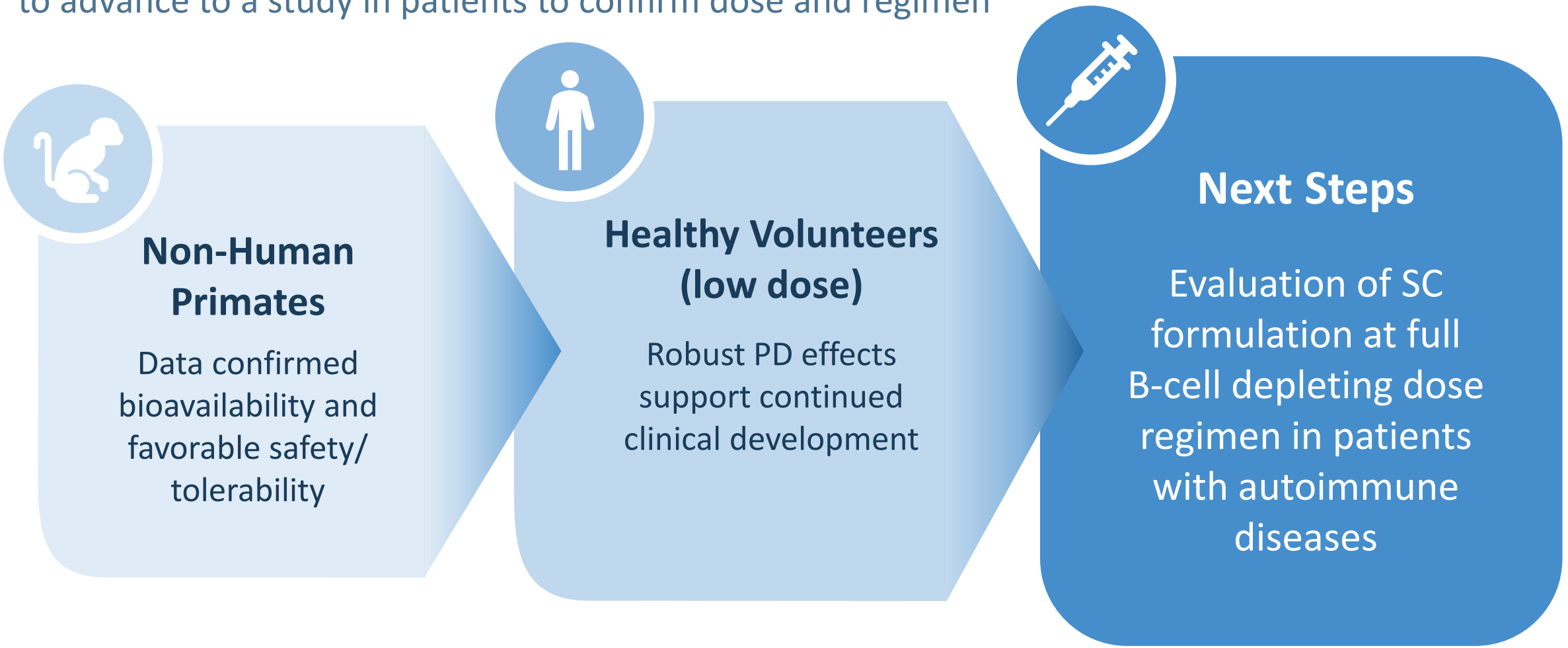


Budoprutug SC was well-tolerated; safety profile consistent between SC and IV

AUC = area under the curve, HV = healthy volunteers, IV = intravenous, SC = subcutaneous
Company data on file from Phase 1 placebo-controlled, single ascending dose study in HV (NCT07090655); 3 cohorts: SC 3 mg, SC 6 mg, IV 6 mg, with 8 subjects per SC cohort (6 budoprutug: 2 placebo) and 14 subjects in IV cohort (12 budoprutug: 2 placebo). Low doses of budoprutug were chosen to achieve non-complete (~50%) peripheral B cell depletion in HV.

Budoprutug SC: Future Direction

Data in NHPs and healthy volunteers support continued development of SC formulation, with plans to advance to a study in patients to confirm dose and regimen



NHPs = nonhuman primates, PD = pharmacodynamic, SC = subcutaneous

CLYM116

Anti-APRIL mAb

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

Initial PK/PD data in healthy volunteers 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 & immunogenicity profile

- Phase 1 clinical trial in HV ongoing, with initial PK/PD data expected late summer
- Mabworks Phase 1 in HV ongoing; Phase 2 portion in IgAN patients expected to initiate Q3*

*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
- US 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;** with potential for a further streamlined path based on recent NKF/FDA workshop[6]
- **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] KDIGO 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. [6] Vera press release June 2, 2026

