Corporate Presentation

OCTOBER 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 enrolling patients in Climb Bio’s ongoing and planned clinical trials; plans for the development strategy for budoprutug and CLYM116; the anticipated timelines for announcing data from Climb Bio’s ongoing and planned clinical trials of budoprutug and CLYM116 and Beijing Mabworks Biotech Co., Ltd.’s (“Mabworks”) trial of CLYM116; potential commercial opportunity and limited competitive landscape for budoprutug in primary membranous nephropathy, immune thrombocytopenia, and systemic lupus erythematosus and for CLYM116 in IgA nephropathy; projections and estimates derived from pharmacokinetic and pharmacodynamic modeling; expectations regarding cash runway and 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 regulations; 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; expectations regarding formulation and device readiness; 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; the possibility that models used in clinical development may be inaccurate; 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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OUR MISSION

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

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Scaling New Heights in the Development of Transformative Immune Medicines

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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

Wrapping up a **data-rich 2026** with **multiple clinical readouts** across both 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 the second half of 2028**

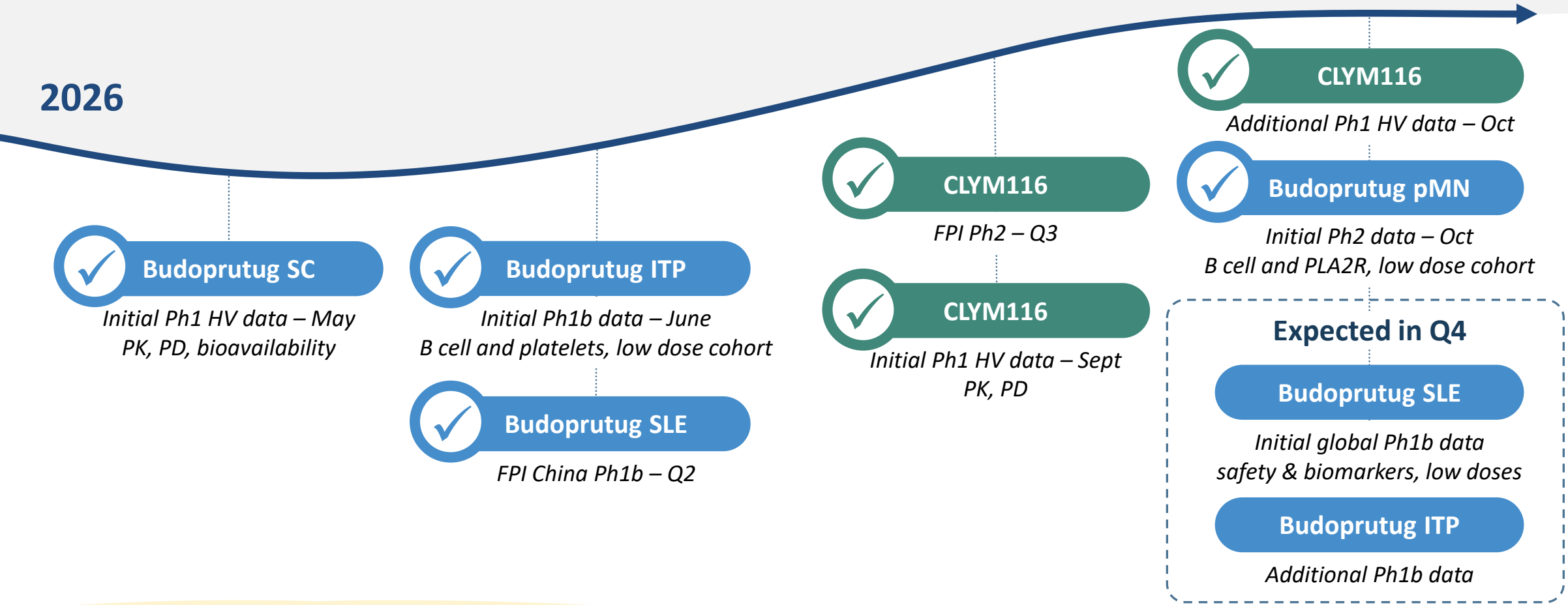
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pMN = primary membranous nephropathy, ITP = immune thrombocytopenia, SLE = systemic lupus erythematosus, IgAN = IgA nephropathy

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Climb Bio: Data-Rich 2026

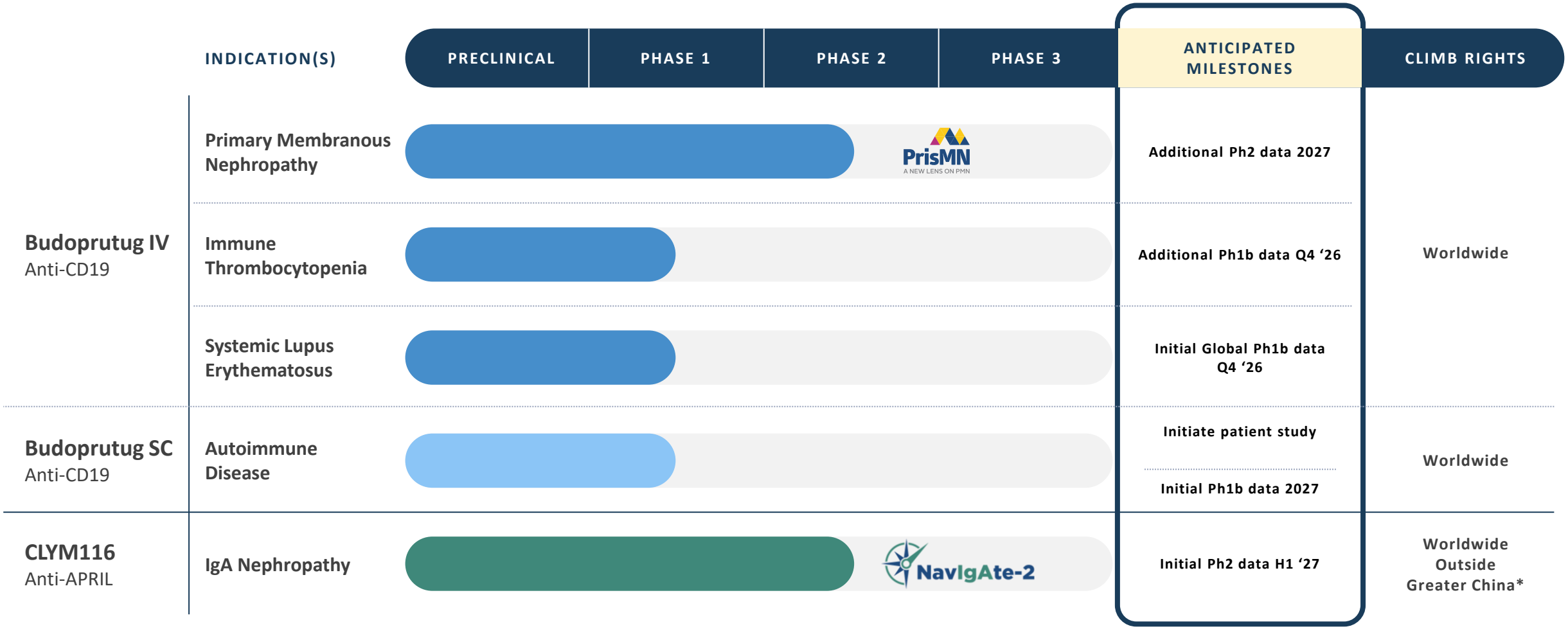
Delivered on 2026 data guidance, with budoprutug SLE and ITP readouts to close out the year



FPI = first patient in, HV = healthy volunteers, ITP = immune thrombocytopenia, PD = pharmacodynamics, PK = pharmacokinetics, pMN = primary membranous nephropathy, SC = subcutaneous, SLE = systemic lupus erythematosus

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Pipeline of Highly Differentiated mAbs



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.

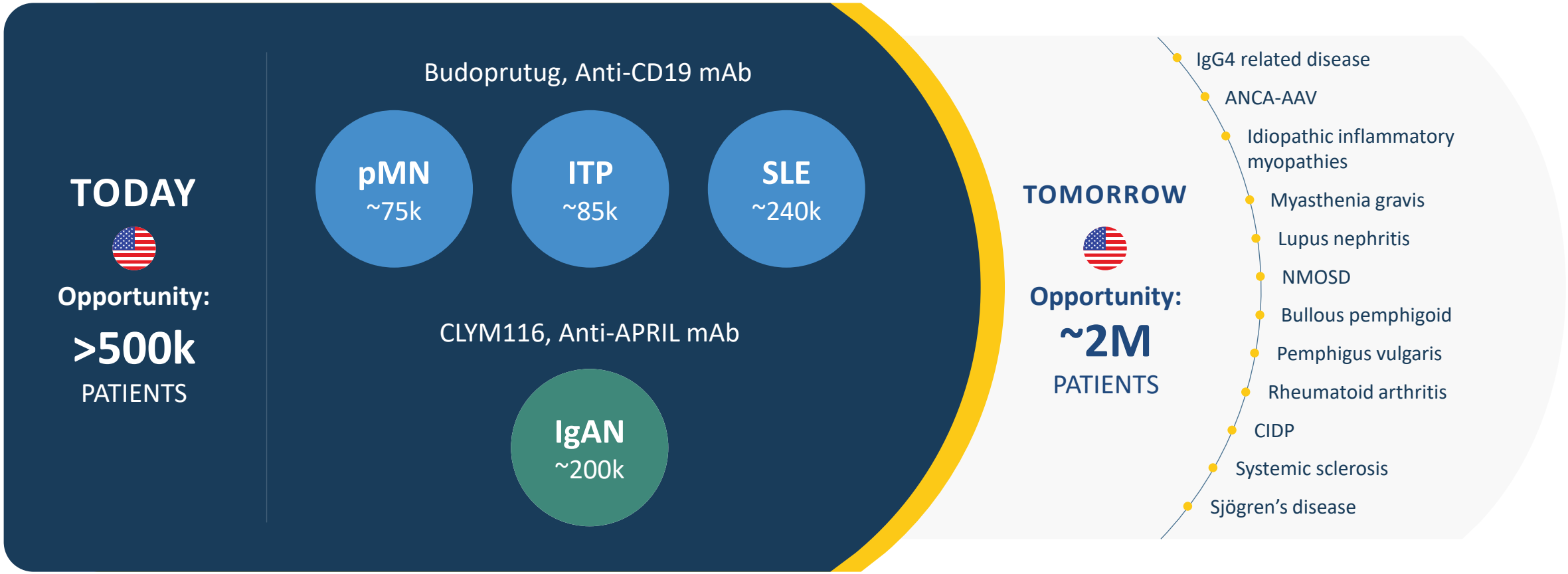
APRIL = a proliferation-inducing ligand, IV = intravenous, mAbs = monoclonal antibodies, SC = subcutaneous, HV = healthy volunteers

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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).

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Climb Bio Team Poised to Deliver for Patients



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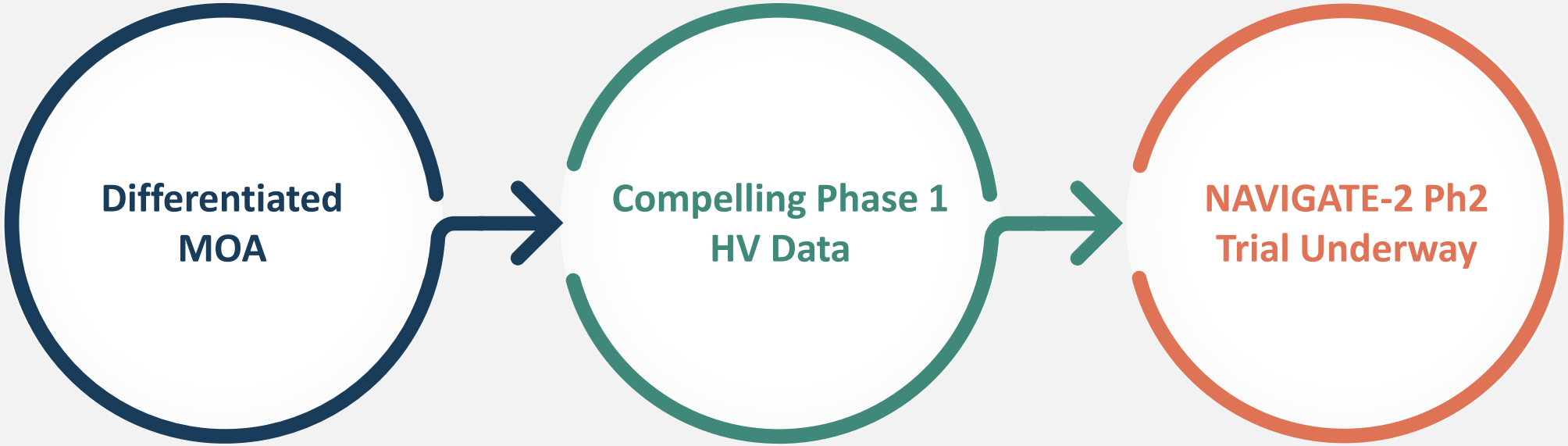
CLYM116

Anti-APRIL mAb

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

Initial data from NAVIGATE-2 Phase 2 trial expected in 1H 2027



- Sweeper mechanism facilitates recycling of CLYM116 and degradation of APRIL
- CLYM116 is designed for improved activity, less frequent dosing, and favorable safety profile

- CLYM116 Phase 1 data demonstrates potential best-in-class profile
  - ✓ Deep & durable APRIL, IgA and Gd-IgA1 suppression
  - ✓ Potential for every-12-week dosing
  - ✓ Favorable safety & tolerability profile

- NAVIGATE-2 Phase 2 trial in IgAN* initial data expected 1H 2027
- PFS development complete, autoinjector compatible drug product
- Phase 3 initiation anticipated in 2027, pending regulatory feedback

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*NAVIGATE-2 (NCT07375758) is being conducted in collaboration with our partner, Beijing Mabworks Biotech.
APRIL = a proliferation-inducing ligand, Gd = galactose deficient, HV = healthy volunteer, IgAN = IgA nephropathy, PFS = pre-filled syringe

