

Corporate Presentation

AUGUST 2026



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; 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 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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OUR MISSION

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



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 the second half of 2028**

Climb Bio: Data-Rich 2026

Continuing the ascent with initial readouts anticipated from all ongoing trials

2026



Budoprutug SC

*Initial Ph1 HV data – May
PK, PD, bioavailability*



Budoprutug ITP

*Initial Ph1b data – June
B cell and platelets, low dose cohort*



Budoprutug SLE

FPI China Ph1b – Q2

CLYM116

FPI Ph2 – Q3

CLYM116

*Initial Ph1 HV data – Sept
PK, PD*

Budoprutug pMN

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

CLYM116

Additional Ph1 HV data – Q4

Budoprutug SLE

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

Budoprutug ITP

Additional Ph1b data – Q4

Pipeline of Highly Differentiated mAbs

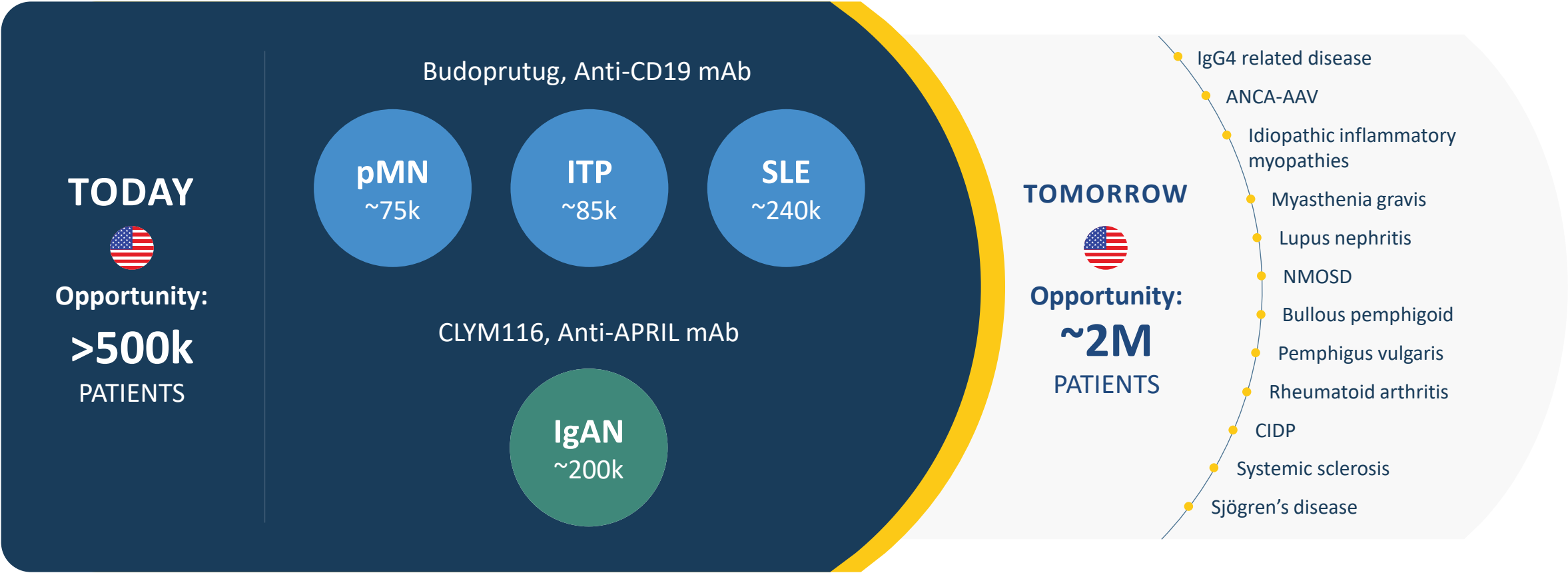
Anticipating initial readouts from all ongoing trials in 2026

	INDICATION(S)	PRECLINICAL	PHASE 1	PHASE 2	PHASE 3	ANTICIPATED MILESTONES	CLIMB RIGHTS
Budoprutug IV Anti-CD19	Primary Membranous Nephropathy					Initial Ph2 data Q4 '26	Worldwide
	Immune Thrombocytopenia					Additional Ph1b data Q4 '26	
	Systemic Lupus Erythematosus					Initial Global Ph1b data Q4 '26	
Budoprutug SC Anti-CD19	Autoimmune Disease					Initiate multiple dose patient study Initial Ph1b data 2027	Worldwide
CLYM116 Anti-APRIL	IgA Nephropathy					Initial PK/PD Ph1 HV data Sept '26 FPI in Ph2 Q3 '26 Additional Ph1 data Q4 '26	Worldwide Outside Greater China*