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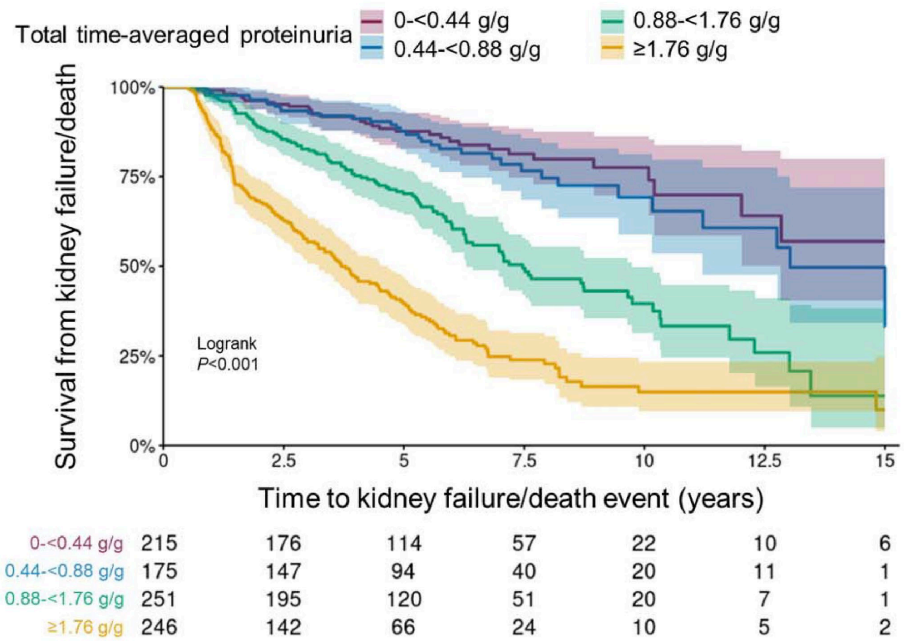
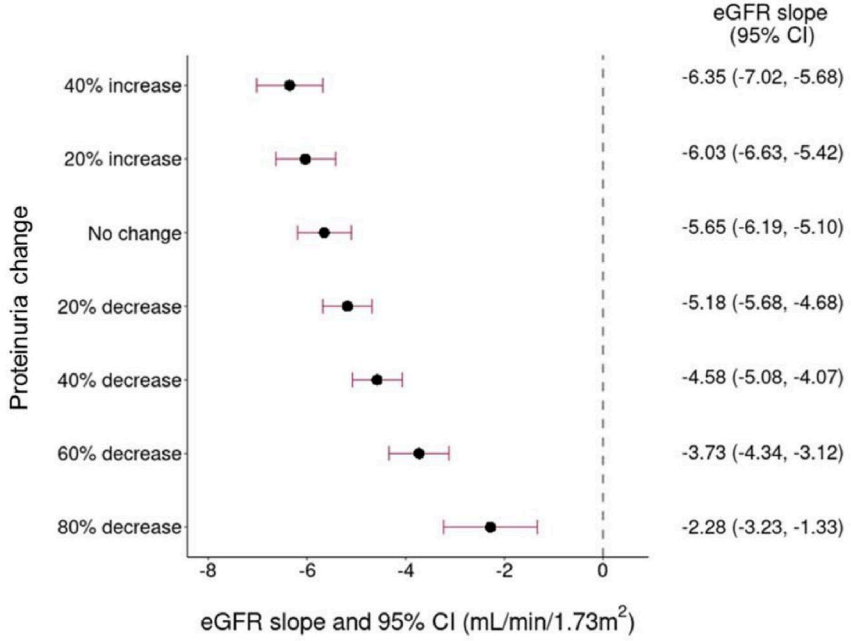
Magnitude of Proteinuria Predicts Development of Kidney Failure