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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, Gd-IgA1, 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, Gd-IgA1 = galactose-deficient IgA1, 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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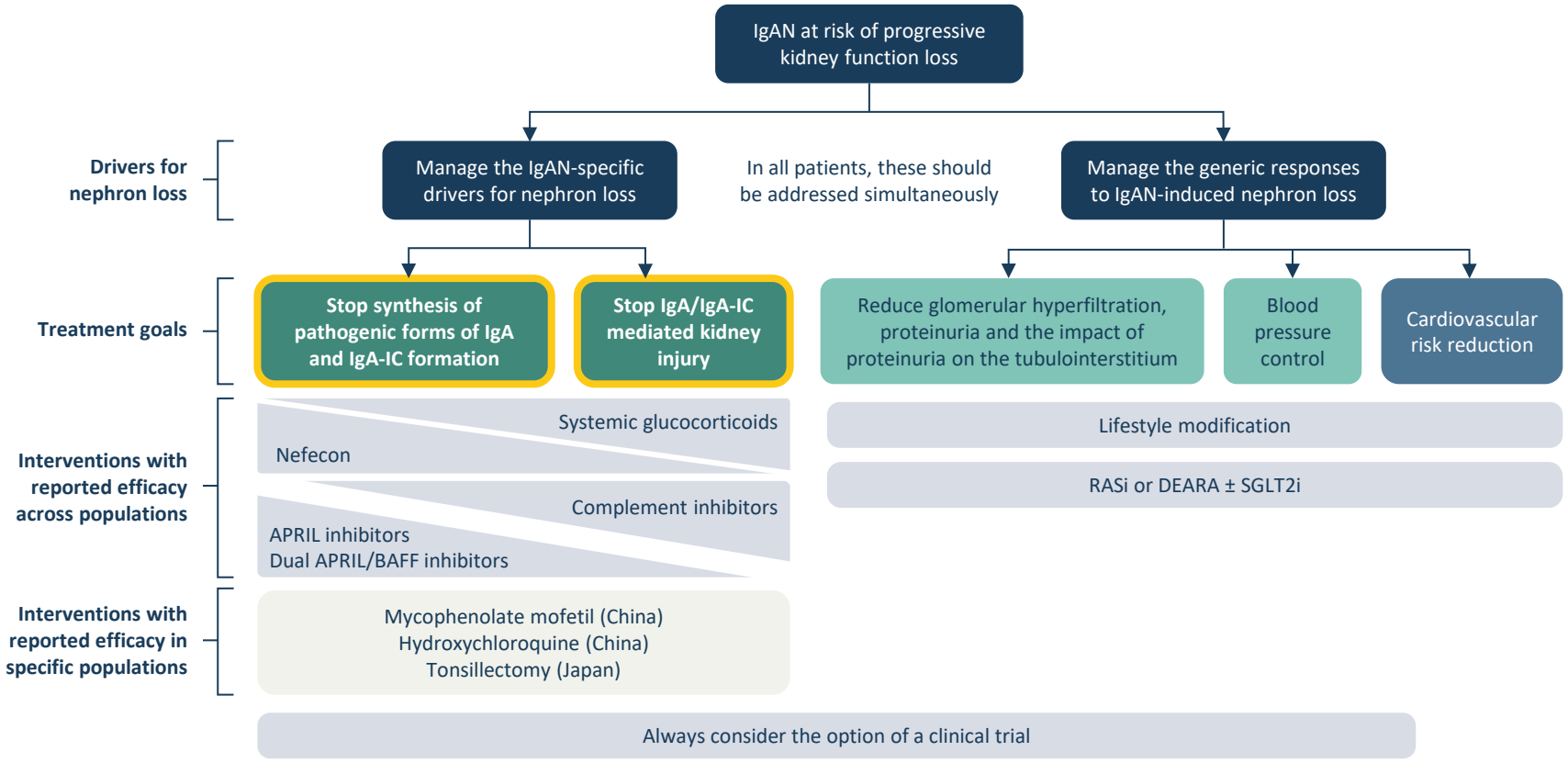
KDIGO Guidelines Highlight Impact of IgA Reduction in IgAN

KDIGO commentary includes anti-APRIL therapies as a core pillar in the treatment of IgAN, reinforcing need for disease modifying treatments that stop production of pathogenic IgA (Gd-IgA1)

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

Rovin Kidney International 2026

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APRIL is a Validated Target in IgAN with Further Potential to Unlock

Phase 3 results and recent approvals establish the APRIL class and create an opportunity for differentiated next-generation therapies

First Generation: Where We Are Now

APPROVED **Sibeprenlimab**[1,2] APRIL	**51.2%** placebo-adjusted UPCR reduction	Stabilization of eGFR through 2 years in Phase 3	**Q4W**
APPROVED **Atacicept**[3,4] BAFF/APRIL	**41.8%** placebo-adjusted UPCR reduction	Stabilization of eGFR through 2 years in Phase 3	**Q1W**
BLA FILED **Povetacicept**[5,6] BAFF/APRIL	**49.8%** placebo-adjusted UPCR reduction	Stabilization of eGFR through 48 weeks in Phase 1/2*	**Q4W**

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.

Next Generation: Where We're Headed

Depth and Durability of Suppression
Near-complete APRIL suppression

Robust IgA Response
Reduced pathogenic forms of IgA and IgA immune complex formation

Convenient Dosing Regimen
Substantive decrease from 12 to 4 injections annually

*Phase 3 eGFR data not yet available.
APRIL = a proliferation-inducing ligand, BAFF = B-cell activating factor, eGFR = estimated glomerular filtration rate, IgAN = IgA nephropathy, UPCR = urine protein creatinine ratio
[1] Perkovic NEJM 2025, [2] Rizk GlomCon 2026, [3] Lafayette NEJM 2025, [4] Barratt NEJM 2026, [5] Vertex Press Release, March 9, 2026, [6] Madan KI Reports 2025

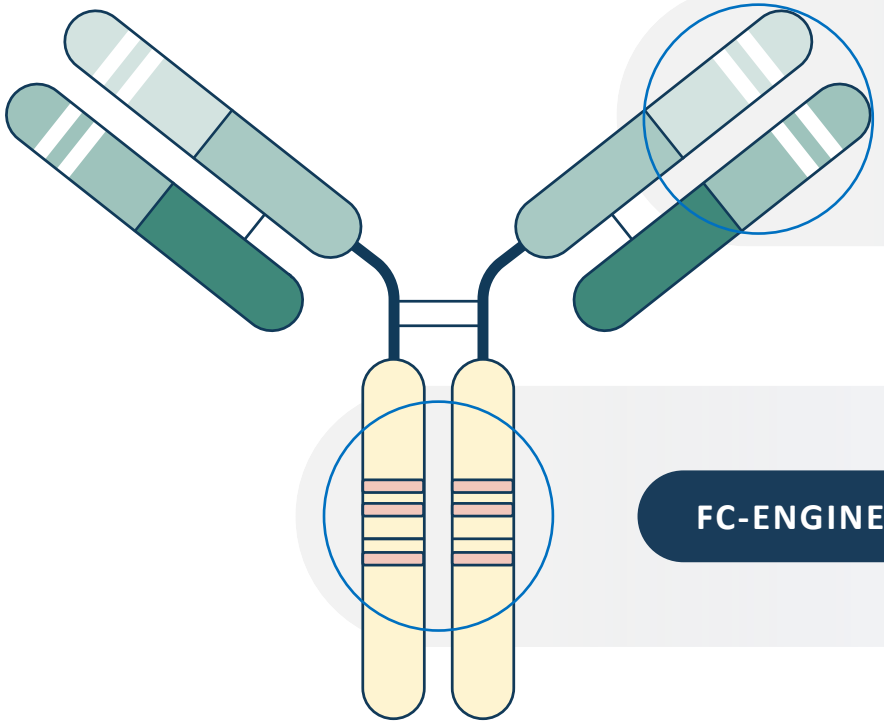
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CLYM116 Is The Only Known “Sweeper” Anti-APRIL In Development

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

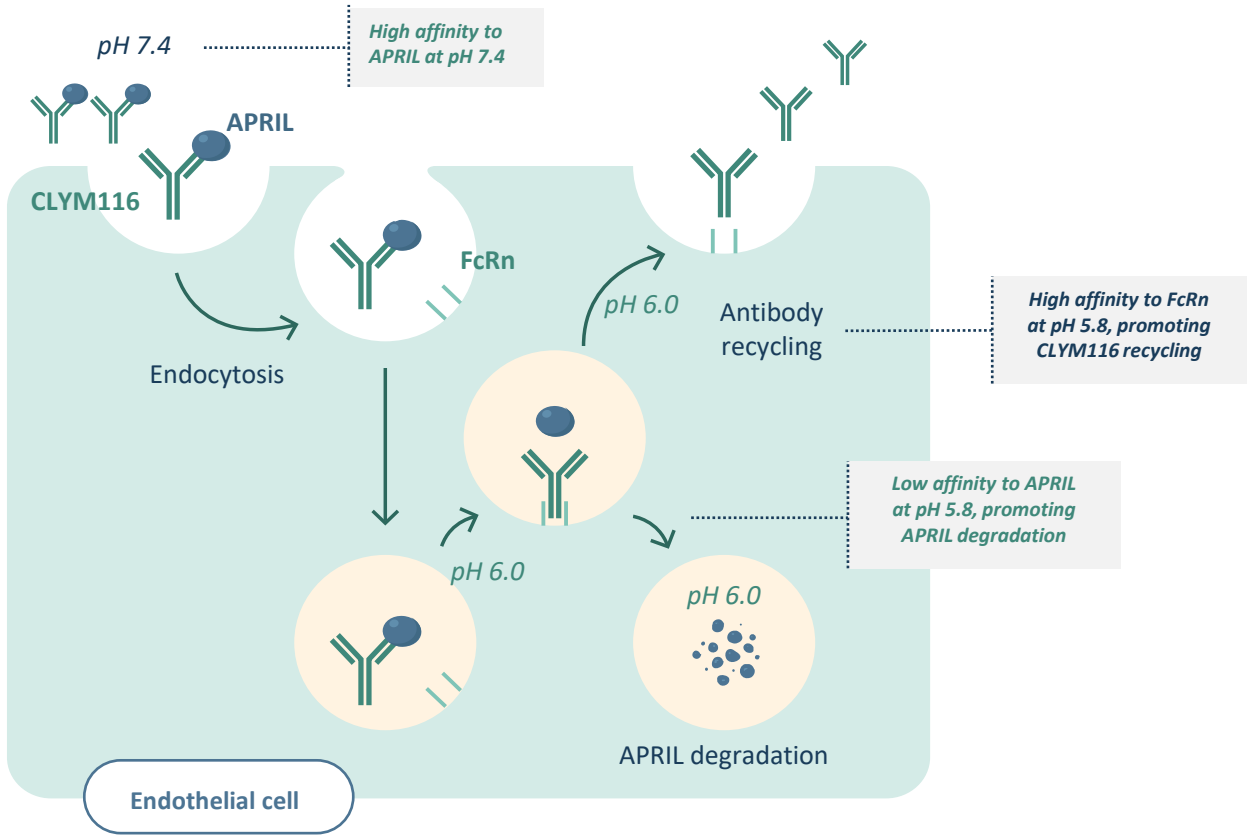
Facilitates recycling of CLYM116 and elimination of APRIL

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

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CLYM116 “Sweeper” Designed for a Differentiated Clinical Profile

pH-dependent binding and FcRn recycling allow CLYM116 to release captured APRIL for degradation and re-enter circulation to bind again



Enhanced clearance beyond conventional neutralization:

- ✓ **Deeper target suppression**
  Repeated capture and clearance of APRIL
- ✓ **Sustained activity**
  Antibody recycling enables continued target engagement
- ✓ **Less frequent dosing**
  Prolonged pharmacology may support an extended maintenance interval beyond half-life extension alone

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

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Phase 1 Data Support Best-in-Class Potential of CLYM116

Data from healthy volunteer study demonstrate differentiation across key clinical attributes

EFFICACY

Robust APRIL & IgA Suppression

- **>90% free APRIL suppression** through Week 12 after a single SC dose of ≥480 mg
- **~70% reduction in Gd-IgA1**, the pathogenic form of IgA, and **~60% reduction in IgA** at Week 12 after a single 800 mg dose

CONVENIENCE

Potential for Every 12-Week Dosing

- **Half-life of ~33 days**, ~3.5x longer than sibeprenlimab
- **Low dosing burden with target regimen;** 5 doses in year 1, then 4 doses per year

SAFETY

Favorable Safety Profile

- Generally well-tolerated
- **No treatment-related SAEs or DLTs**
- **No hypogammaglobulinemia**

Luo ASN 2026; Liang ASN 2026

APRIL = a proliferation-inducing ligand, DLTs = dose limiting toxicities, Gd = galactose deficient, SAEs = serious adverse events, SC = subcutaneous

Projected CLYM116 half-life based on 800 mg and 320 mg MAD. Target regimen: 800 mg loading dose, followed by 400 mg Q12W. Hypogammaglobulinemia defined as IgG < 300 mg/dL.

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CLYM116 Global Phase 1 Strategy In Healthy Volunteers

Data demonstrating deep, durable APRIL suppression and robust Gd-IgA1 reduction presented at ASN Kidney Week 2026

Strategy:
Parallel Australia 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

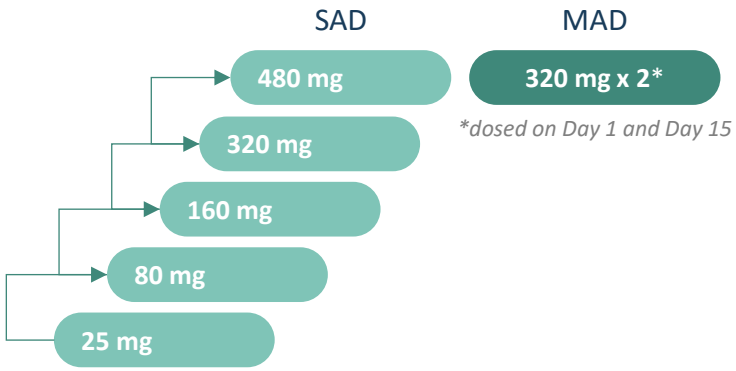
Secondary Objectives
- Pharmacokinetic profile
- Pharmacodynamics, including effects on serum APRIL and immunoglobulins (IgA, IgM, IgG, Gd-IgA1)

Australia Phase 1

Climb Bio sponsored study

ASCENDING DOSE COHORTS, N=46

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

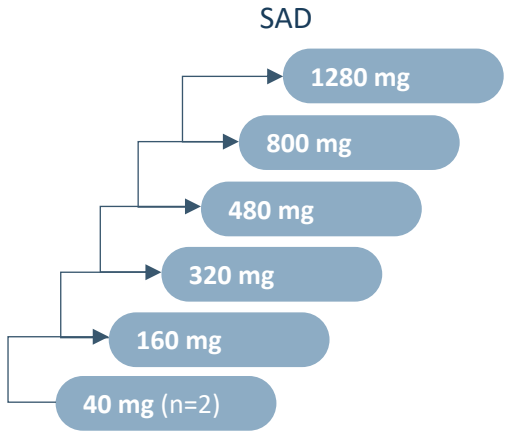


China Phase 1

Mabworks sponsored study

ASCENDING DOSE COHORTS, N=42

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



Luo ASN 2026; Liang ASN 2026
APRIL = a proliferation-inducing ligand, IgAN = IgA nephropathy, MAD = multiple ascending dose, SAD = single ascending dose
Australia Ph1: NCT07248865; China Ph1: NCT07375758 conducted by our partner, Beijing Mabworks Biotech

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CLYM116 Demonstrated Favorable Safety and Tolerability in HV

Well-tolerated across 25-1280 mg, with no treatment-related SAEs or hypogammaglobulinemia

Safety

67 participants received CLYM116 across both studies

- No dose-limiting toxicities
- No treatment-related SAEs
- No discontinuations due to AEs
- No hypogammaglobulinemia
- Injection-site reactions observed in 5 patients, all Grade 1

	CLYM116 (pooled, N=67)	**Placebo** (pooled, N=21)
Any TEAE, n (%)	42 (63%)	10 (48%)
Any TRAE, n (%)	15 (22%)	6 (29%)
Grade ≥3 TRAE, n (%)	0	0
Treatment-related SAE, n	0	0
Discontinuation due to AE, n	0	0