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

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

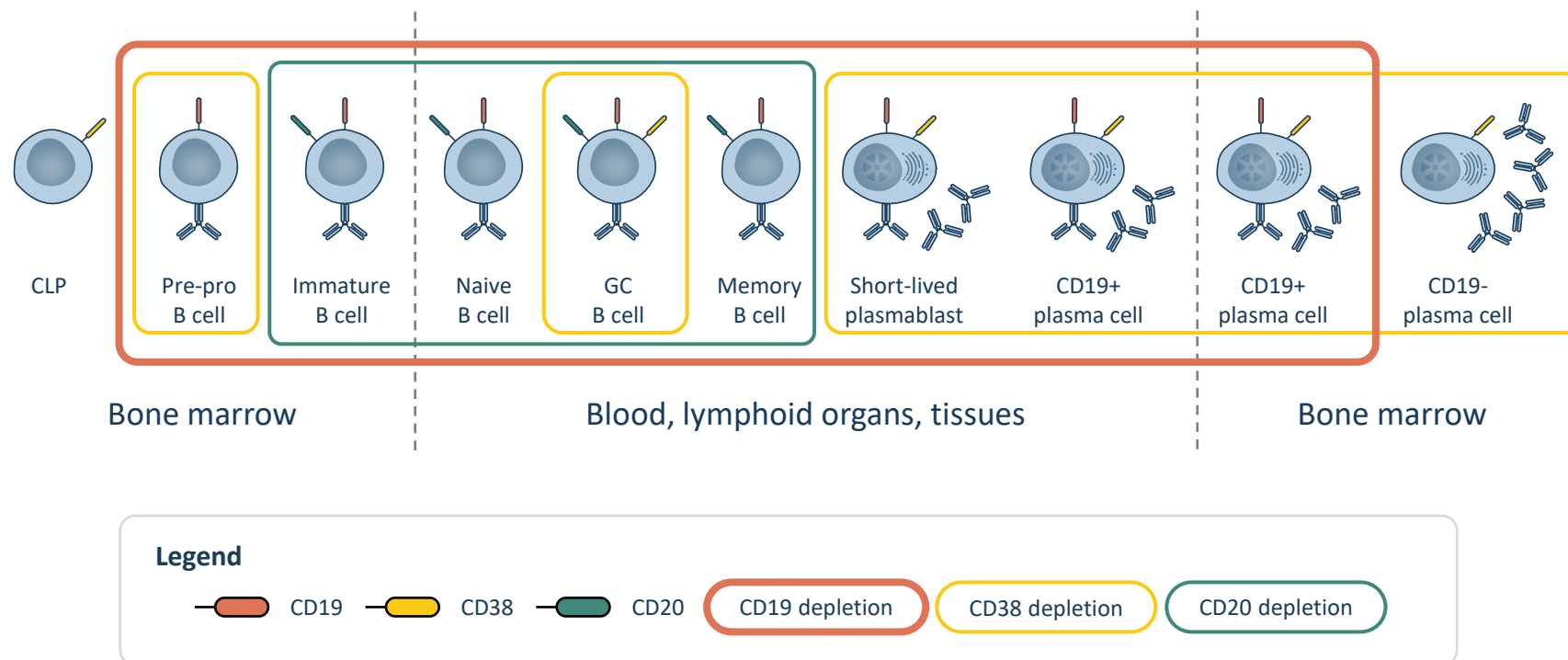
Clear Opportunity

- **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+

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 autoantibodies¹

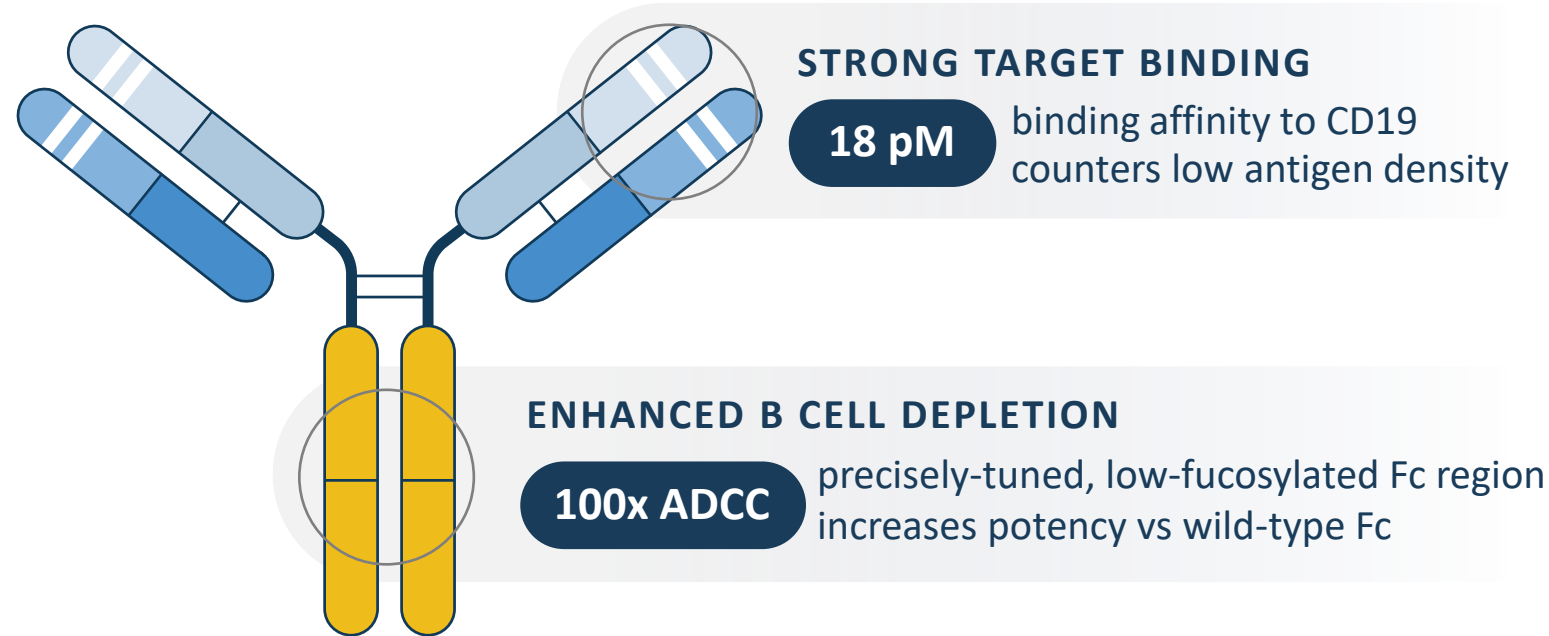


- Naked mAbs targeting CD20 have been shown to deplete B cells in tissue²
- Deeper depletion may be achieved by targeting CD19

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



SUBCUTANEOUS DOSING POTENTIAL

≥175 mg/mL

High concentration
formulation with low
viscosity

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 ~75,000 patients^{1,2} No approved therapies	Chronic bleeding disorder characterized by the destruction of platelets.⁶ ~85,000 patients^{2,3} Poor QoL, with majority of previously treated patients failing to achieve durable platelet response⁶	Chronic autoimmune condition with severe disease manifestations that can affect virtually any organ system.⁷ ~240,000 patients^{2,4} Majority of patients fail to achieve disease control with existing treatments⁷
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