Proteinuria improvement correlates with long-term kidney function stabilization

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





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

Pitcher CJASN 2023

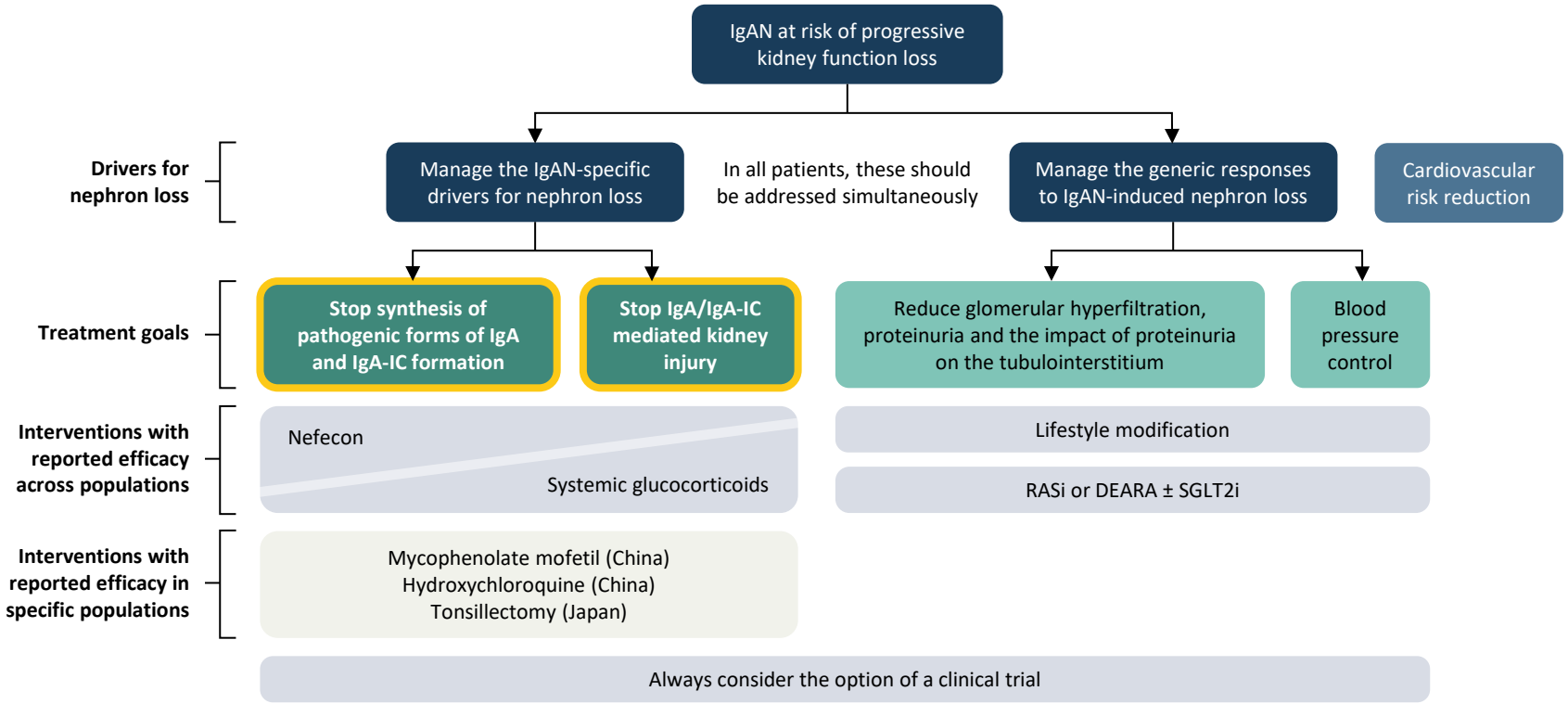
KDIGO 2025 Guideline Highlights Role for IgA Reduction in IgAN

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

KDIGO 2025 Clinical Practice Guideline for the Management of IgAN

Selective APRIL Inhibition Has Been Clinically Validated in IgAN

In Phase 3 IgAN studies, anti-APRIL mAb, sibeprenlimab shows numerically better or similar proteinuria (UPCR) reductions compared to anti-BAFF/APRIL antagonists, atacicept and povetacicept

	Sibeprenlimab[1] (anti-APRIL mAb)	**Atacicept[2]** (TACI-IgG Fc)	**Povetacicept[3]** (TACI-Fc Fusion)
Dose	400 mg SC, Q4W	150 mg SC, QW	80 mg SC, Q4W
N	320*	203**	199±
UPCR change at 9 months	**-50.2%** vs. +2.1% for placebo	-46% vs. +7% for placebo	-52.0% vs. -4.3% for placebo
UPCR reduction at 9 months (placebo-adjusted)	**51.2%** p<0.0001	**42%** p<0.0001	**49.8%** 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.

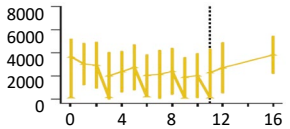
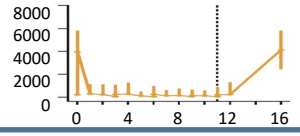
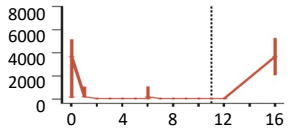
*Interim analysis of 320 patients, total of 530 enrolled; **Interim analysis of 203 patients, total of 431 enrolled; ±Interim analysis of 199 patients, total of 605 enrolled.
[1] Perkovic ERA 2025, 40 week endpoint, [2]Vera Corporate Presentation, June 2025, 36 week endpoint. [3]Vertex Press Release, March 9, 2026. 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