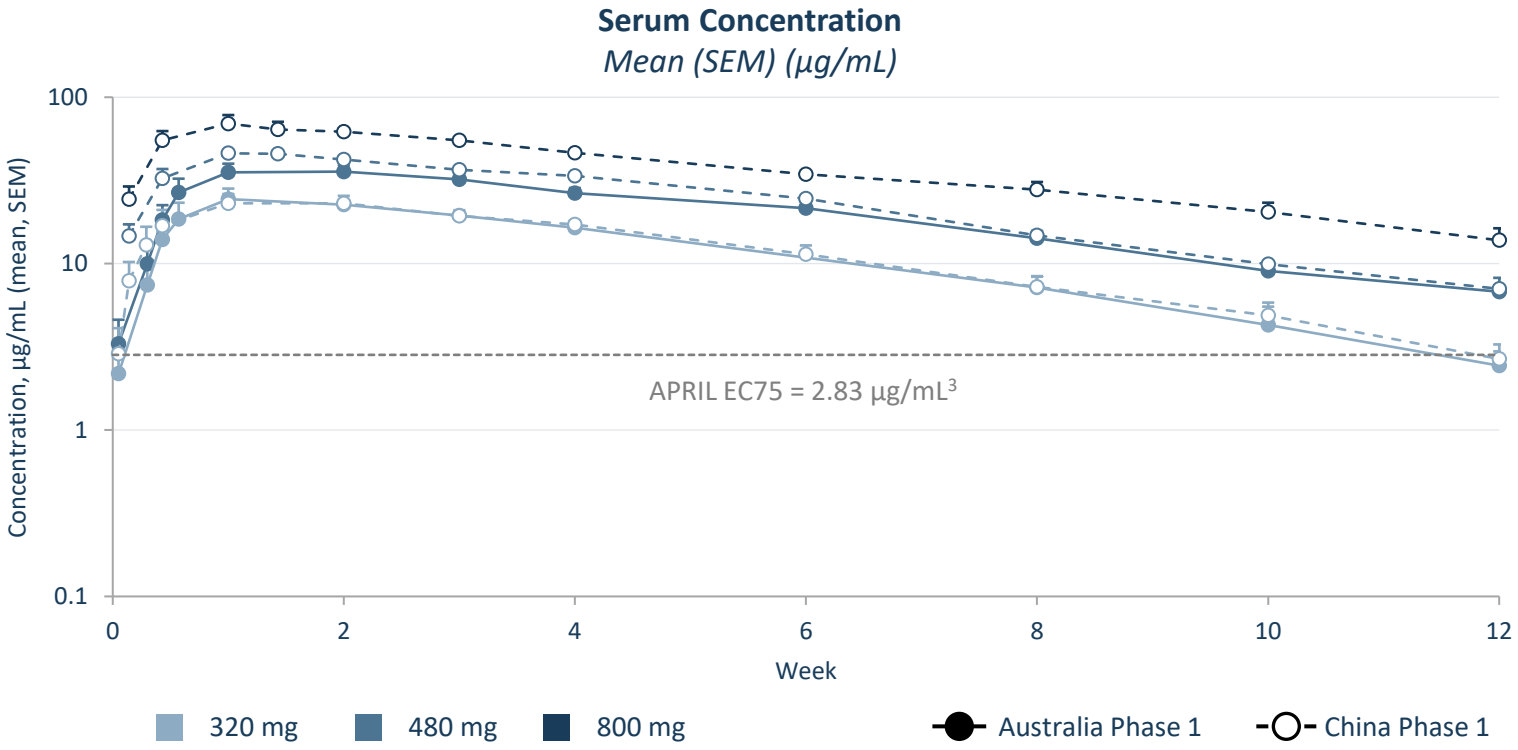
Pooled across Australia Phase 1 (25-480 mg SAD; 320 mg Day 1 and Day 15 MAD) and China Phase 1 (40-1280 mg SAD). Source: Luo ASN 2026; Liang ASN 2026
AE = adverse event, DLT = dose-limiting toxicity, HV = healthy volunteers, SAE = serious adverse event, TEAE = treatment-emergent AE; TRAE = treatment related AE; hypogammaglobulinemia defined as IgG < 300 mg/dL

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CLYM116 Exposure Profile Supports Long Dosing Interval

~33-day half-life, ~3.5x longer than sibeprenlimab, with dose-proportional exposure and consistent PK across the Australia and China studies



Pharmacokinetic Profile

- **Half-life of ~33 days** vs. 9.3 days for sibeprenlimab[1,2]
- **Dose-proportional exposure** across the dose range
- **Linear clearance** above 1 µg/mL; no TMDD at doses ≥160 mg over 12 weeks
- **Data consistent** across Australia and China studies

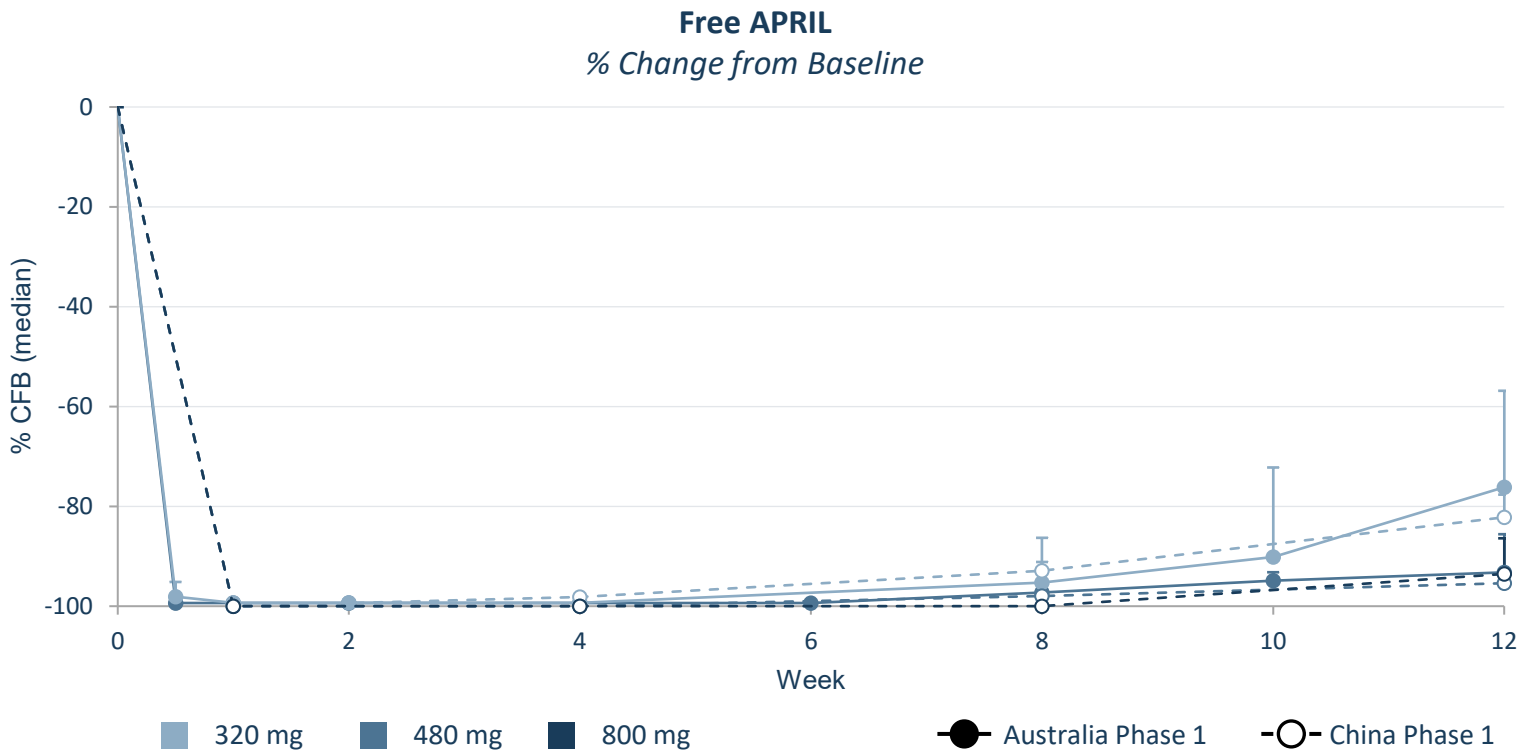
Mean serum concentration after a single SC dose (n=6 per cohort); error bars show +SEM. Projected half-life based on 320 mg MAD and 800 mg; half-life dependent on dose and dose interval.
Source: Luo ASN 2026; Liang ASN 2026.
APRIL = a proliferation-inducing ligand, EC75 = concentration producing 75% of maximal APRIL suppression, SC = subcutaneous, TMDD = target-mediated drug disposition.
[1] Zhang Clin Pharm Drug Dev 2023, [2] Sibeprenlimab US Prescribing Information, [3] Based on a PK/PD model developed from individual subject Ph1 data

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CLYM116 Achieved Rapid and Sustained Free APRIL Suppression

≥90% free APRIL suppression through Week 12 with single doses ≥480 mg, consistent across the Australia and China studies



APRIL Suppression

- **≥98% suppression by Day 4** at doses ≥160 mg
- **≥90% suppression through Week 12** at doses ≥480 mg
- **Data consistent** across Australia and China studies

Median % change from baseline after a single SC dose; error bars show the upper quartile (Q3); MAD = 320 mg on Day 1 and Day 15 (Australia).
Source: Luo ASN 2026; Liang ASN 2026

APRIL = a proliferation-inducing ligand, CFB = change from baseline, MAD = multiple ascending dose, SC = subcutaneous

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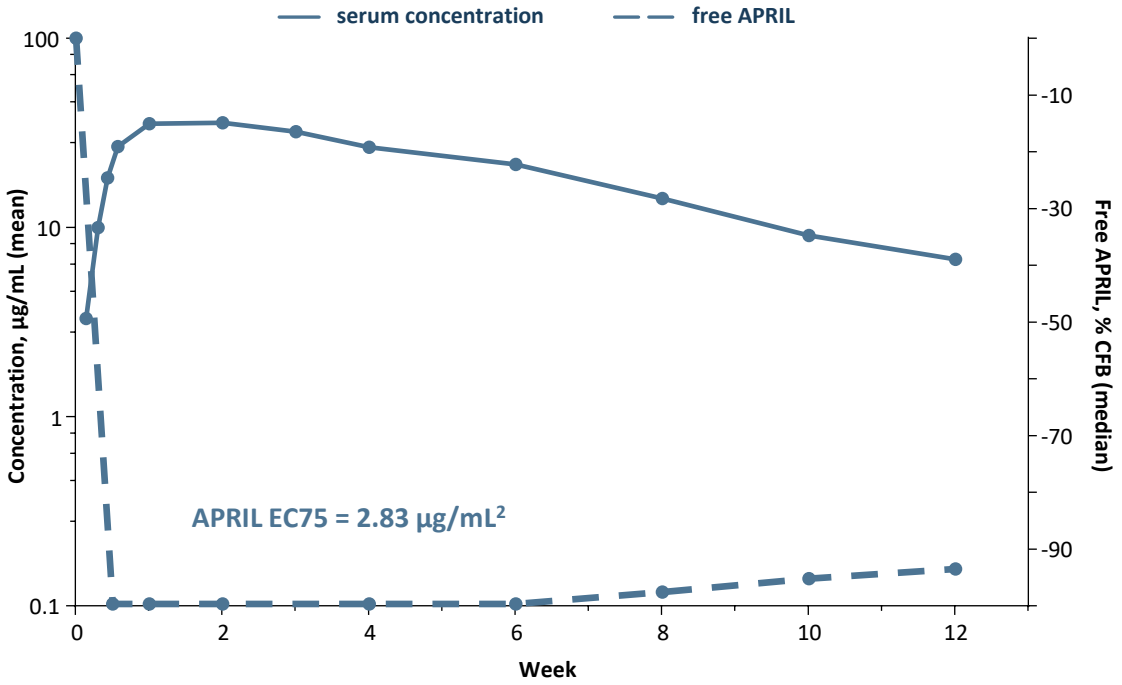
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CLYM116: Longer APRIL Suppression vs. Sibeprenlimab

CLYM116 showed greater in vivo potency and maintained ≥90% free APRIL suppression through Week 12 at 480 mg, while sibeprenlimab suppression waned after Week 6

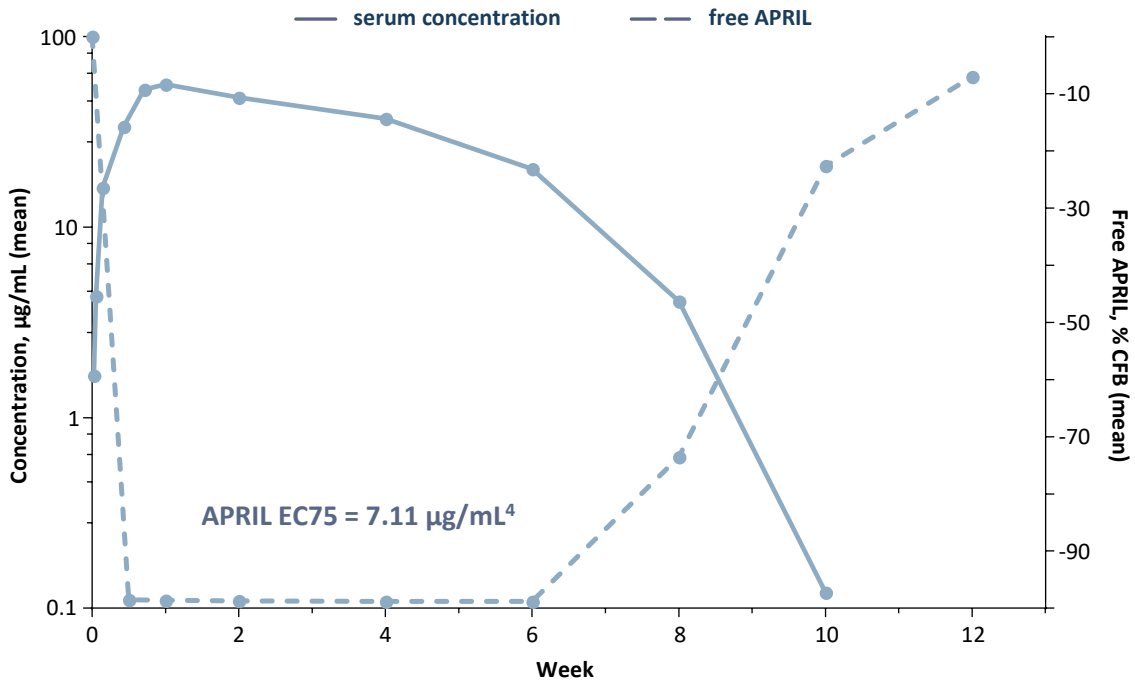
CLYM116[1]

480 mg SC (single dose)



Sibeprenlimab[3]

400 mg SC (single dose)



Above reflects cross-trial comparisons of observed single-dose healthy-volunteer data and not data from head-to-head studies; cross-trial comparisons are limited by differences in study design, populations, assays, regimens, and follow-up.