BUDOPRUTUG OPPORTUNITY

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

Primary Membranous Nephropathy

Seferiana, living with pMN



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

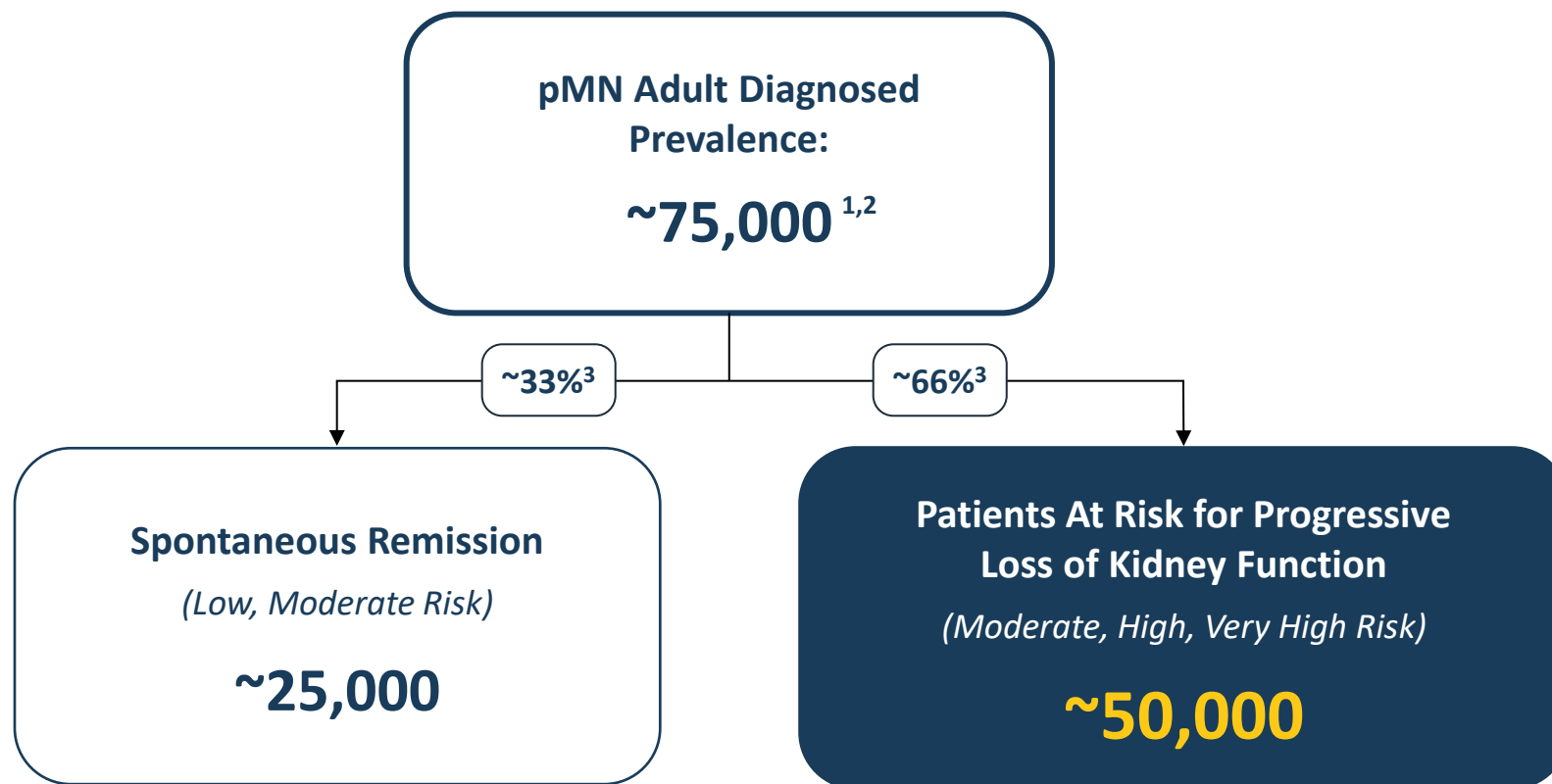
~**75,000** patients
in the US alone

THE CLIMB SOLUTION

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

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



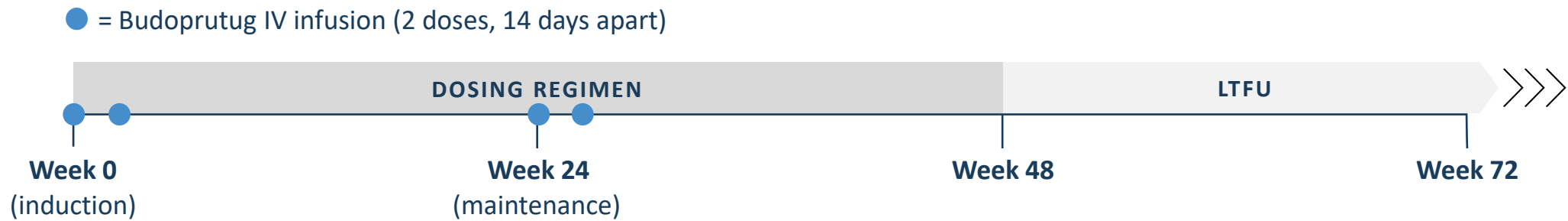
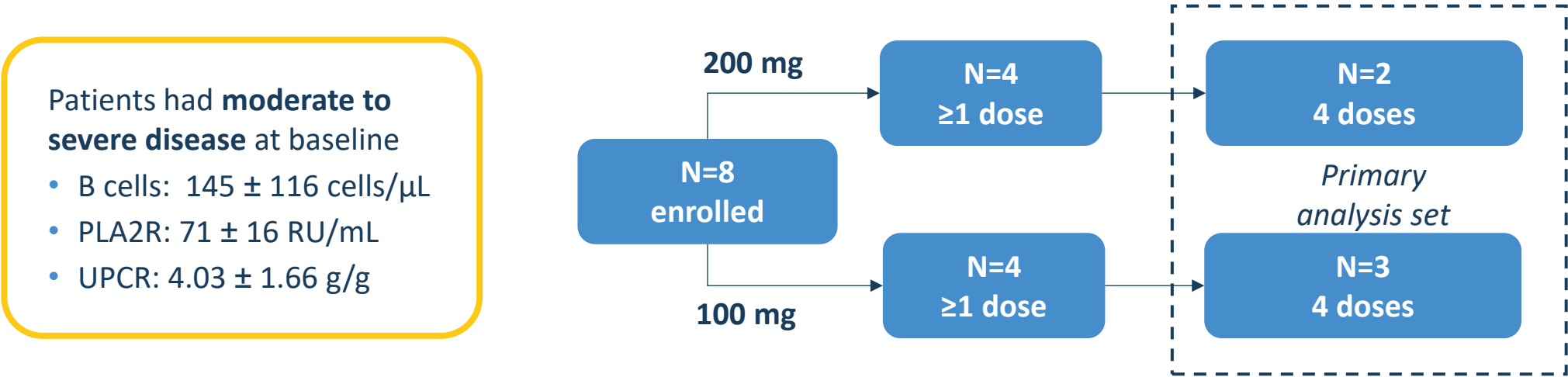
KDIGO Recommendation⁴