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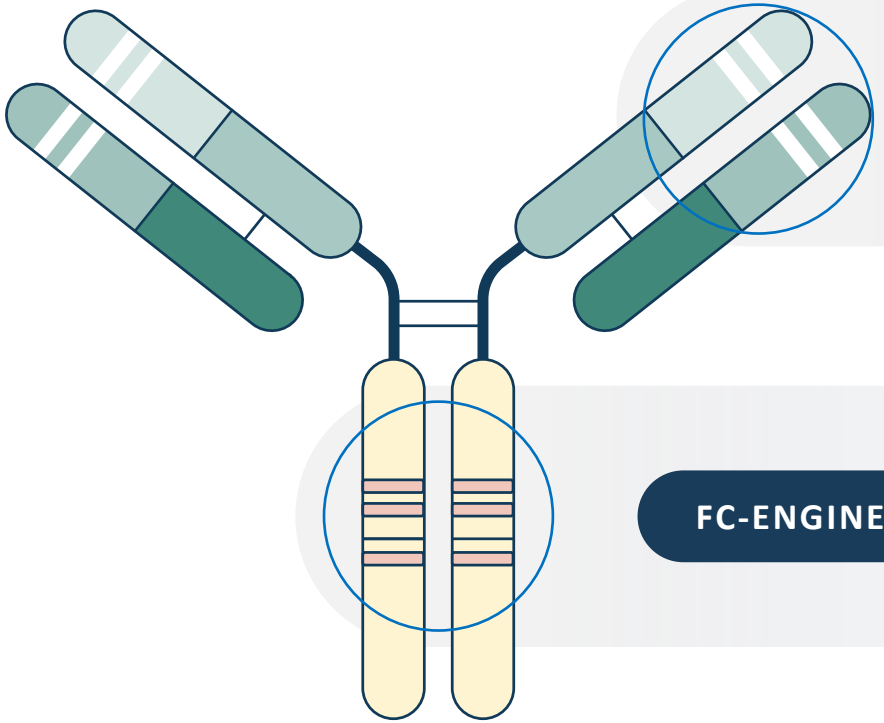
APRIL = a proliferation-inducing ligand, IV = intravenous, mo = months, Q4W = once every four weeks, SC = subcutaneous, UPCR = urine protein creatinine ratio, wks = weeks

[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).