CLYM116 free APRIL shown as median and sibeprenlimab as mean (as published). APRIL = a proliferation-inducing ligand, CFB = change from baseline, PD = pharmacodynamic, PK = pharmacokinetic, SC = subcutaneous, TMDD = target-mediated drug disposition. [1] Luo ASN 2026, [2] Based on a PK/PD model developed from individual subject Ph1 data, [3] Adapted from Zhang Clin Pharm Drug Dev 2023, [4] Based on a PK/PD model developed from published mean cohort Ph1 data (Mathur KI Reports 2022; Zhang Clin Pharm Drug Dev 2023)

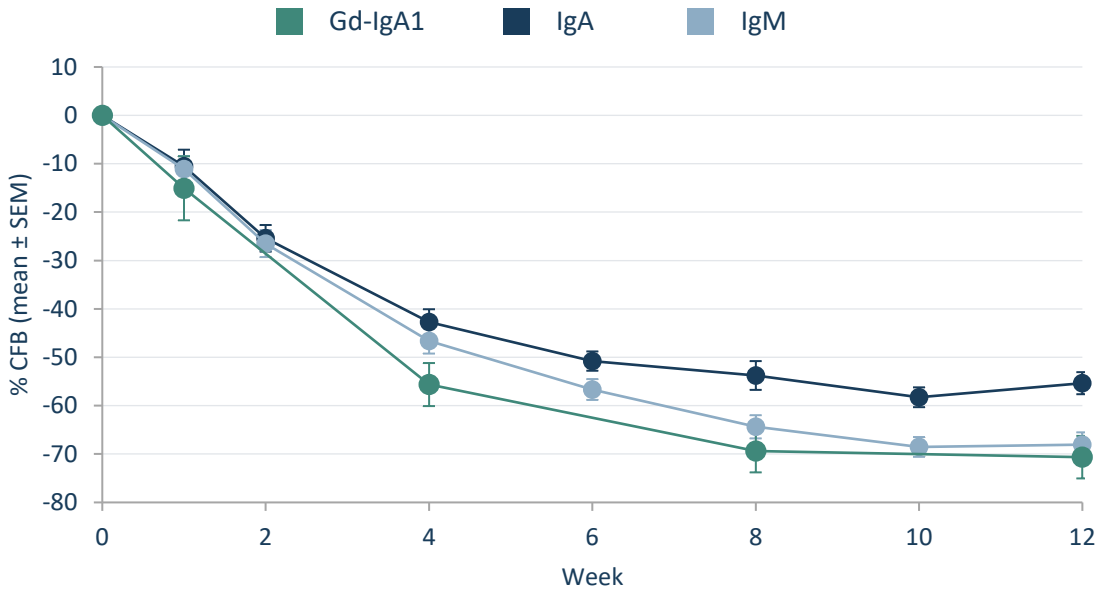
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CLYM116 Demonstrated ~70% Reduction in Gd-IgA1

Deep, durable reductions in Gd-IgA1, IgA and IgM

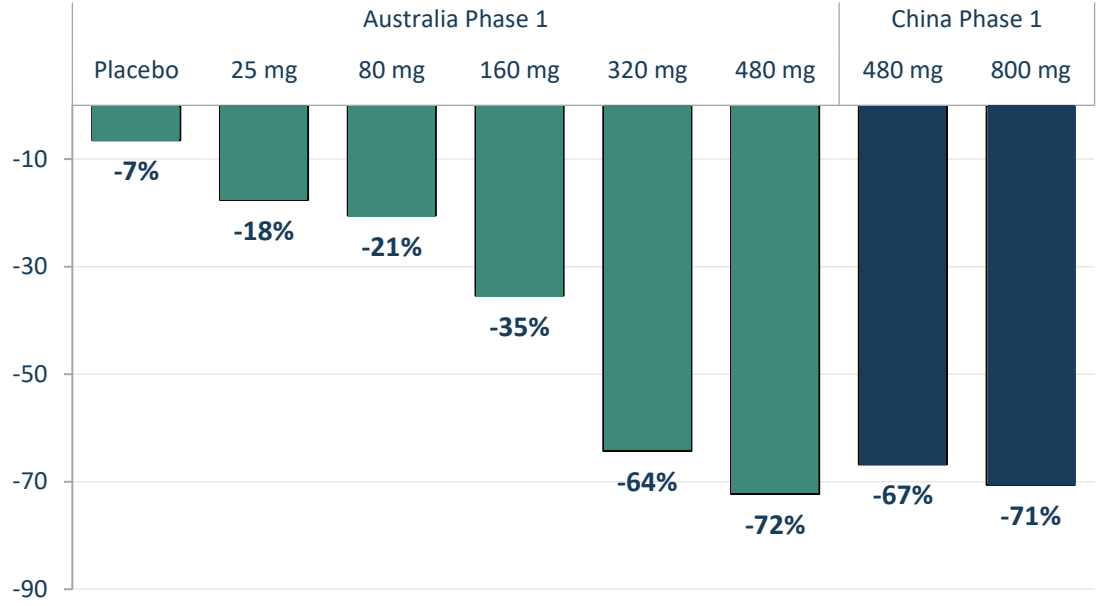
800 mg Single Dose

% change from baseline, mean ± SEM



Gd-IgA1 at Week 12

% change from baseline, mean



IgG reductions did not exceed 30% at any dose; lowest individual IgG 5.8 g/L vs. 3 g/L hypogammaglobulinemia threshold; no meaningful change in IgE

Source: Liang ASN 2026; Luo ASN 2026

Left: China Phase 1, 800 mg SAD, Right: Australia Phase 1 SAD (placebo pooled, n=11) and China Phase 1 SAD. Australia MAD cohort (320 mg Day 1 and Day 15) achieved 78% Gd-IgA1 reduction at Week 12; CFB = change from baseline, Gd-IgA1 = galactose-deficient IgA1, MAD = multiple ascending dose, SAD = single ascending dose

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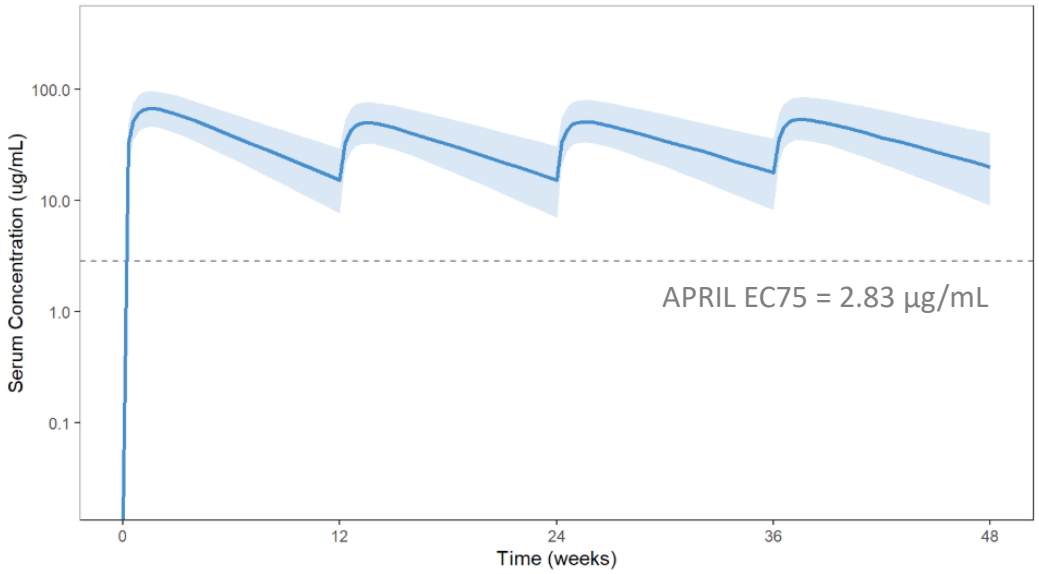
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CLYM116 PK/PD Modeling Supports Q12W Dosing

400 mg Q12W regimen predicted to keep median trough concentrations ~4x above APRIL EC75 and maintain near-complete APRIL suppression through 48 weeks

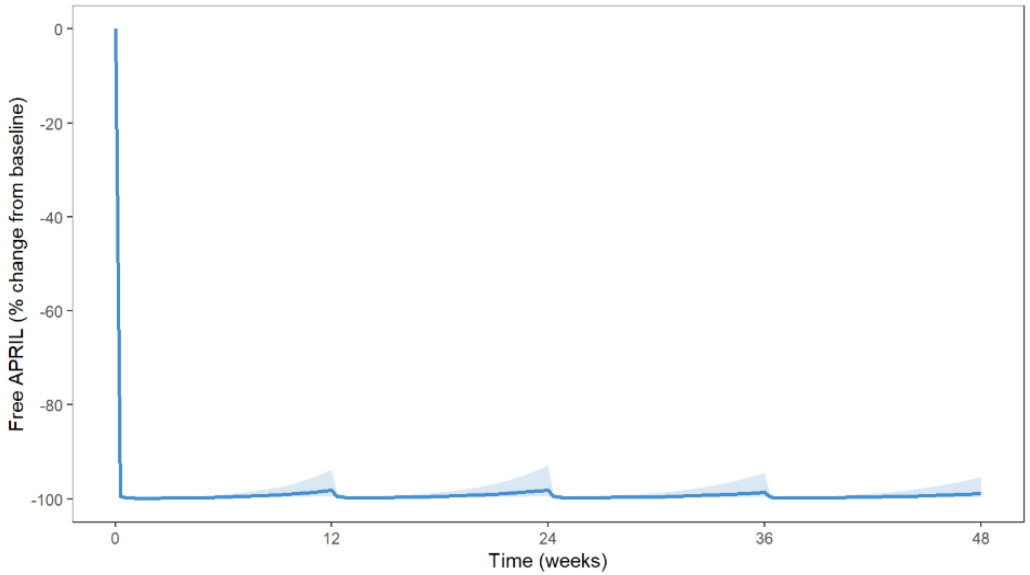
Simulated Serum Concentration Over Time

800 mg loading dose on Day 1, then 400 mg Q12W starting Week 12



Simulated Free APRIL Over Time

800 mg loading dose on Day 1, then 400 mg Q12W starting Week 12



Median 25-75% Percentile

Source: Luo ASN 2026; Liang ASN 2026. Simulated serum concentrations are derived from a population pharmacokinetic model, and free APRIL levels are derived from a population pharmacokinetic / pharmacodynamic model. Both models were developed with interim data from two Phase 1 studies in healthy subjects. The simulations show the median and 25-75th percentiles of 1000 virtual subjects with the indicated dosing regimen.
APRIL = a proliferation-inducing ligand, PD = pharmacodynamic, PK = pharmacokinetic

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CLYM116 NAVIGATE-2 Phase 2 Study in IgAN Ongoing

NavIgAte-2

Initial data anticipated in H1 2027; designed to confirm integrated Phase 1 PK/PD modeling and support advancing Q12W regimen into Phase 3

Population:

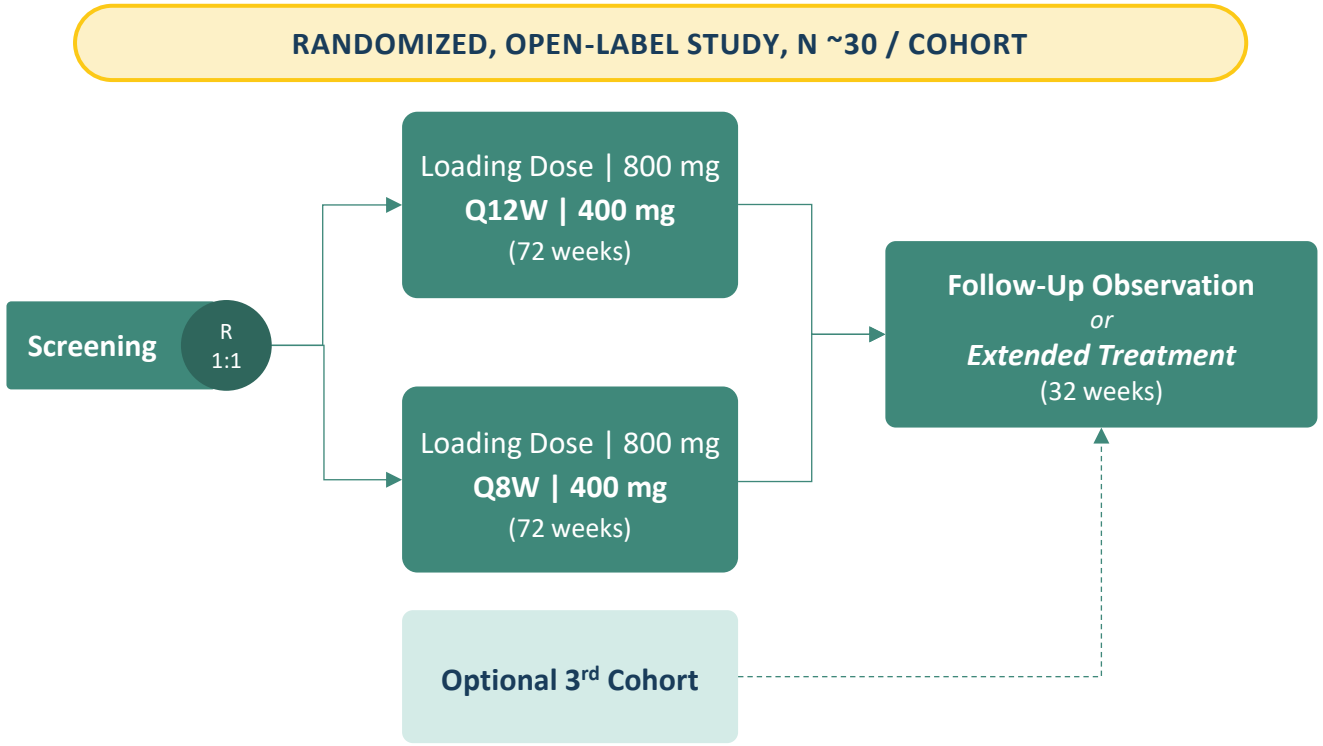
- Adults with biopsy-proven IgAN
- 24-hour UPCR ≥ 0.5 g/g or 24-hour urine protein ≥ 0.75 g
- eGFR ≥ 30 mL/min/1.73 m²
- Stable, maximum tolerated ACEi/ARB for ≥ 12 weeks

Designed to evaluate:

- Safety and tolerability
- Gd-IgA1
- 24-hour UPCR
- eGFR slope
- Serum immunoglobulin profile
- Free APRIL

NAVIGATE-2 Phase 2

Climb Bio / Mabworks collaborative, multi-center study being conducted in China



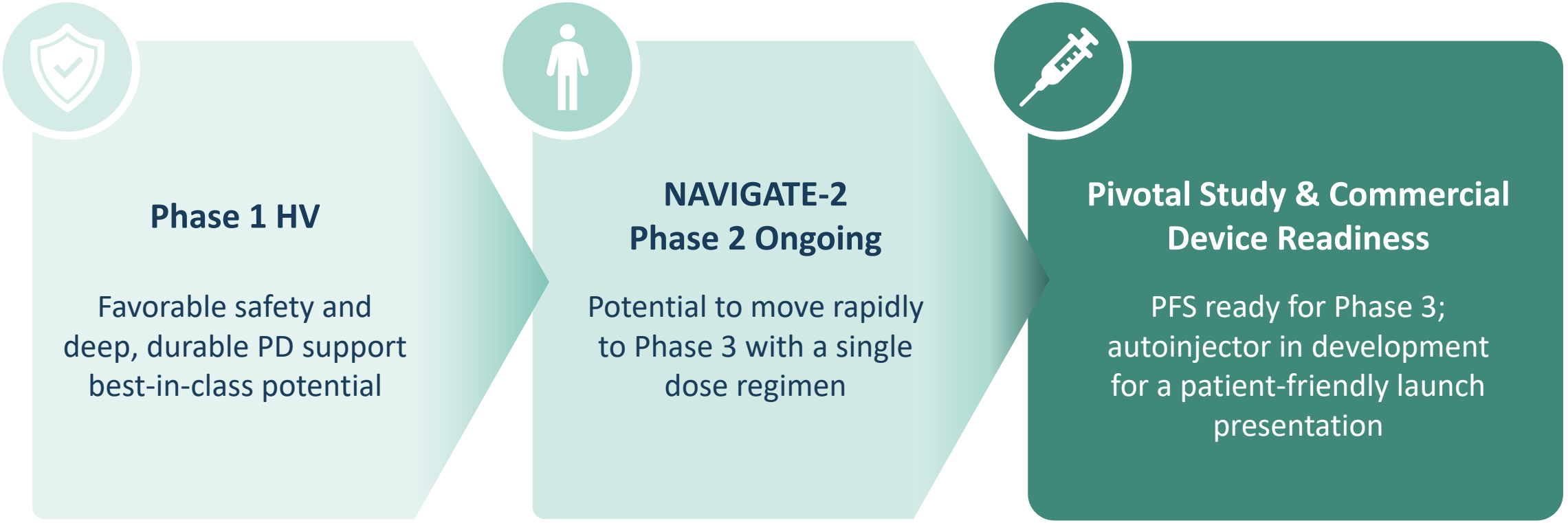
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ACEi = angiotensin-converting enzyme inhibitor, APRIL = a proliferation-inducing ligand, ARB = angiotensin II receptor blocker, eGFR = estimated glomerular filtration rate, Gd = galactose deficient, IgAN = IgA nephropathy, PD = pharmacodynamics, PK = pharmacokinetics, R = randomization, UPCR = urine protein creatinine ratio; NCT07375758; Dose regimens are 800 mg loading dose on Day 1, then 400 mg Q12W starting Week 12, and 800 mg loading dose on Day 1, then 400 mg Q8W starting Week 8; Lv ISN Frontiers 2026