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

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

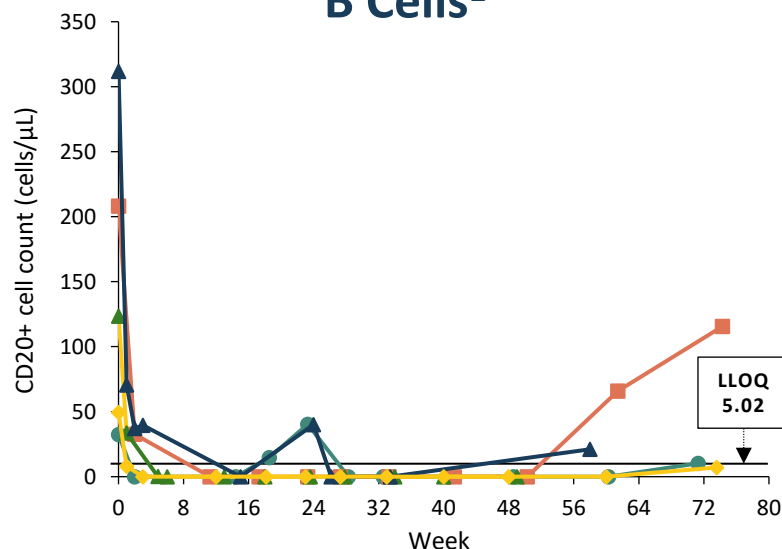
Budoprutug evaluated in an open-label, dose escalation study in adult patients with pMN



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

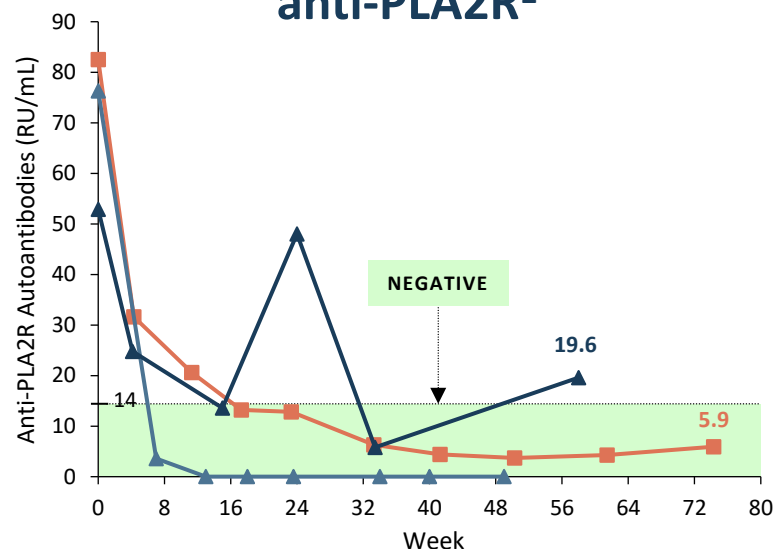
Budoprutug administration resulted in durable B-cell depletion, serologic remission, and clinical remission (as measured by proteinuria)

B Cells¹



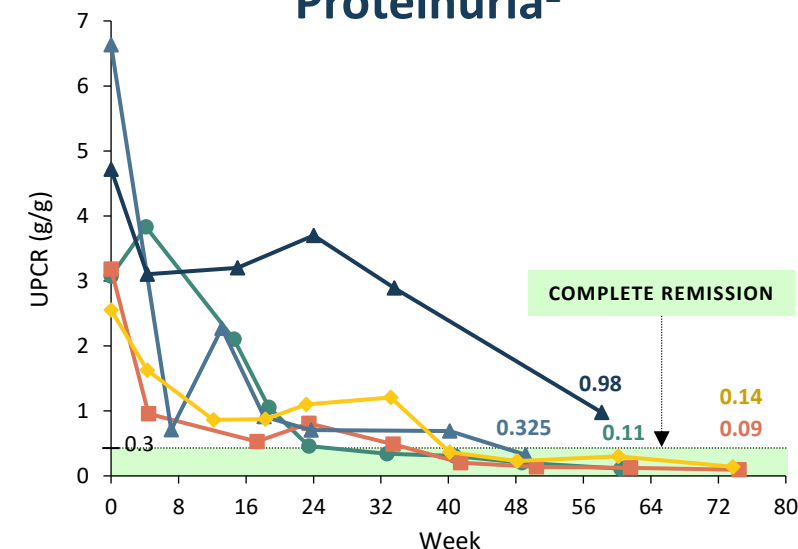
- **Rapid and complete circulating B cell depletion** observed in **100% (5/5) patients** at doses of 100-200 mg
- **Sustained reductions** out to 1 year⁺ after two dose cycles

anti-PLA2R¹



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

Proteinuria¹



- 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 doses²



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

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

PrisMN: Budoprutug Phase 2 pMN Study Enrolling



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 $\geq$ 2.0 g/g; designed to ensure adequate enrollment of patients with UPCR $>$ 5.0 g/g
- PLA2R antibody positive