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

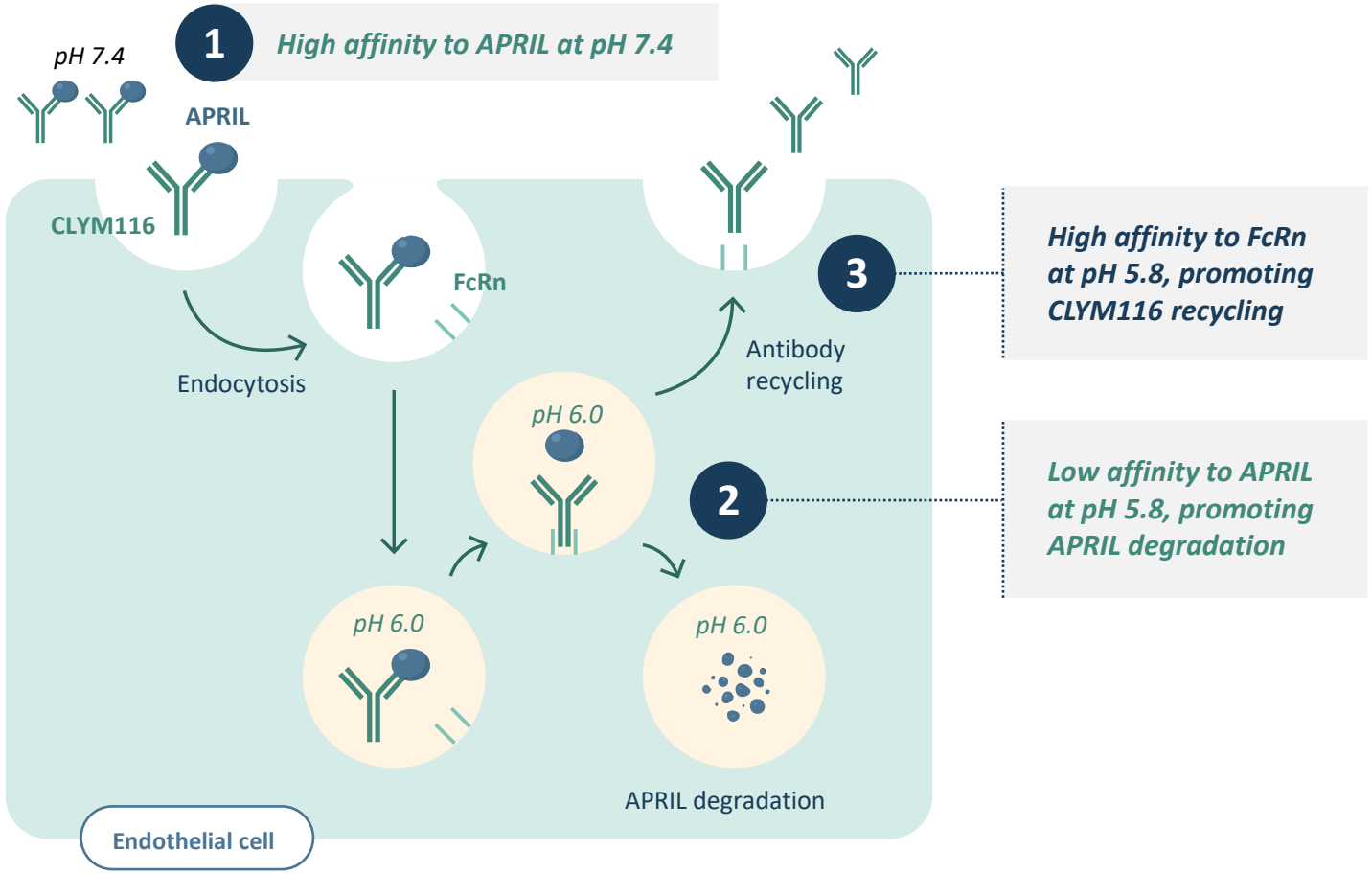
SWEEPER MECHANISM

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

CLIMBBIO

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

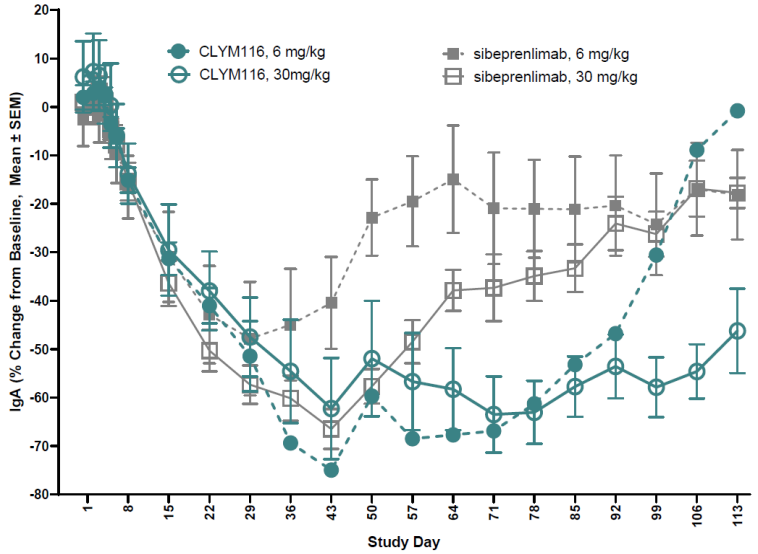
APRIL = a proliferation-inducing ligand, mAbs = monoclonal antibodies; Beeck, ASN 2025

CLYM116 Showed a Differentiated PK/PD Profile in NHPs

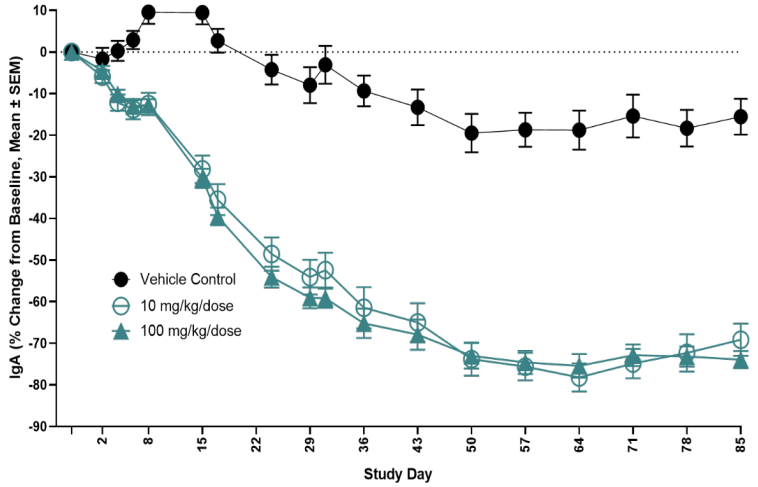
CLYM116 demonstrated deep and durable IgA suppression and a ~2-3 fold longer half-life vs. sibeprenlimab in NHPs

CLYM116 demonstrated deep and durable IgA suppression

Deep IgA suppression, with longer duration of effect vs. sibeprenlimab after a single SC administration*



Similar magnitude of IgA suppression (~70%) observed in a repeat dose toxicology study†



Confirmed ADA+ animals were excluded from the analysis

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

- **~2-3x longer serum half-life** vs. sibeprenlimab
- **Deeper IgA suppression** with a **longer duration of effect** vs. sibeprenlimab
- **~85% bioavailability** and **favorable tolerability**

ADA = anti-drug antibody, NHPs = nonhuman primates, SC = subcutaneous

*Data from a head-to-head study in nonhuman primates, 4-6 animals per cohort, single SC administration. †Toxicity study in nonhuman primates, 10-14 animals per cohort, SC administration on days 1, 15, 29. A subset were observed through an 8-wk recovery; 4 in the control and 100 mg/kg cohort were observed through an extended 6-mo recovery.