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CLYM116: Compelling Phase 1 Data, Clear Path Ahead

CLYM116 offers the potential for durable disease control with convenient Q12W dosing; NAVIGATE-2 Phase 2 ongoing and device development advancing



HV = healthy volunteer, PD = pharmacodynamics, PFS = pre-filled syringe

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Budoprutug

Anti-CD19

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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+

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

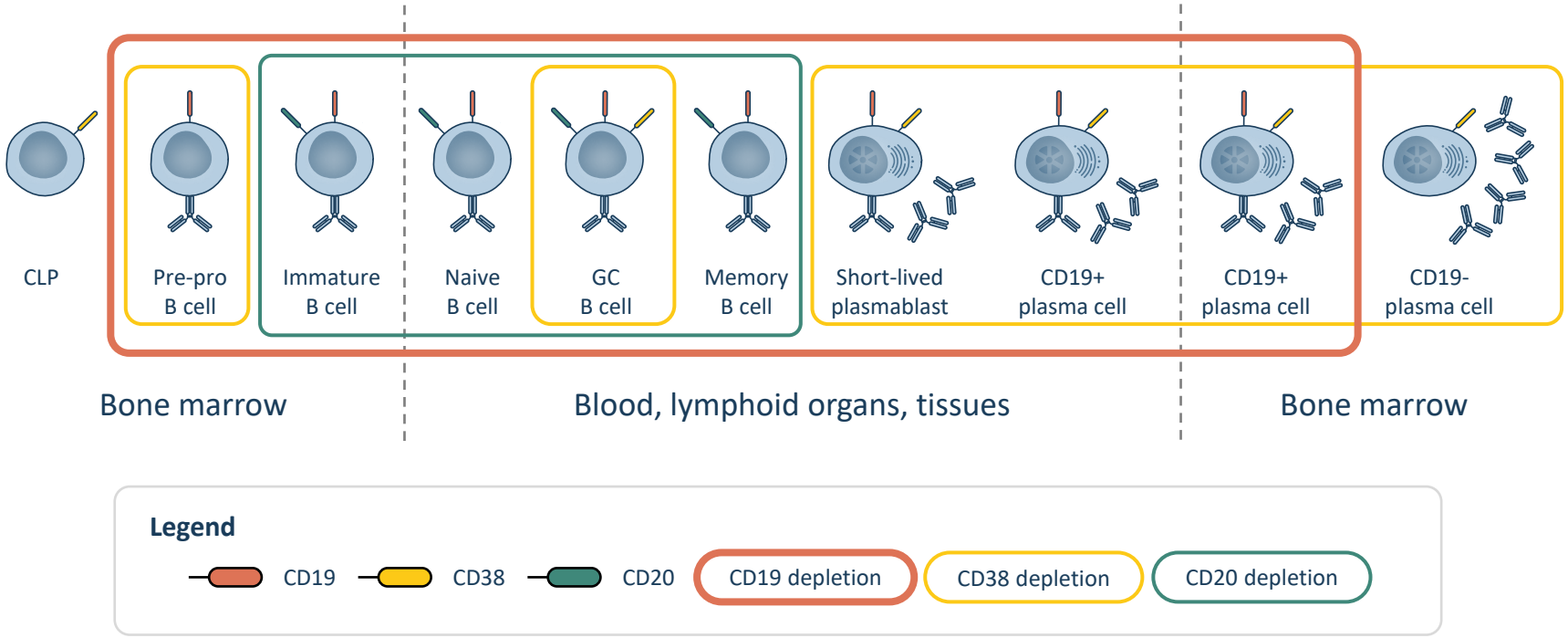
Cortazar ASN 2024

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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

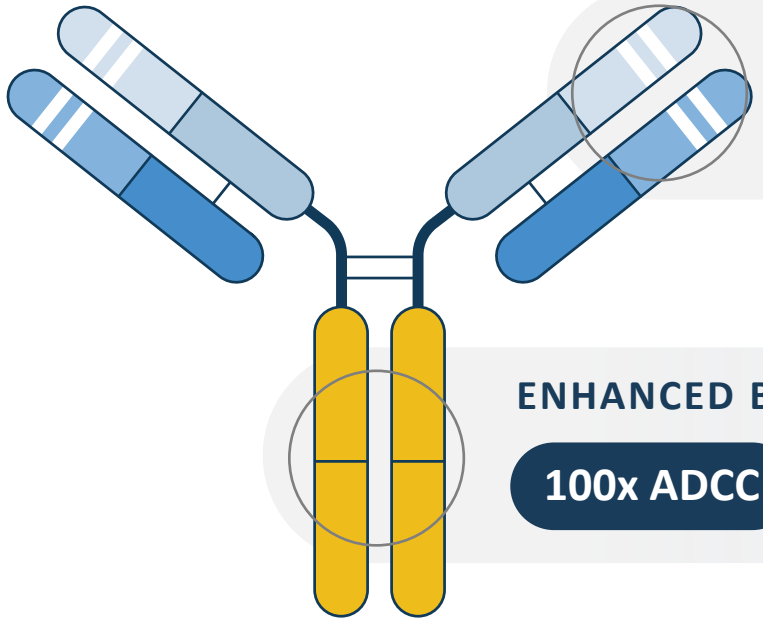
[1] Adapted from Suan J Immunol 2025, [2] Tur Annals Rheum Dis 2024

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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

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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

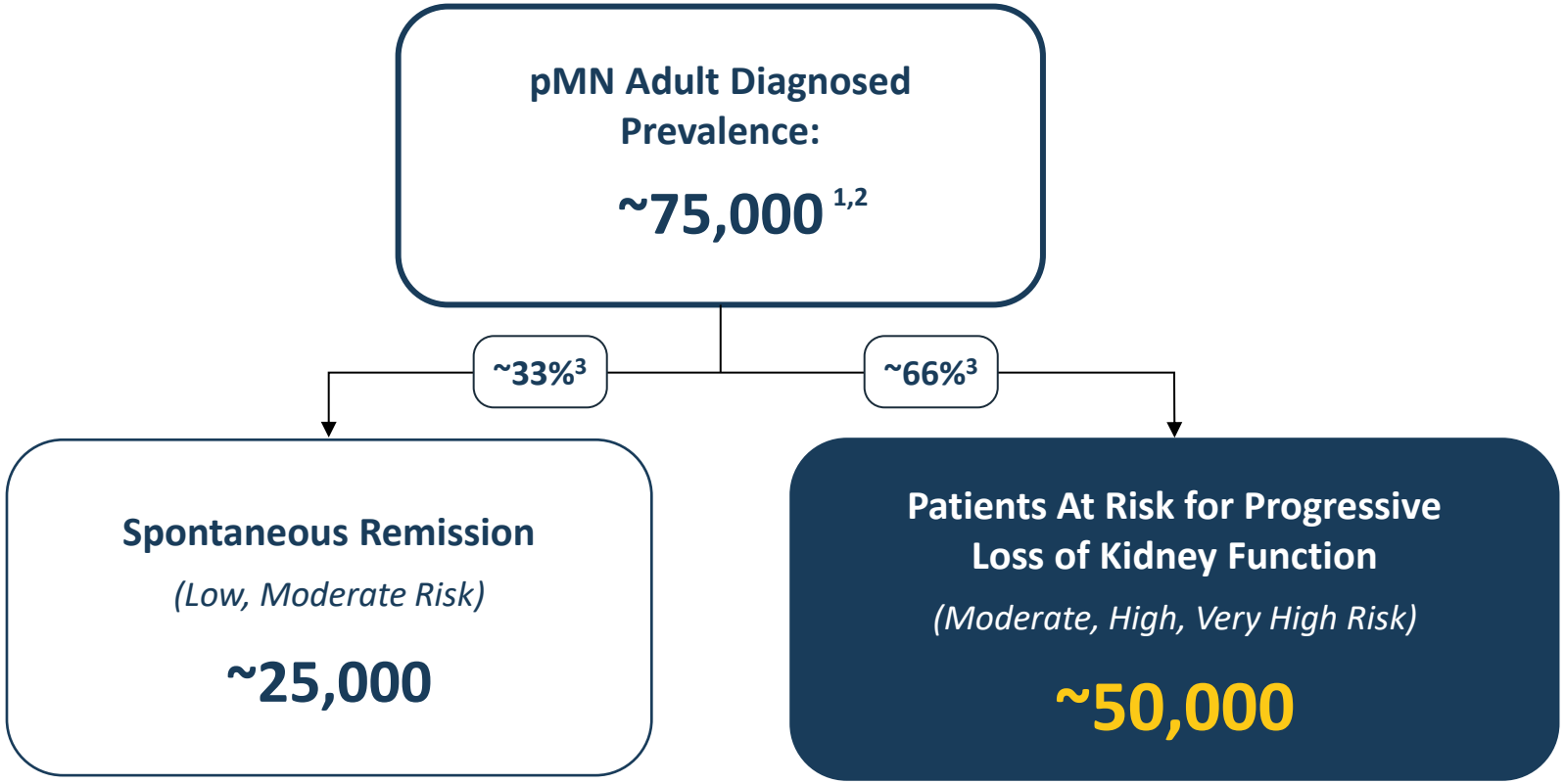
pMN = primary membranous nephropathy; Couser CJASN 2017, Ronco J Clin Med 2021, Teisseyre Front Immunol 2022, KDIGO 2021 guidelines
US Prevalence: McGrogan Nephrol Dial Transplant 2011, ~1 per 100,000; U.S. Census Estimates 2020-2025, assumes 28-year duration of pMN
No FDA-approved therapies for pMN as of October 2026; FDA decision on obinutuzumab (Gazyva) for pMN expected November 2026

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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

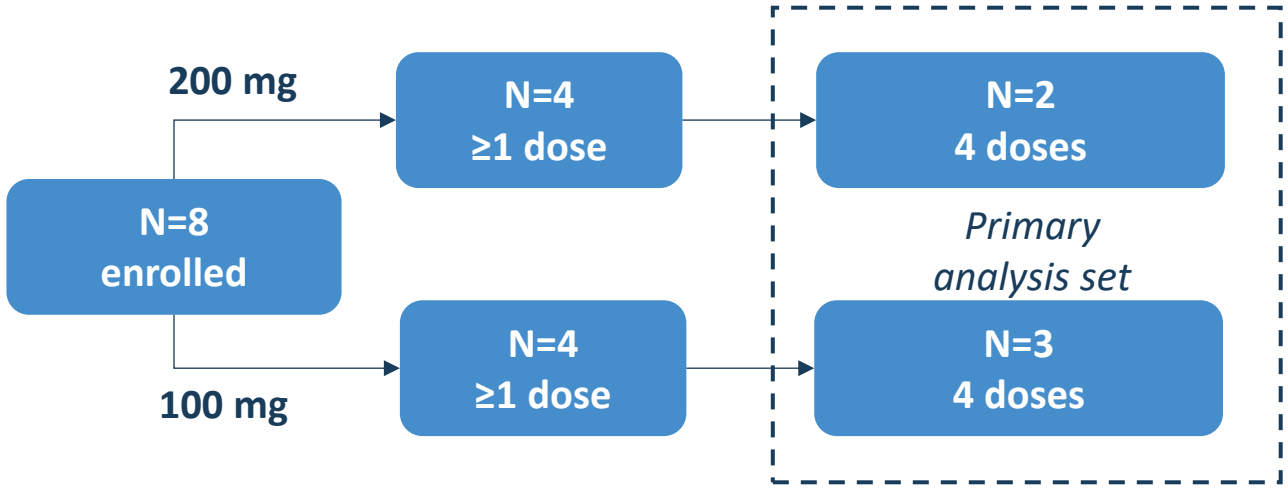
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Completed Phase 1b Study Established Proof-of-Concept in pMN

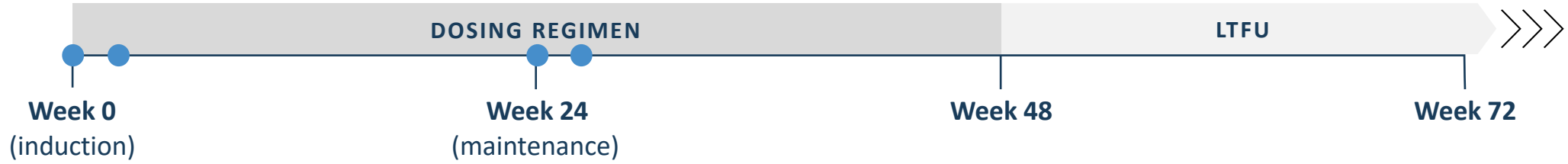
Budoprutug evaluated in an open-label, dose escalation study in adult patients with pMN

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



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



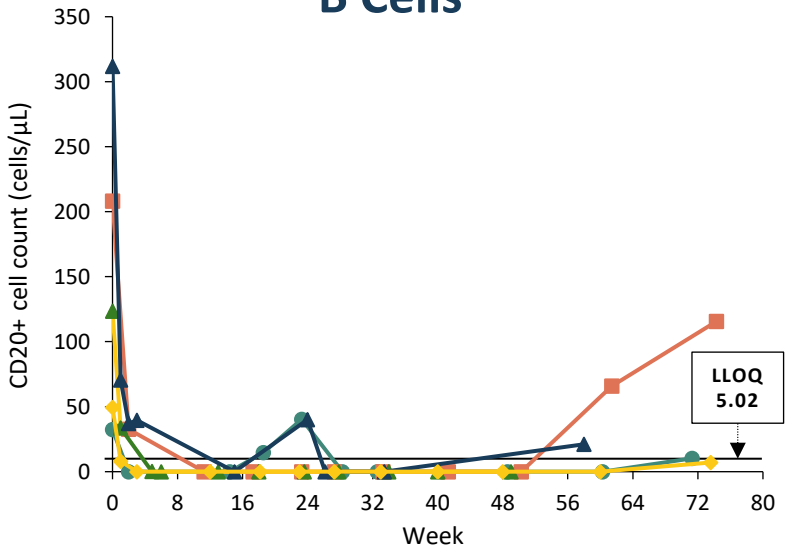
NCT04652570; IV = intravenous, LTFU = long term follow-up, PLA2R = phospholipase A2 receptor, pMN = primary membranous nephropathy, UPCR = urine protein creatinine ratio
Cortazar ASN 2024; Baseline characteristics for primary analysis set (N=5), PLA2R from patients with positive anti-PLA2R antibodies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

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

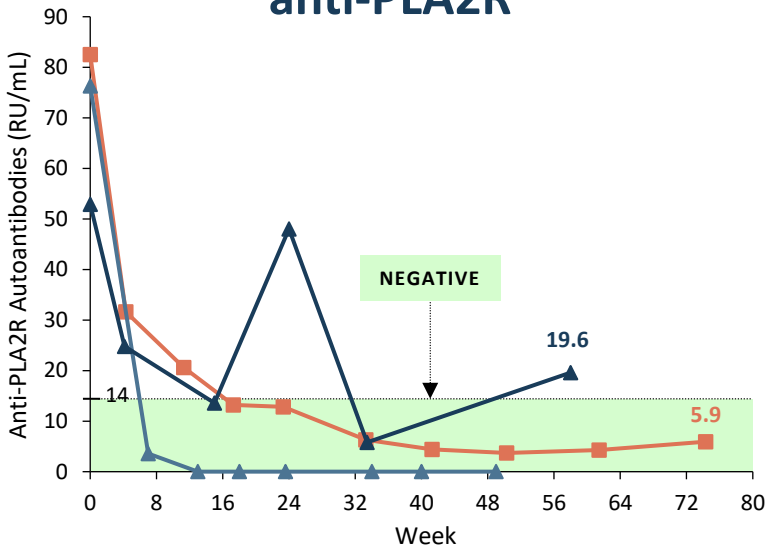
Budoprutug administration resulted in durable B-cell depletion, serologic remission, and clinical remission (as measured by proteinuria)