Primary Objective

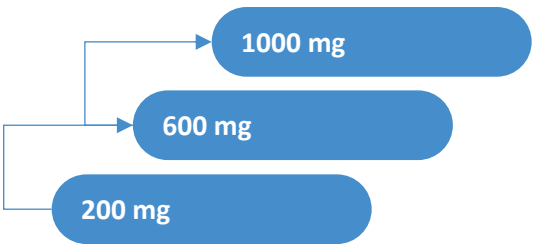
- Safety and tolerability

Secondary Objectives

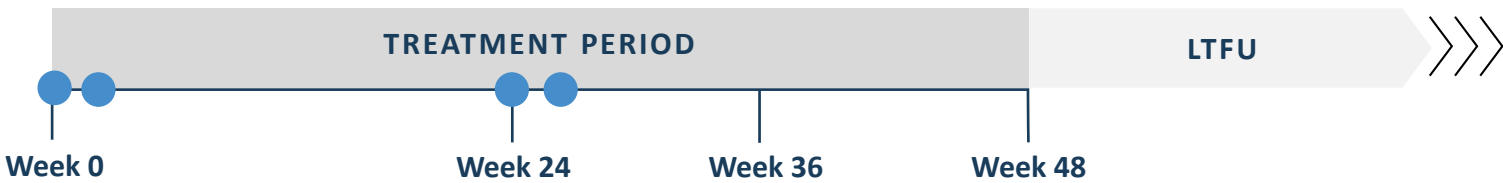
- Preliminary PK and PK/PD
- PD markers (B cells, anti-PLA2R, total Ig)
- Preliminary efficacy: complete and partial remission at week 48 (UPCR and eGFR)

DOSE ESCALATION

15 patients per cohort, enrolled sequentially



● = Budoprutug IV (2 doses, 14 days apart at Week 0 and Week 24)