Luo ERA 2026; Beeck ASN 2025

CLIMBBIO

Clear Translation of Data from NHPs to HV to IgAN Patients

Emerging CLYM116 data support potential to have a best-in-class profile in IgAN

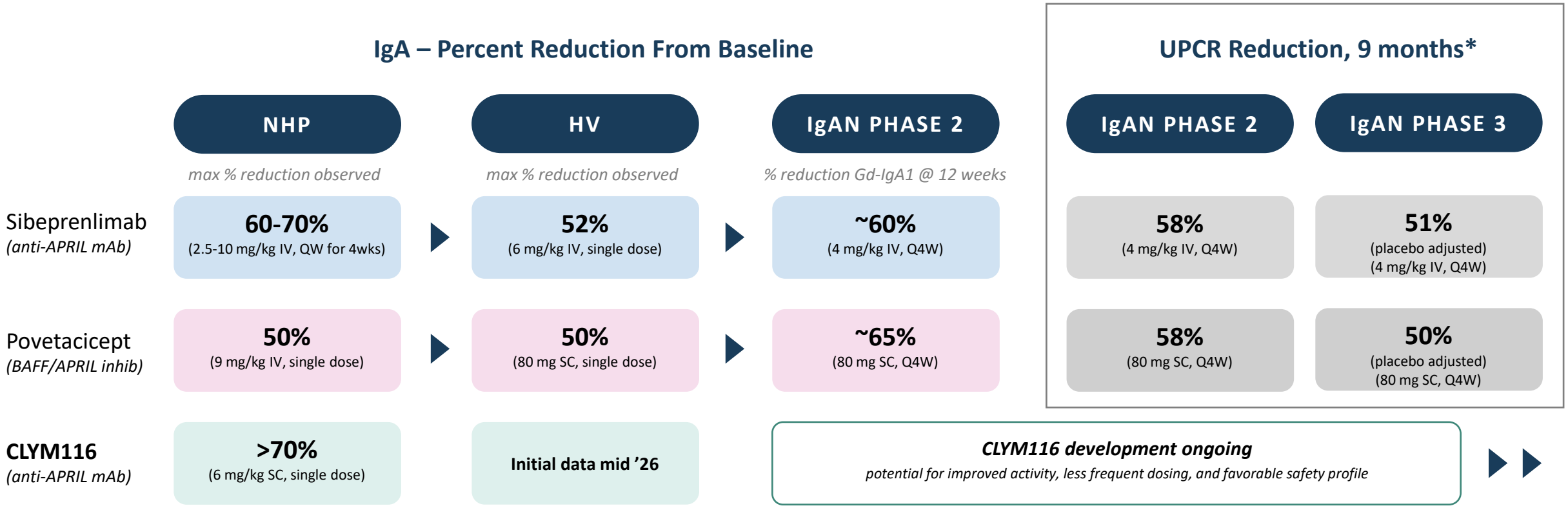


Table above reflects cross-study and cross-trial comparisons and not data from head-to-head studies; differences exist between trial designs and participant characteristics and caution should be exercised when comparing data across trials.

*Primary endpoint for accelerated approval. Reported as 9 months and Week 40 for sibeprenlimab Phase 2 and Phase 3, respectively, and Week 36 for povetacicept Phase 2 and Phase 3. Sibeprenlimab: Myette Kid Intl 2019, Mathur Kid Intl Reports 2022, Mathur NEJM 2024. Povetacicept: Evans Arthritis & Rheumatology 2023, Davies Clin Transl Sci 2024, Madan KI Reports 2026, Vertex Press Release, Mar 9, 2026. APRIL = a proliferation-inducing ligand, BAFF = B-cell activating factor, HV = healthy volunteers, IgAN = IgA nephropathy, inhib = inhibitor, 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.

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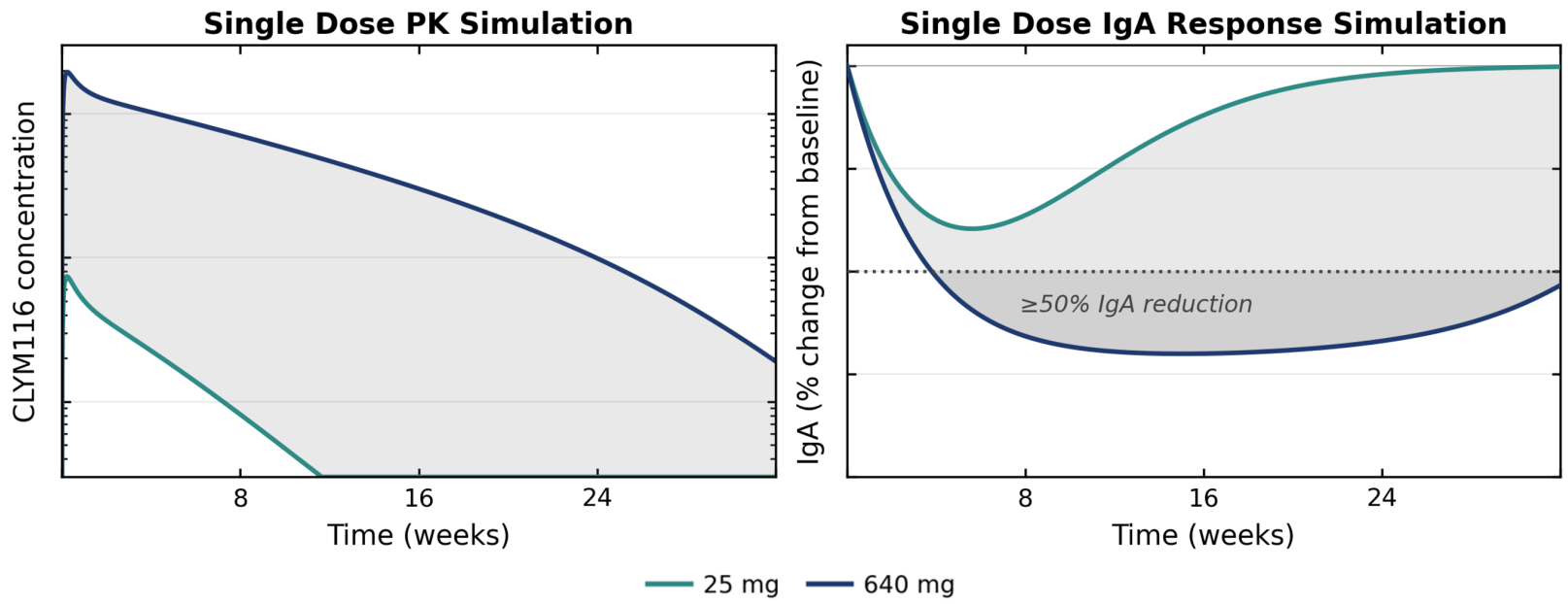
Translational Model Projects Dose-Dependent IgA Suppression in HV