B Cells[1]



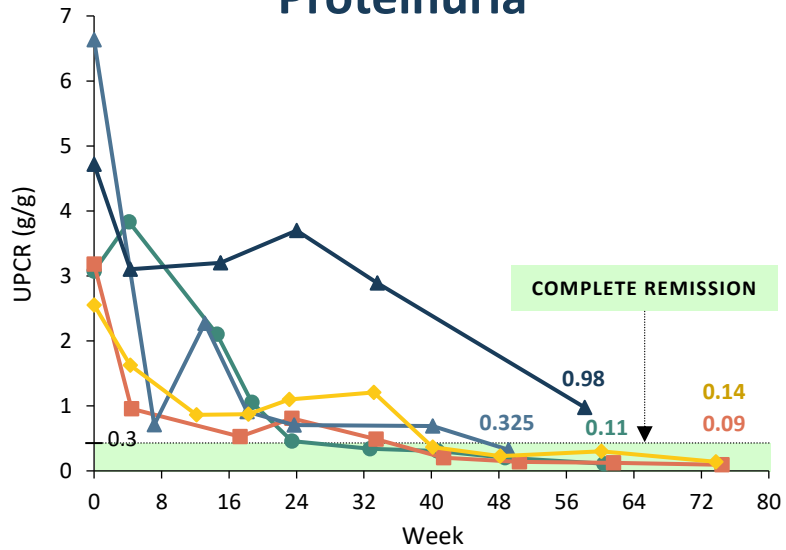
- **Rapid and complete** circulating **B cell depletion** observed in **100% (5/5) patients** at doses of 100-200 mg
- **Sustained reductions** out to 1 year + after two dose cycles

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]

Generally well-tolerated with no deaths, no drug-related SAEs, no discontinuations due to AEs, no DLTs

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AEs = adverse events, DLTs = dose limiting toxicities, LLOQ = lower limit of quantification, PLA2R = phospholipase A2 receptor, pMN = primary membranous nephropathy, SAEs = serious adverse events, 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

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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])	**Obinutuzumab** (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.

No FDA-approved therapies for pMN to date (FDA decision on obinutuzumab expected November 2026)

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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

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PrisMN: Budoprutug Phase 2 pMN Study Enrolling

Biomarker endpoint provides potential to rapidly identify dose to carry forward into Phase 3; initial data presented at ASN with additional data anticipated in 2027

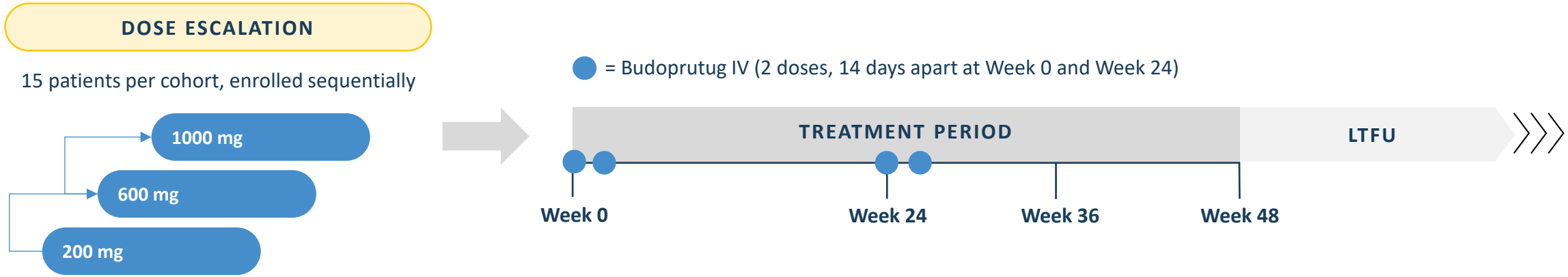
PrisMN
A NEW LENS ON PMN

Open-label, dose escalation study

Population
- 18-75 years of age
- UPCR ≥ 2.0 g/g; designed to ensure adequate enrollment of patients with UPCR > 5.0 g/g
- PLA2R antibody positive or negative if other features of chronic disease present, e.g. nephrotic range proteinuria

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)



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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
Cortazar ASN 2026

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PrisMN: Patient Demographics and Initial Safety Data

Budoprutug was generally well tolerated in the 200 mg and 600 mg cohorts, which enrolled patients with high proteinuria and PLA2R Ab levels at baseline

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	200 mg (n=14)	600 mg (n=15)
Proteinuria 24-hr UPCR, g/g, median	**6.8** range 2.8-13.4	**5.2** range 2.4-11.1 (n=11)
Anti-PLA2R RU/mL, mean	**348.4** SD ± 395.3	**244.5** SD ± 231.0 (n=9)
Prior rituximab n (%)	**4 (29%)** of 14 participants	**3 (23%)** of 13 participants

Safety

Budoprutug was generally well tolerated in the ongoing Phase 2 trial at doses of 200 mg and 600 mg

- No treatment-related SAEs or deaths reported
- Most AEs were mild to moderate (Grade 1-2)
- No dose-limiting toxicities or discontinuations due to AEs
- No hypogammaglobulinemia observed; one patient experienced two events of neutropenia in the 200 mg cohort*

*downgraded from initial SAE assignment

600 mg baseline data incomplete (UPCR n=13; anti-PLA2R n=9, one patient with anti-PLA2R <2 RU/mL; prior therapy n=13). Source: Cortazar ASN 2026

ACEi = angiotensin-converting enzyme inhibitor, ARB = angiotensin receptor blocker, eGFR = estimated glomerular filtration rate, PLA2R = phospholipase A2 receptor, UPCR = urine protein-creatinine ratio, SAE = serious adverse event, AE = adverse event

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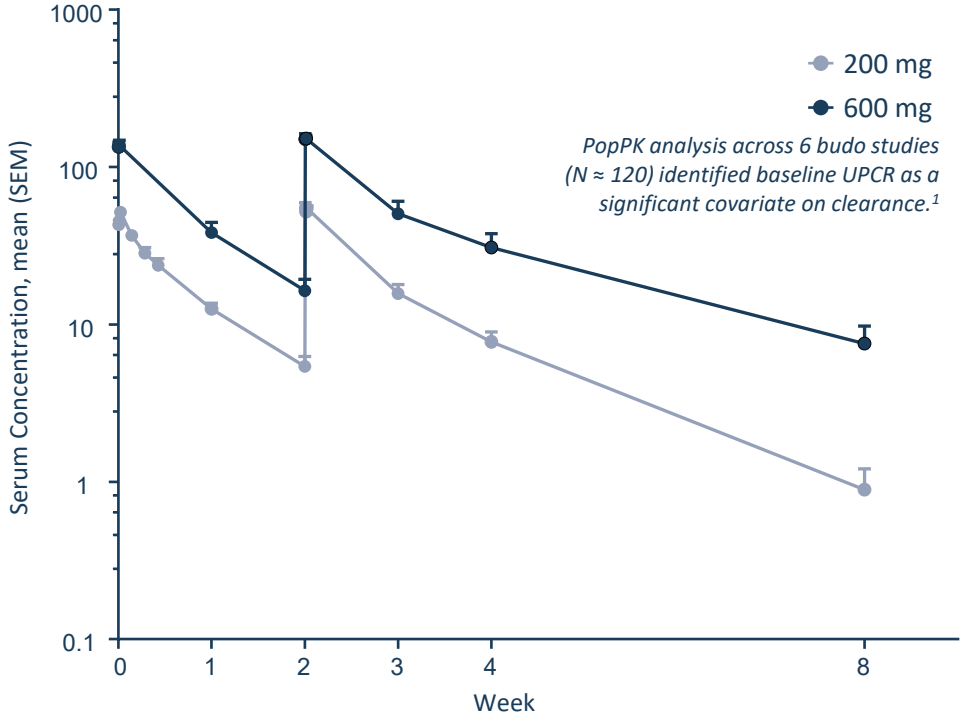
PrisMN: PK/PD Data Support Rapid, Deep B Cell Depletion

Higher rate of clearance and deeper B-cell depletion observed with 600 mg

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Serum Concentration

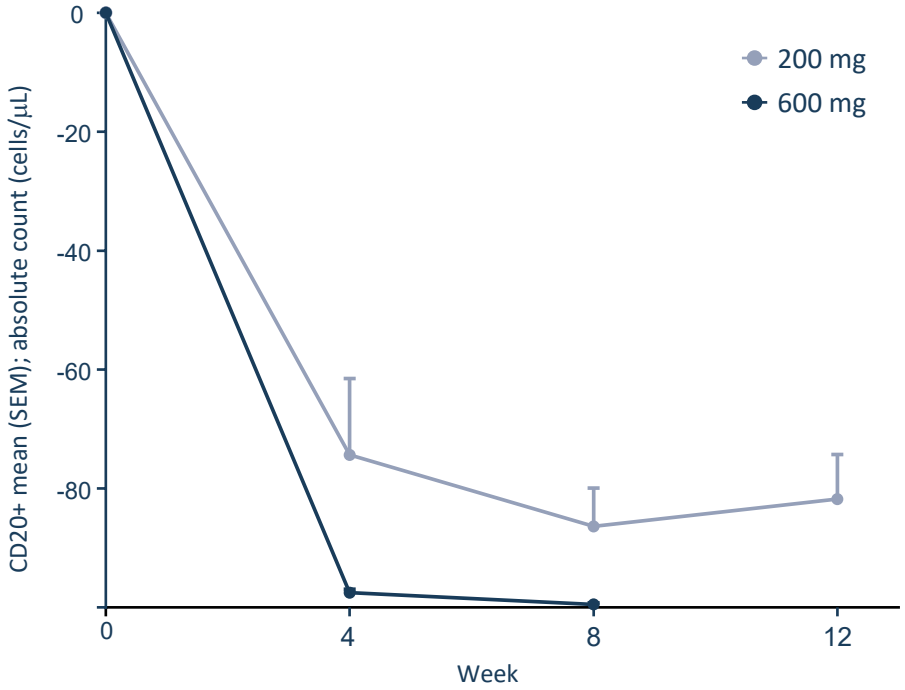
200 mg D 1, 15; 8 weeks of follow-up



- In PrisMN patients with nephrotic-range proteinuria (median baseline 24hr UPCR 5-7 g/g), **linear clearance estimated to be ≥2-fold higher than in non-proteinuric individuals**
- Findings support **evaluation of higher doses (600 / 1000 mg) in pMN** to achieve exposures comparable to those in non-proteinuric populations

Peripheral CD20+ B-cell depletion

200 mg D 1, 15; 12 weeks of follow-up



- **200 mg (n=13):** Mean 86% peripheral CD20+ B-cell depletion by Week 8
- **600 mg (preliminary, n=12 at Week 4):** 98% B-cell depletion at Week 4 compared to 74% at Week 4 with 200 mg

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[1] Data on file.

Source: Cortazar ASN 2026. PK = pharmacokinetics, PopPK = population PK, UPCR = urine protein-creatinine ratio

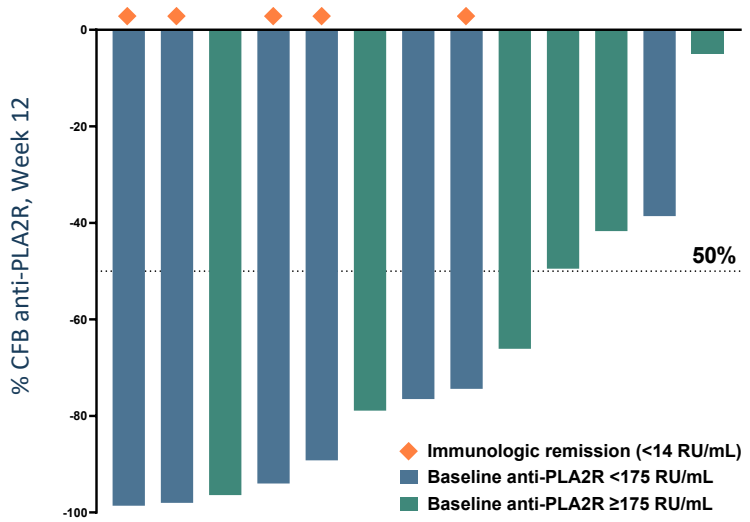
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PrisMN: Encouraging Reductions in Anti-PLA2R

Low-dose budo demonstrated promising impact on anti-PLA2R Ab levels and early positive signal for 24hr UPCR at Week 12

Anti-PLA2R at Week 12

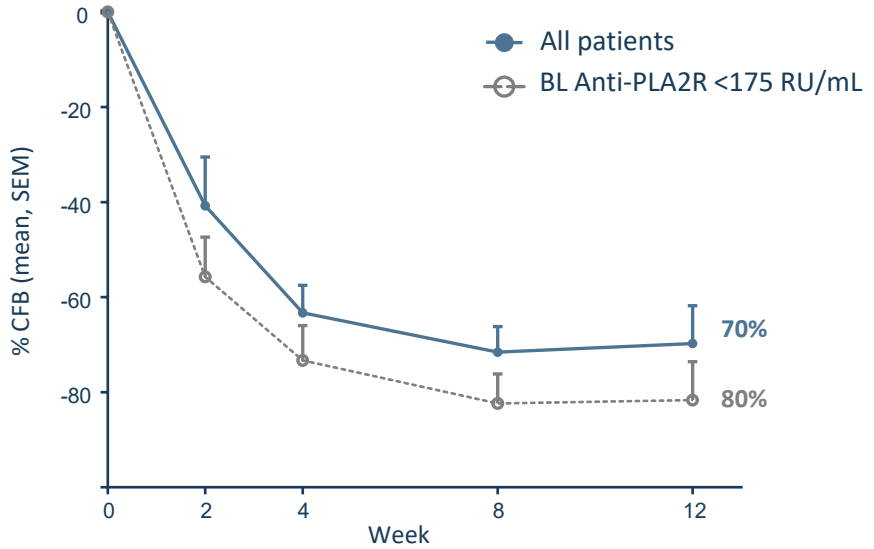
%CFB, individual participants



- Immunologic remission achieved in 5/13 (38%) overall and in 5/7 (71%) of pts with <175 RU/mL anti-PLA2R at BL

Anti-PLA2R

% CFB mean (SEM)



- ≥50% reduction in anti-PLA2R observed in 69% of pts overall, and in 86% of pts with <175 RU/mL anti-PLA2R at BL

24hr UPCR at Week 12: ≥25% reduction observed in 5/12 (42%) overall and in 3/7 (43%) with anti-PLA2R <175 RU/mL at BL

Immunologic remission = <14 RU/mL

N=13 for all time points for anti-PLA2R; N=12 for 24hr UPCR (one patient is missing baseline 24hr UPCR); BL = baseline; CFB = change from baseline; 200 mg D 1, 15; 12 weeks of follow-up

Source: Cortazar ASN 2026

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Low-Dose Budoprutug Week 12 PLA2R Rivals Obinutuzumab RP3D

At just 200 mg, budoprutug reduced anti-PLA2R by 70% at Week 12, in line with prior Phase 1b results, and within range of other agents at Phase 3 or expected labeled doses (−22% to −85%)

Program	Population	N	Dose studied	Mean baseline PLA2R (RU/mL)	Mean change in anti-PLA2R Week 12	Immunologic remission* Week 12
Budoprutug[1] (anti-CD19) PrisMN Ph2	**Anti-PLA2R+**	**13**	**LOW DOSE 200 mg IV** D1, D15, W24, W26	**348**	**-70%**	**38%**
	PLA2R <175	*7*		*93*	*-80%*	*71%*
Obinutuzumab[2] (anti-CD20) Ph3	Anti-PLA2R+	54	EXP. LABEL DOSE 1000 mg IV D1, W2, W24, W26	263	-85%	72%
	PLA2R <175	*24*		*49*	*NR at Week 12*	*96%*
Povetacicept[3] (BAFF/APRIL) Ph1/2	Anti-PLA2R+	10	PHASE 3 DOSE 80 mg SC Q4W	140	-73%	44%
Felzartamab[4] (anti-CD38) Ph1/2	Anti-PLA2R+	26	PHASE 3 DOSE 16 mg/kg IV ×9 over 24 wk	247	-22%**	NR at Week 12

Budoprutug **PLA2R <175 subgroup** **Other agents**

*Immunologic remission = anti-PLA2R <14 RU/mL among patients ≥14 RU/mL at baseline; **Weighted average

Data 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.