Immune Thrombocytopenia

John, living with ITP



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

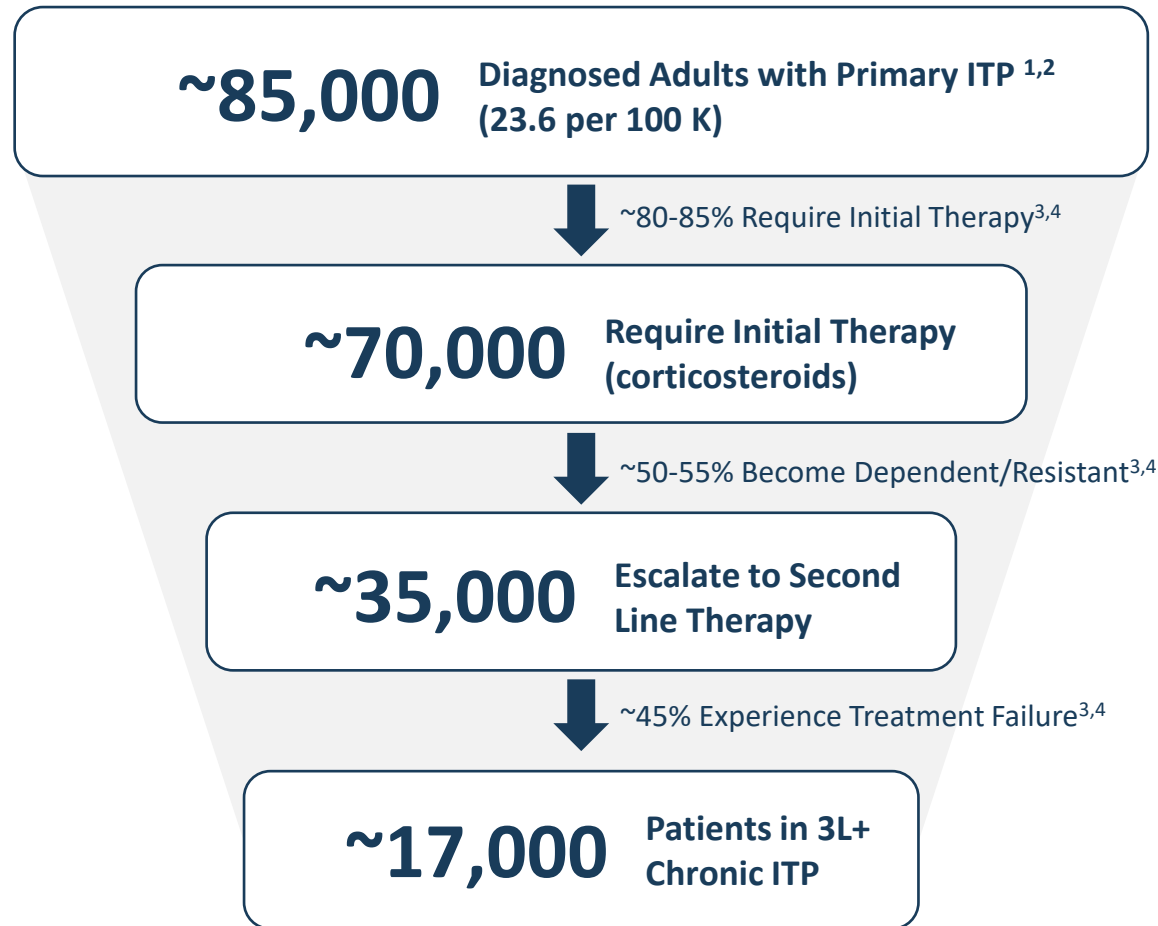
~85,000 patients
in the US alone

THE CLIMB SOLUTION

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

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

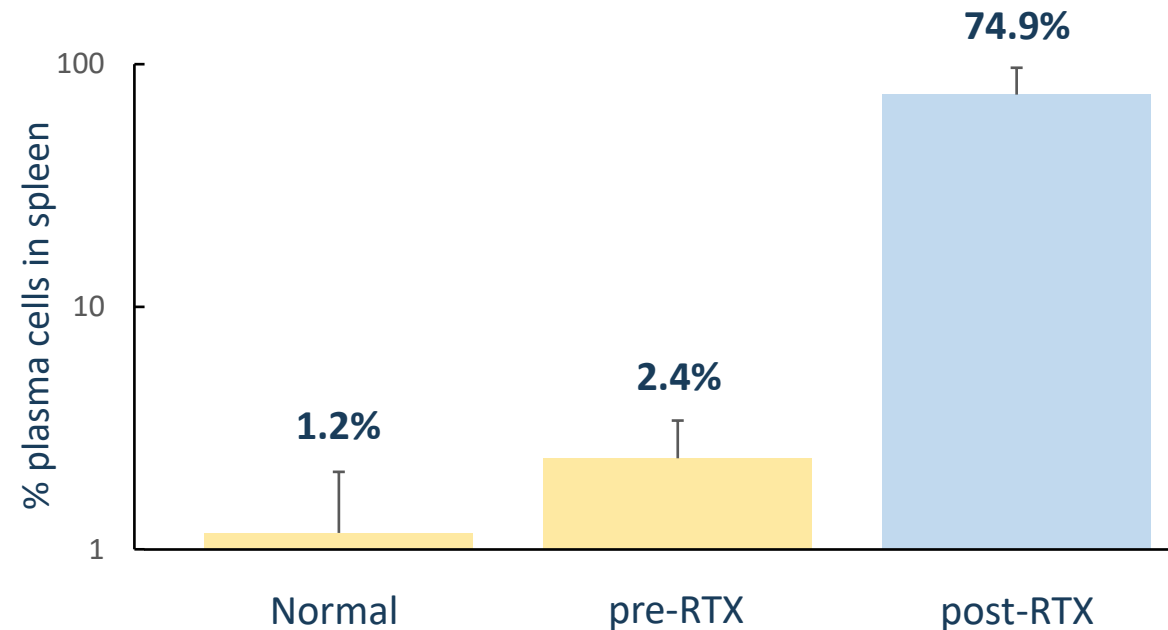
***Later-line ITP represents
a high-value opportunity for
disease-modifying therapies***

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 treatment¹



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

Budoprutug Phase 1b 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

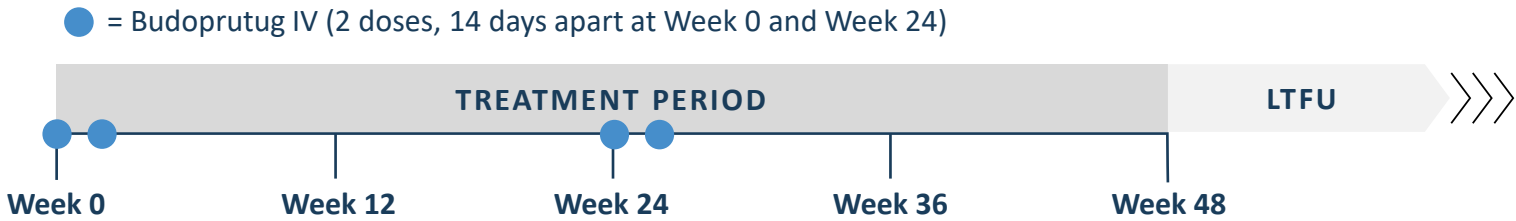
- N ~24 patients
- Insufficient response to 1 or more prior therapies
- Platelet count <30,000/ μ L

Primary Objective

- Safety and tolerability

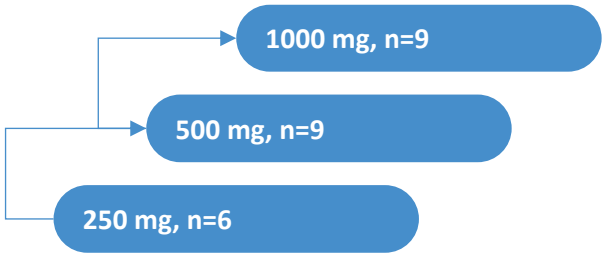
Secondary Objectives

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



DOSE ESCALATION – N ~24

6-9 patients per cohort, enrolled sequentially