CLYM116-specific PK/PD modeling derived from NHP data projected HV exposure and dose-dependent IgA suppression across the Ph1 dose range, with potential for less frequent dosing

Modeling Workflow

- Cyno PK/PD modeling (2-compartment + TMDD; Indirect-response IgA, IgG, IgM)
- Allometric extrapolation to human using exponents tuned for half-life-extended mAbs
- Healthy-volunteer SAD/MAD simulations (PK, IgA, Gd-IgA1, IgG, IgM)
- Validation against emerging Phase 1 observations

Extended Half-Life Supports Durable, Dose-Dependent IgA Suppression



HV = healthy volunteer, mAbs = monoclonal antibodies, MAD = multiple ascending dose, NHP = nonhuman primate, PD = pharmacodynamic, PK = pharmacokinetic, SAD = single ascending dose, TMDD = target-mediated drug disposition; Luo ERA 2026; Modeling based on pooled data from 4 SAD and MAD NHP studies (0.5–100 mg/kg SC, 6 mg/kg IV)

CLYM116 Global Phase 1 Strategy In Healthy Volunteers

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

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

Strategy: Parallel ex-China and China SAD datasets will yield a robust population PK foundation with built-in ethnic sensitivity assessment to de-risk next phase dose selection

Population
- Healthy volunteers

Primary Objective
- Safety and tolerability

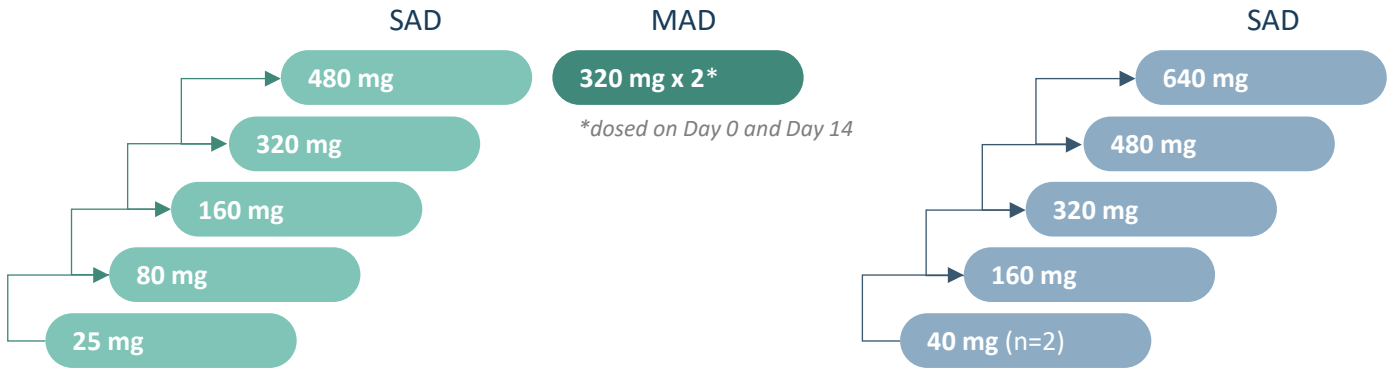
Secondary Objectives
- Pharmacokinetic profile
- Effect on immunoglobulins (IgA, IgM, and IgG) and APRIL levels (pharmacodynamic response)

Australia Phase 1 Study
Climb Bio

China Phase 1/2 Study
Mabworks

ASCENDING DOSE COHORTS, N ~80

Subcutaneous administration
8 subjects per cohort (6 CLYM116: 2 placebo)



APRIL = a proliferation-inducing ligand, IgAN = IgA nephropathy, MAD = multiple ascending dose, SAD = single ascending dose
Climb Bio Ph1: NCT07248865; Mabworks Ph1/2: NCT07375958

Safety

CLYM116 has been generally well tolerated in HV to date, with no unexpected safety findings

Preliminary safety data from ongoing studies in Australia and China evaluating single doses up to 320 mg (n=49)*