PrisMN Low-Dose Highlights†

200 mg Budoprutug **low-dose** cohort; two infusions (D1, D15) before the Week 12 readout

−70% **Anti-PLA2R at Week 12**, on par with povetacicept Phase 3 dose (−73%) and >3× felzartamab (−22%)

71% **Immunologic remission** at Week 12 in patients with BL PLA2R <175; 38% across all anti-PLA2R+

† Low-dose results in line with prior Phase 1b, which demonstrated 75% reduction in PLA2R and 33% immunologic remission at Week 12[1,5]

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BL = baseline, EXP = expected, NR = not reported; PLA2R = phospholipase A2 receptor, RP3D = recommended Phase 3 dose;
[1] Cortazar ASN 2026, [2] Fervenza NEJM 2026, [3] Madan KI Rep 2026, [4] Rovin KI Rep 2024, [5] Company data on file

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PrisMN: Encouraging Preliminary Data at Lowest Dose

Enrollment and follow-up are ongoing; additional data expected in 2027

PrisMN
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SAFETY

Budoprutug has been generally well tolerated at doses of up to 600 mg

BIOMARKERS

Promising PLA2R Ab reduction and immunologic remission rate consistent with Phase 1b observations at Week 12

DOSING

Dose dependent increases in exposure and B-cell depletion observed; confirmed higher clearance in proteinuric population justifies exploration of higher doses

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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

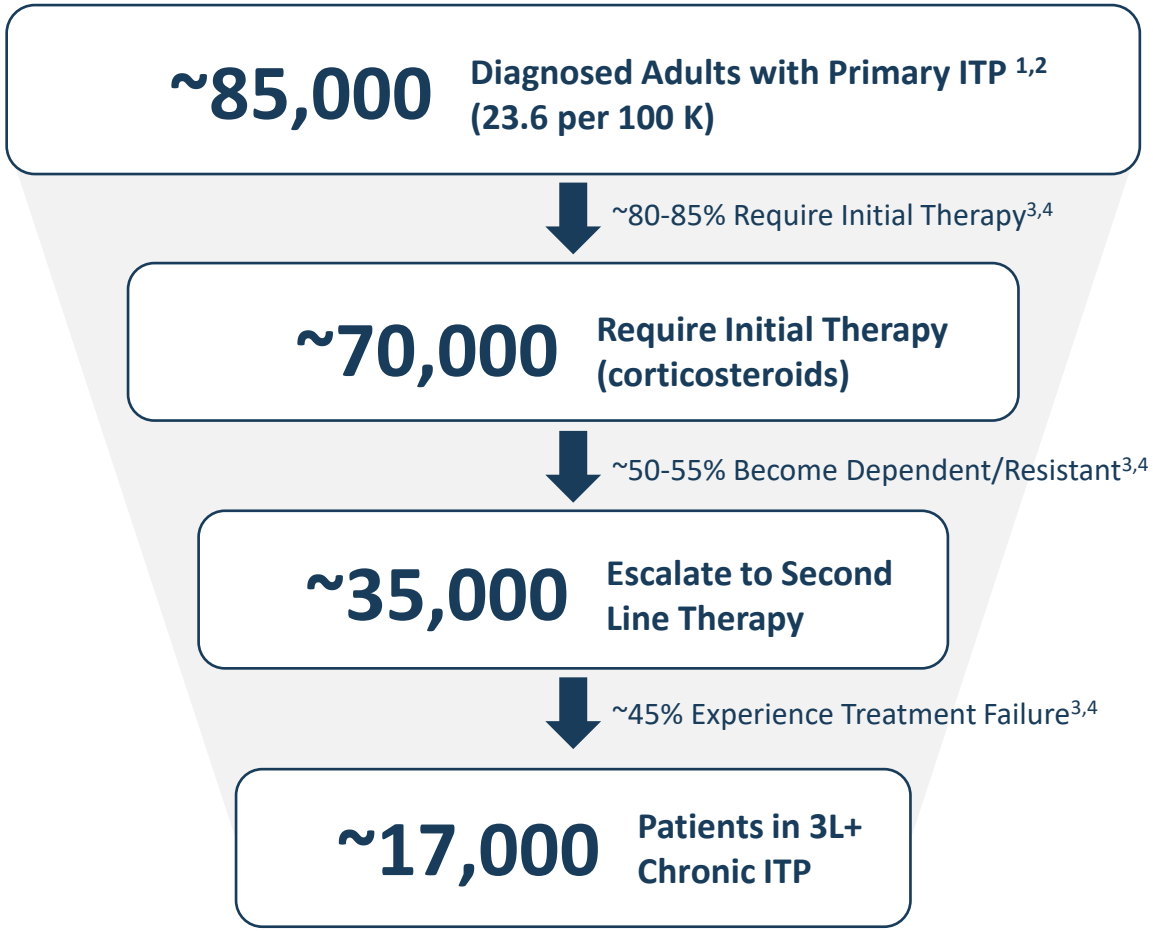
ITP = immune thrombocytopenia; 1L = first line; 2L = second line; 3L = third line

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

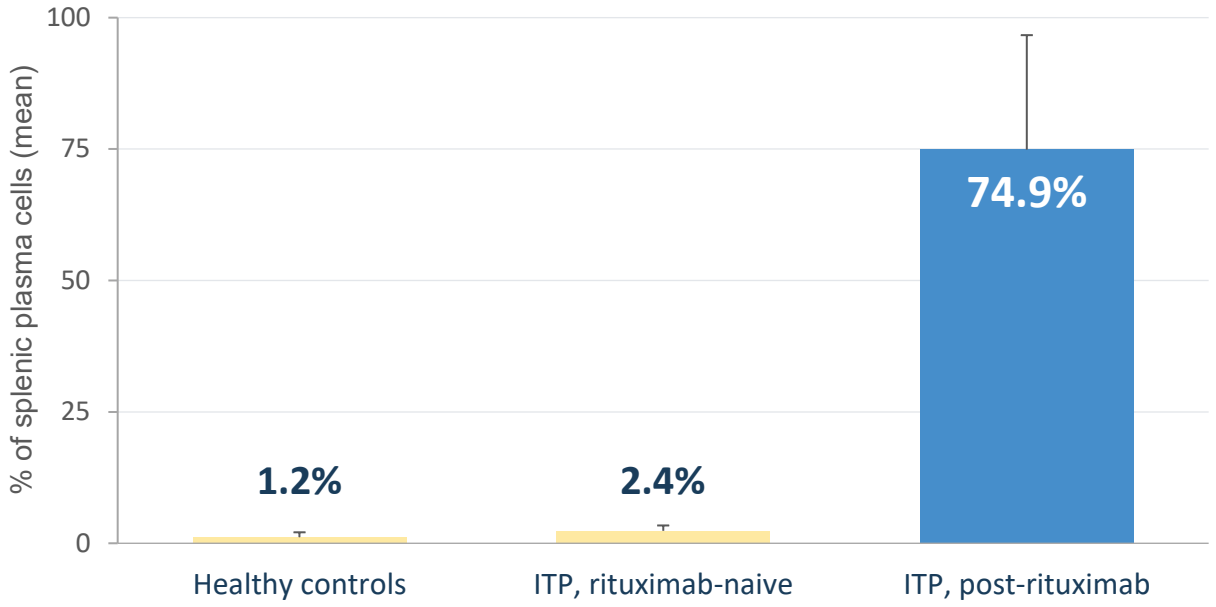
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Strong Rationale for CD19 Approach in ITP

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

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Budoprutug Phase 1b ITP Trial Ongoing

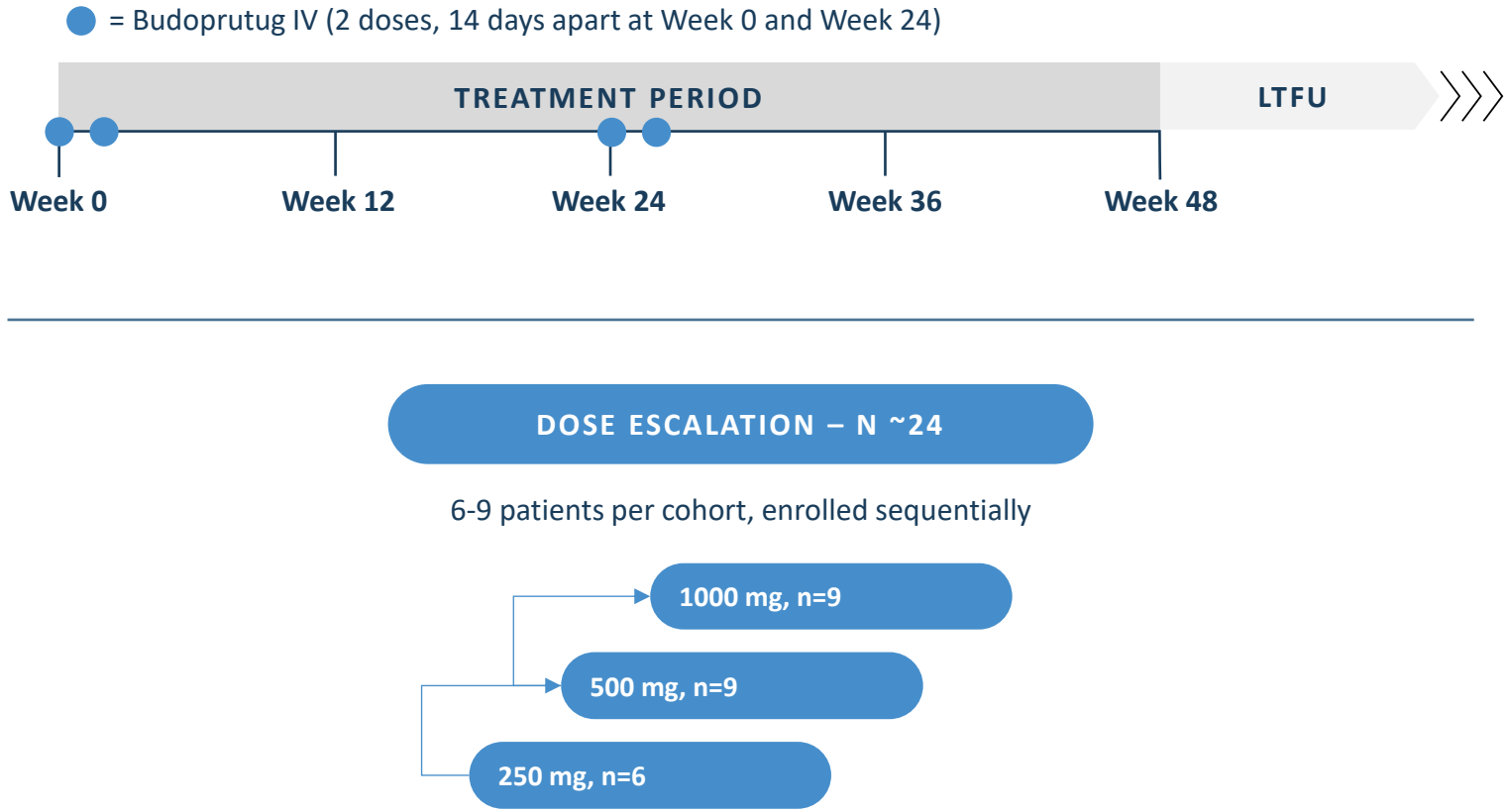
Designed to define dose and regimen, and establish depth and duration of platelet response and B cell depletion; additional data from 500 and 1000 mg cohorts expected in Q4 2026

Open label, dose escalation and expansion study

Population
- N ~24 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

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ITP Phase 1b: 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

	250 mg (n=6)	500 mg (n=9)
Prior lines of therapy Median	**7.5** range 4-18	**6** range 2-15
Prior rituximab n (%)	**4 (67%)** of 6 participants	**2 (22%)** of 9 participants
Baseline platelets $\times 10^3/\mu L$, median	**13** range 4-29	**7** range 2.5-12

Also at baseline: median disease duration 6.5 (250 mg) / 15 years (500 mg); prior TPO-RA 4 patients (250 mg) / 7 patients (500 mg); 1 prior splenectomy (500 mg)

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

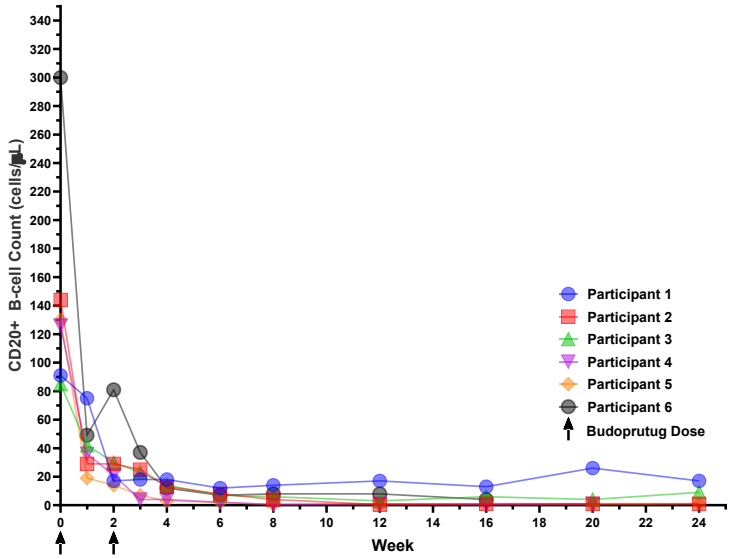
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Clinical Proof-of-Concept Demonstrated in ITP Phase 1b Cohort 1

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

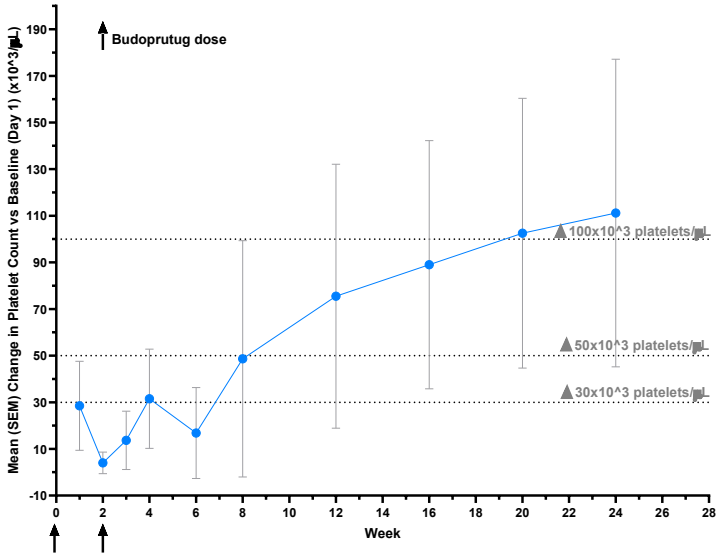
B-Cell Depletion



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

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Systemic Lupus Erythematosus

Marisa, living with SLE



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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.