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)
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

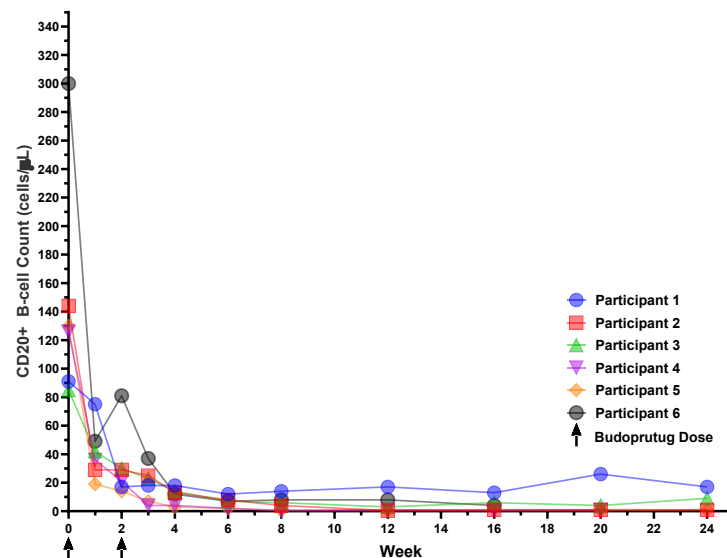
- ✓ No SAEs or deaths reported
- ✓ All AEs were Grade 1 or Grade 2; majority of AEs belonged to the gastrointestinal system organ class
- ✓ No discontinuations due to AEs
- ✓ No infusion-related reactions
- ✓ No participants had hypogammaglobulinemia

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

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)

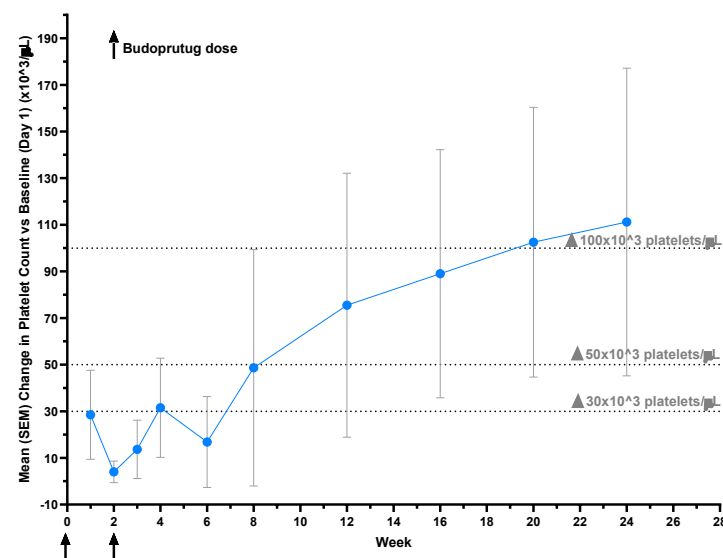
B-Cell Depletion



High sensitivity assay, LLOQ of 0.4 cells/ μ L

Platelet Response Assessments

At **Week 24**, mean (SEM) increase in platelet count from baseline was **111 (66) $\times 10^3$ platelets/ μ L**



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 $\geq 100 \times 10^3/\mu$ L 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: $\geq 30 \times 10^3/\mu$ L and $\geq 2 \times$ BL at 6 months; Complete Response: $\geq 100 \times 10^3/\mu$ L 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.