- No dose-limiting toxicities, serious adverse events, Grade ≥3 adverse events, or AE-related discontinuations
- All AEs mild to moderate (Grade 1-2), transient, and self-resolving
- Injection site reactions observed in 2 patients, both Grade 1, resolved without intervention

**China study is placebo-controlled and remains blinded. The reported population (n=49) includes participants who received CLYM116 (n=39) or placebo (n=10)*

AE = adverse event; HV = healthy volunteers
Luo ERA 2026

Looking ahead

Climb Bio: Data-Rich 2026

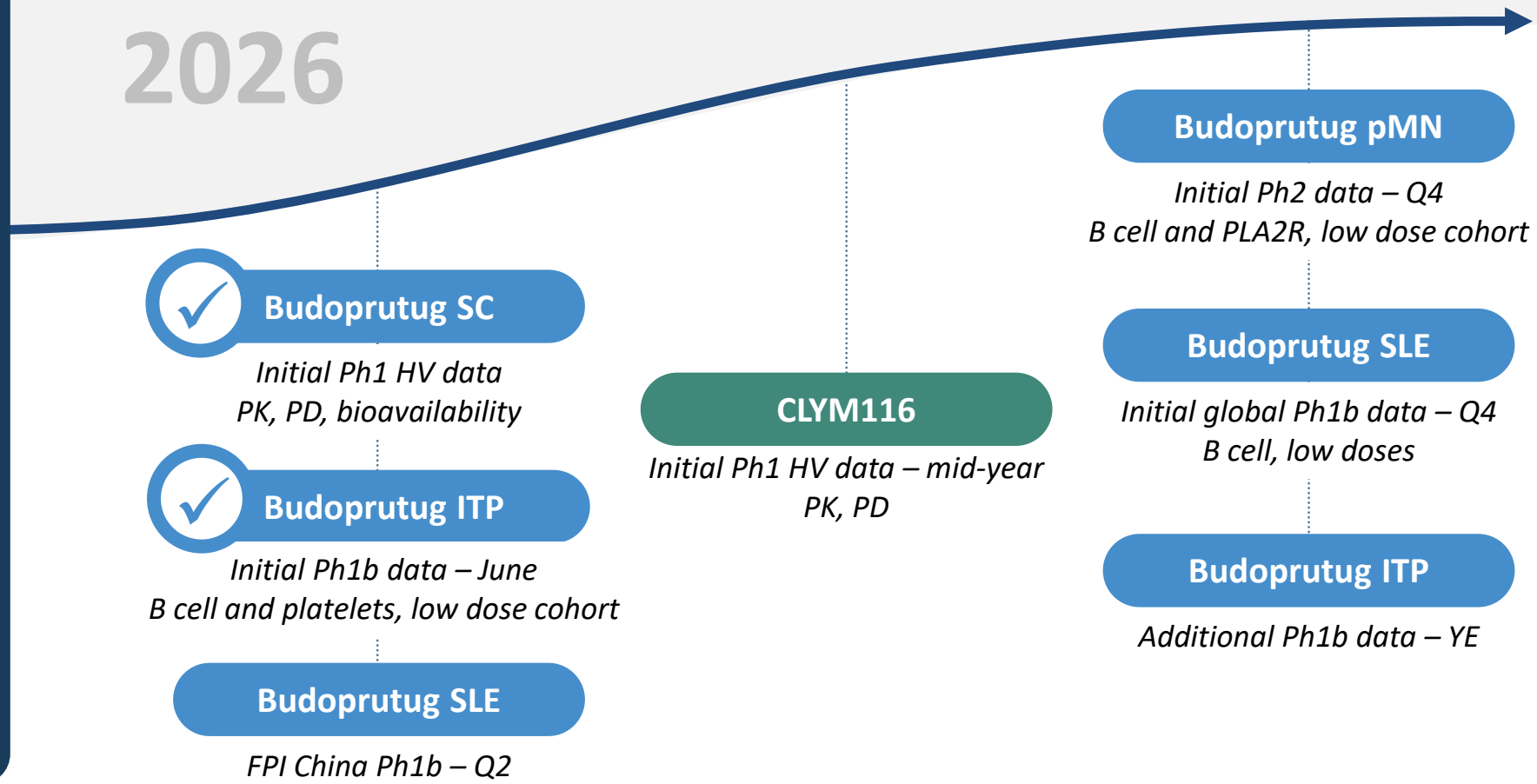
Continuing the ascent with initial readouts anticipated from all ongoing trials

$146.3M*

RUNWAY INTO 2028

79.8M†

FULLY DILUTED SHARES OUTSTANDING



*Cash, cash equivalents, and marketable securities as of March 31, 2026, excluding the proceeds received from the April 2026 private placement; †57.3 million common shares outstanding as of June 17, 2026; 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; Fast Track and Orphan Drug Designation granted for pMN
- **CLYM116 -** anti-APRIL mAb in development for IgAN

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

*Excluding the proceeds received from the April 2026 private placement

pMN = primary membranous nephropathy; ITP = immune thrombocytopenia; SLE = systemic lupus erythematosus; IgAN = IgA nephropathy