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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 approved for LN; rituximab 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

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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

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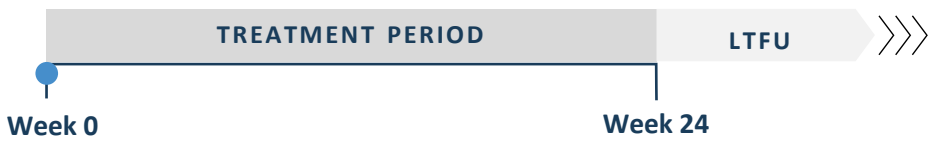
Parallel Global and China SLE Studies Enrolling

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

Global Phase 1b Study

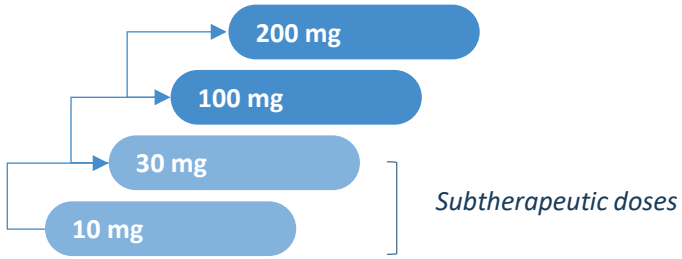
Enrolling adults with seropositive SLE; SLEDAI ≥ 8; refractory to adequate trials of at least 2 therapies

● = Budoprutug IV Day 1



SINGLE ASCENDING DOSE COHORTS, N ~30

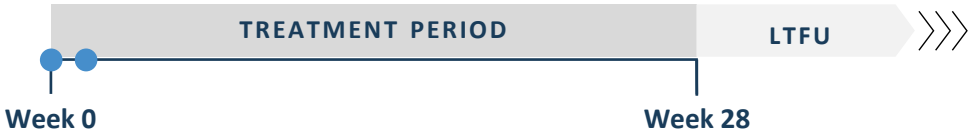
Up to 6 patients per cohort enrolled sequentially



China Phase 1b/2a Study

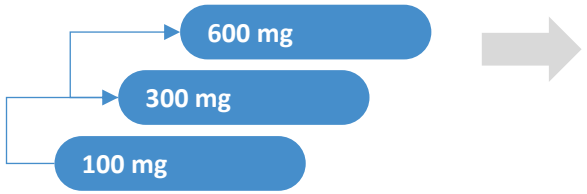
Enrolling adults with seropositive SLE; SLEDAI ≥ 6; refractory to adequate trial of at least 1 therapy

● = Budoprutug IV Day 1 and Day 15



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

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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

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Budoprutug – Subcutaneous (SC) Formulation

Anti-CD19

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Budoprutug SC Formulation Provides Optionality and Differentiation

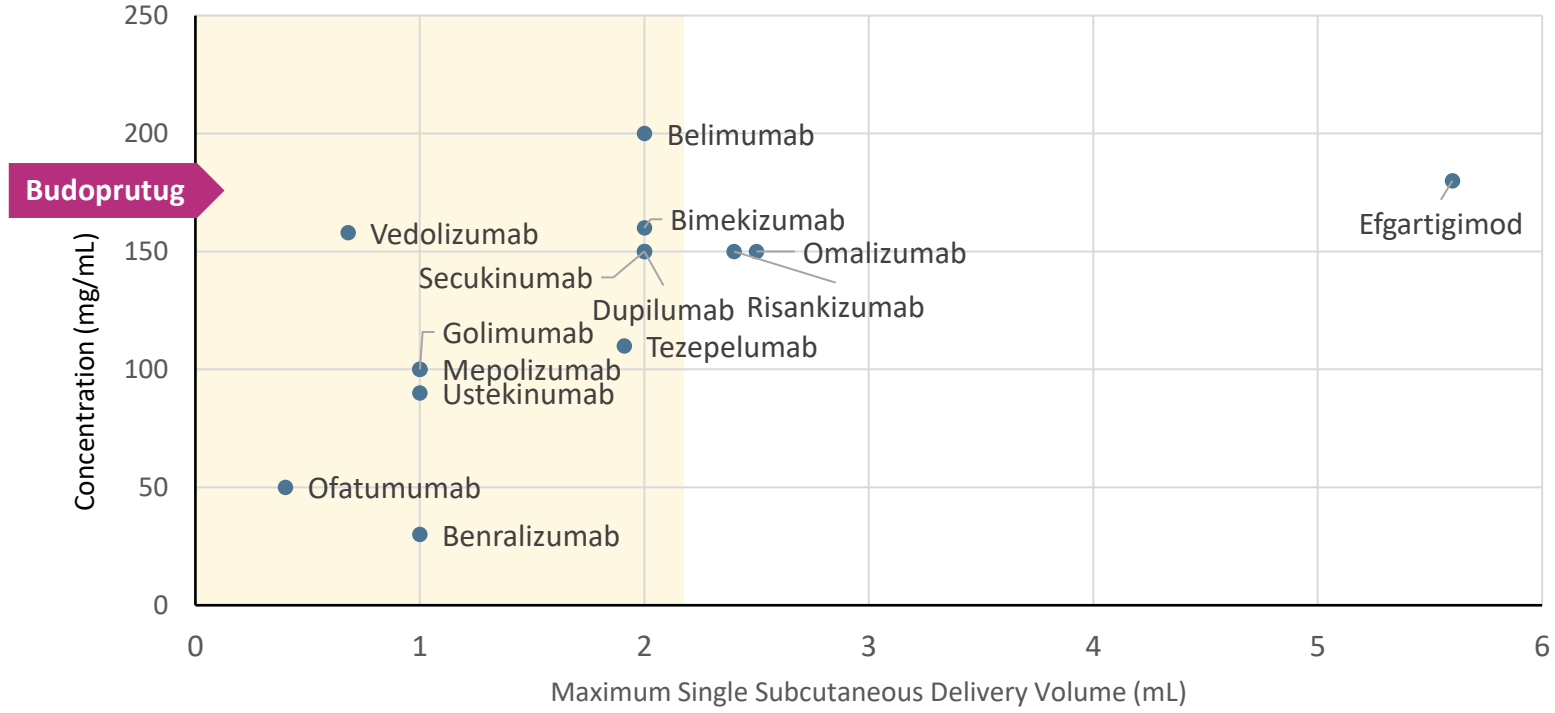
Ability to formulate as both IV and SC provides optionality in development and may be a differentiating feature of budoprutug relative to other anti-CD19 mAbs

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



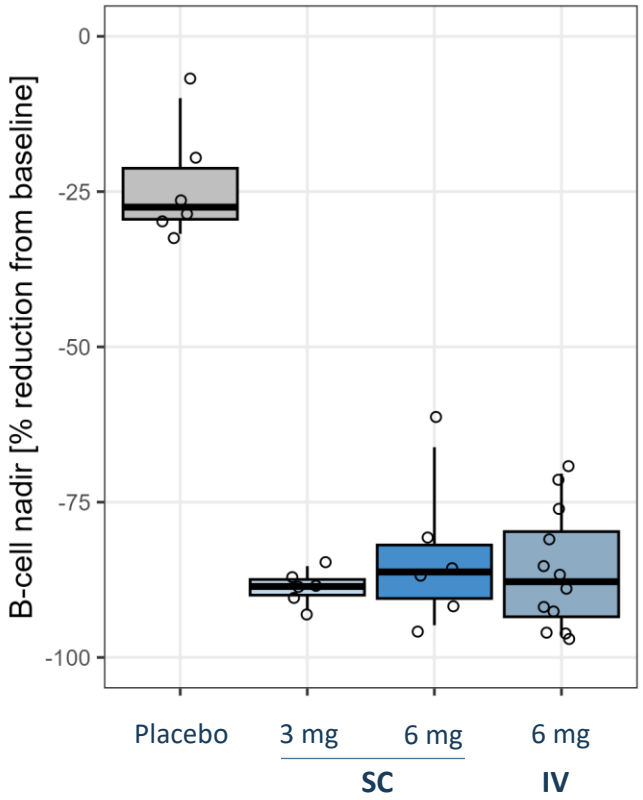
IV = intravenous, mAbs = monoclonal antibodies, SC = subcutaneous

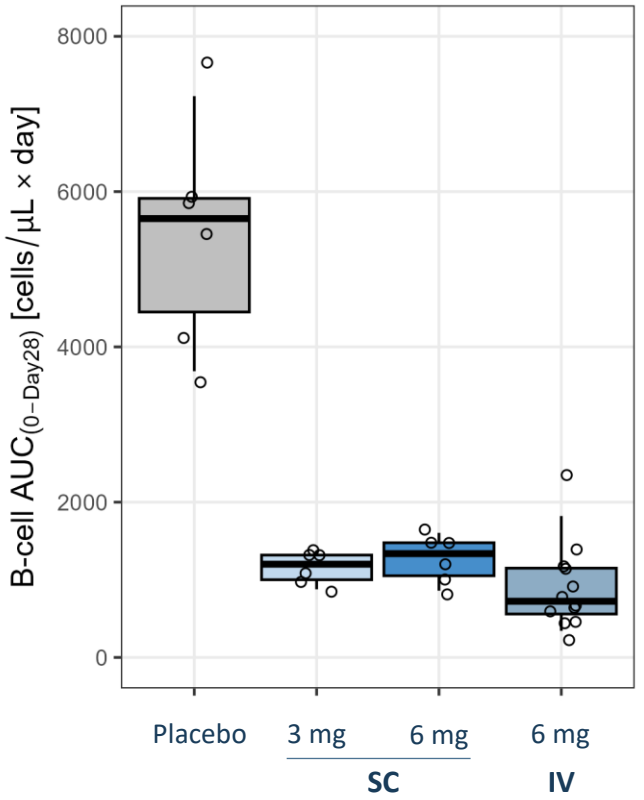
Source: FDA-approved labels. Concentration normalized to mg/mL; where multiple indications or dosing regimens are approved, the highest volume single dose was used

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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





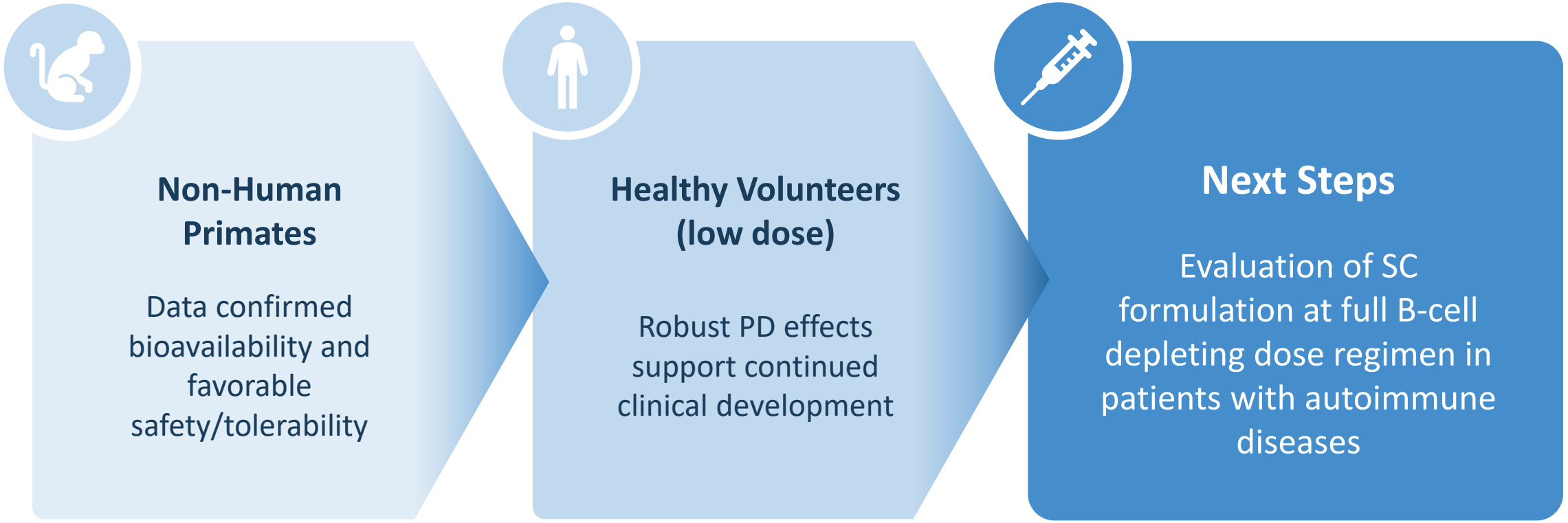
Budoprutug SC was well-tolerated; safety profile consistent between SC and IV

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.

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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 with initial data expected in 2027



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

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Looking ahead

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Climb Bio: Continuing the Ascent Into 2027

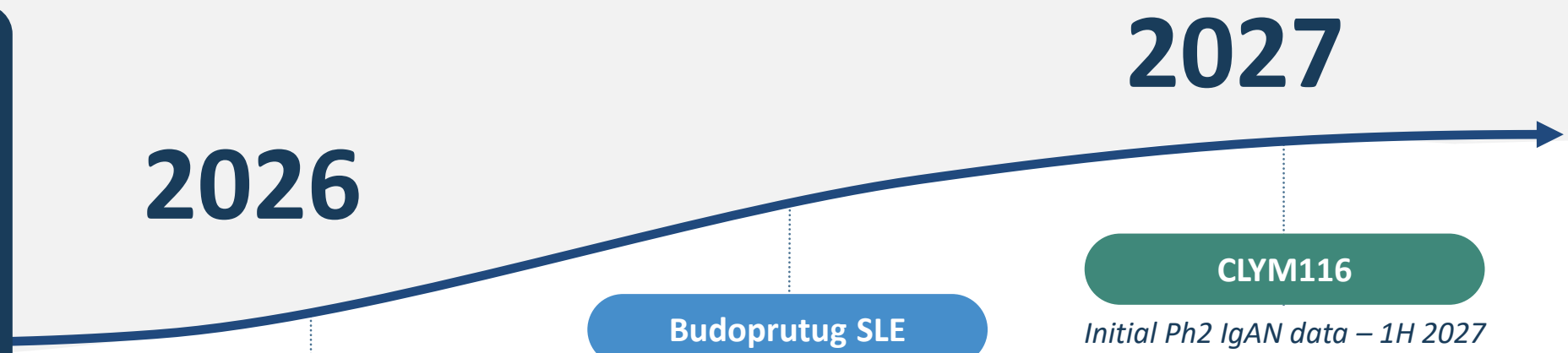
Numerous milestones across both assets through 2027, funded into 2H 2028

$239.2M*

RUNWAY INTO 2H 2028

79.8M†

FULLY DILUTED SHARES OUTSTANDING



2026

CLYM116 ✓

Additional Ph1 HV data – Q4

Budoprutug pMN ✓

Initial Ph2 data – Q4
B cell and PLA2R, low dose cohort

Budoprutug SLE

Initial global Ph1b data – Q4
safety & biomarkers, low doses

Budoprutug ITP

Additional Ph1b data – Q4

2027

CLYM116

Initial Ph2 IgAN data – 1H 2027

Ph3 initiation, pending regulatory feedback – 2027

Budoprutug

pMN all dose cohorts and CR data – 2027

Initial Ph1b SC data – 2027

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*Cash, cash equivalents, and marketable securities as of June 30, 2026; †57.3 million common shares outstanding as of June 17, 2026

HV = healthy volunteers, IgAN = IgA nephropathy, ITP = immune thrombocytopenia, pMN = primary membranous nephropathy, SC = subcutaneous, SLE = systemic lupus erythematosus

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