Systemic Lupus Erythematosus

Marisa, living with SLE



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

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/LN¹, 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

Budoprutug – Subcutaneous (SC) Formulation

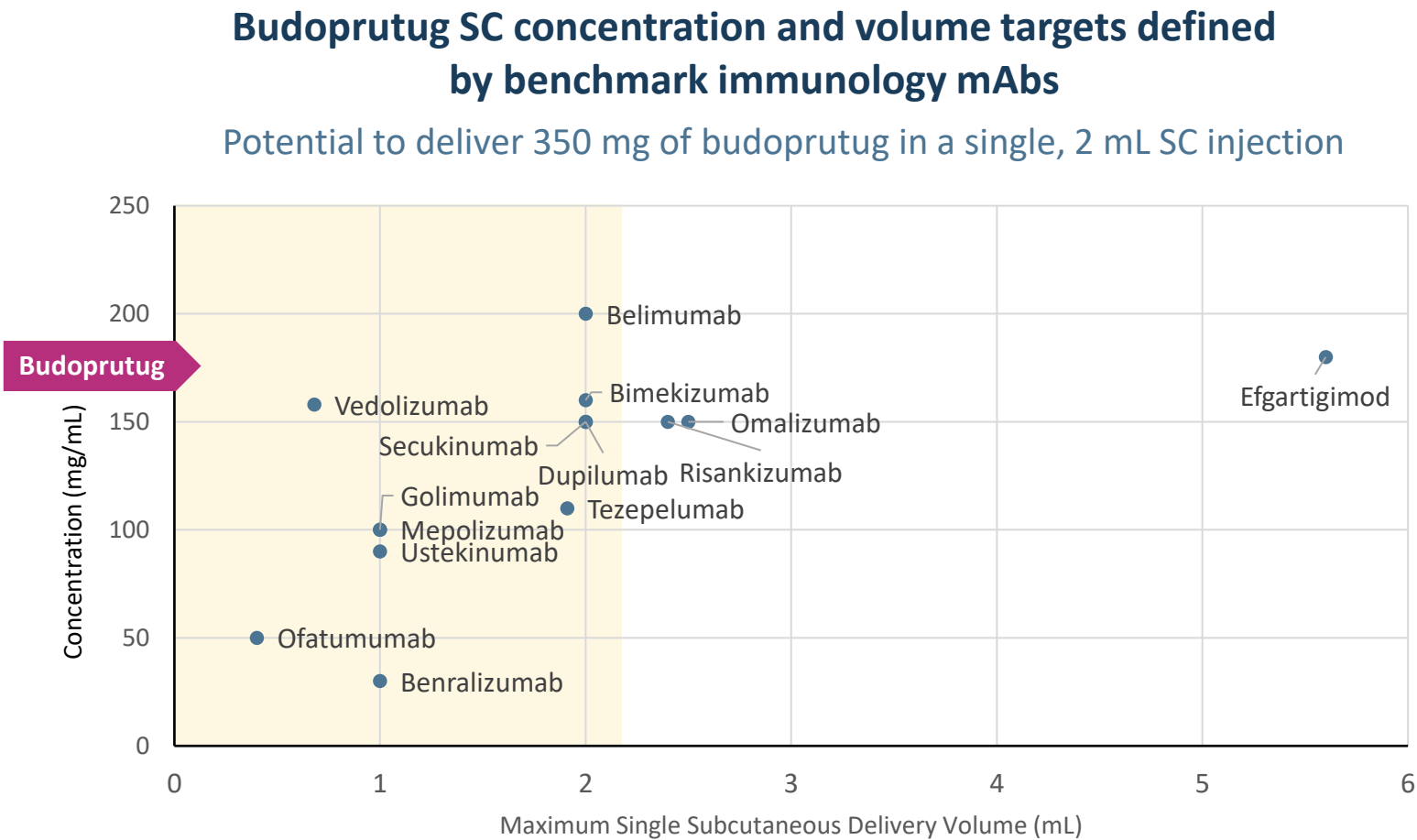
Anti-CD19

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

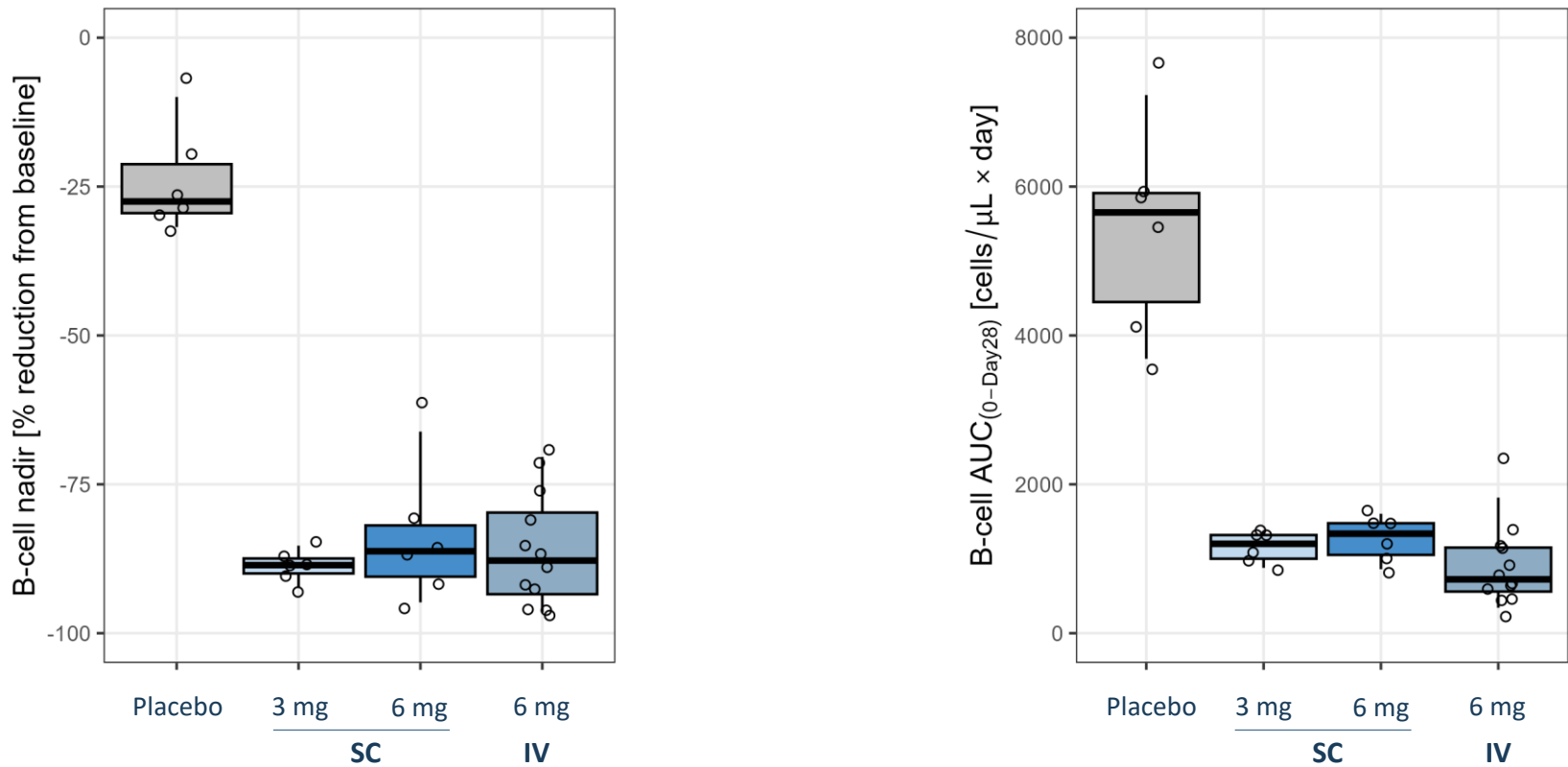
Potential Value of SC Administration

- 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



Robust B-cell Depletion Observed with Budoprutug SC in HV

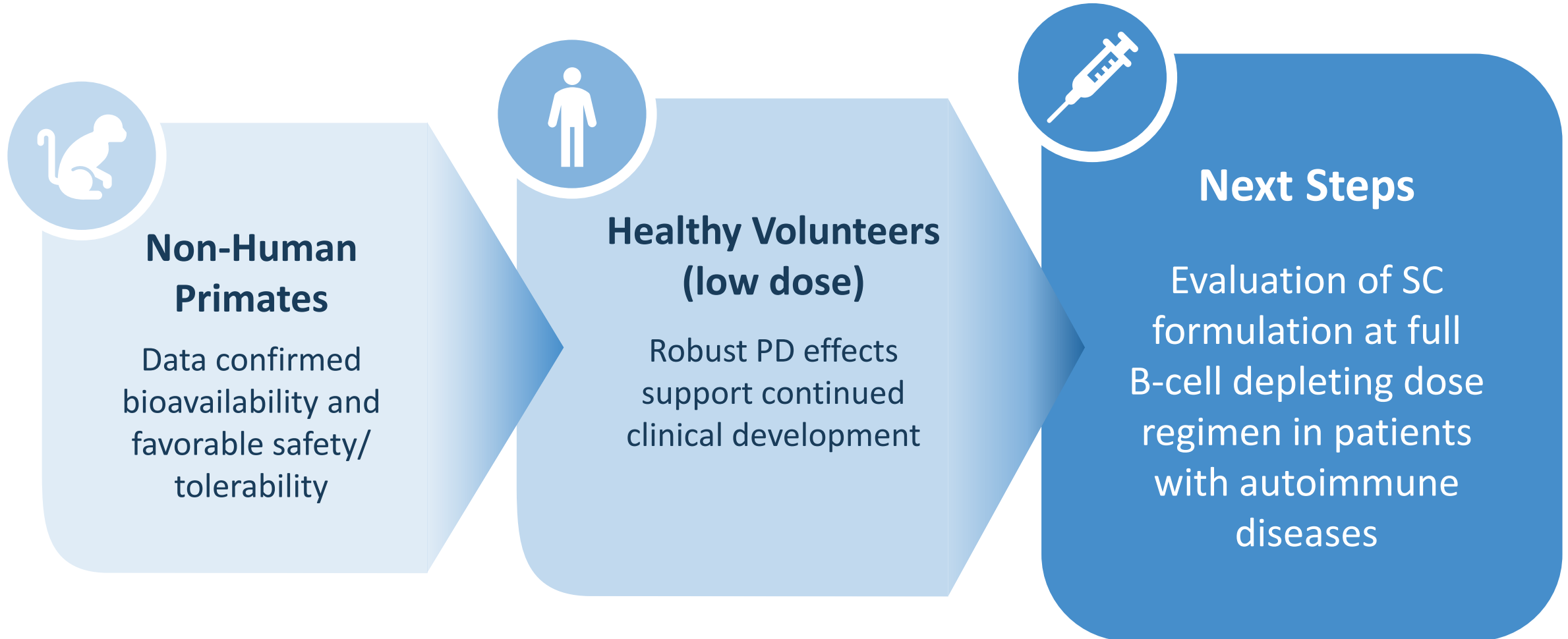
Budoprutug SC administration demonstrated ~80% B-cell depletion; similar magnitude and time course of depletion between SC and IV



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

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

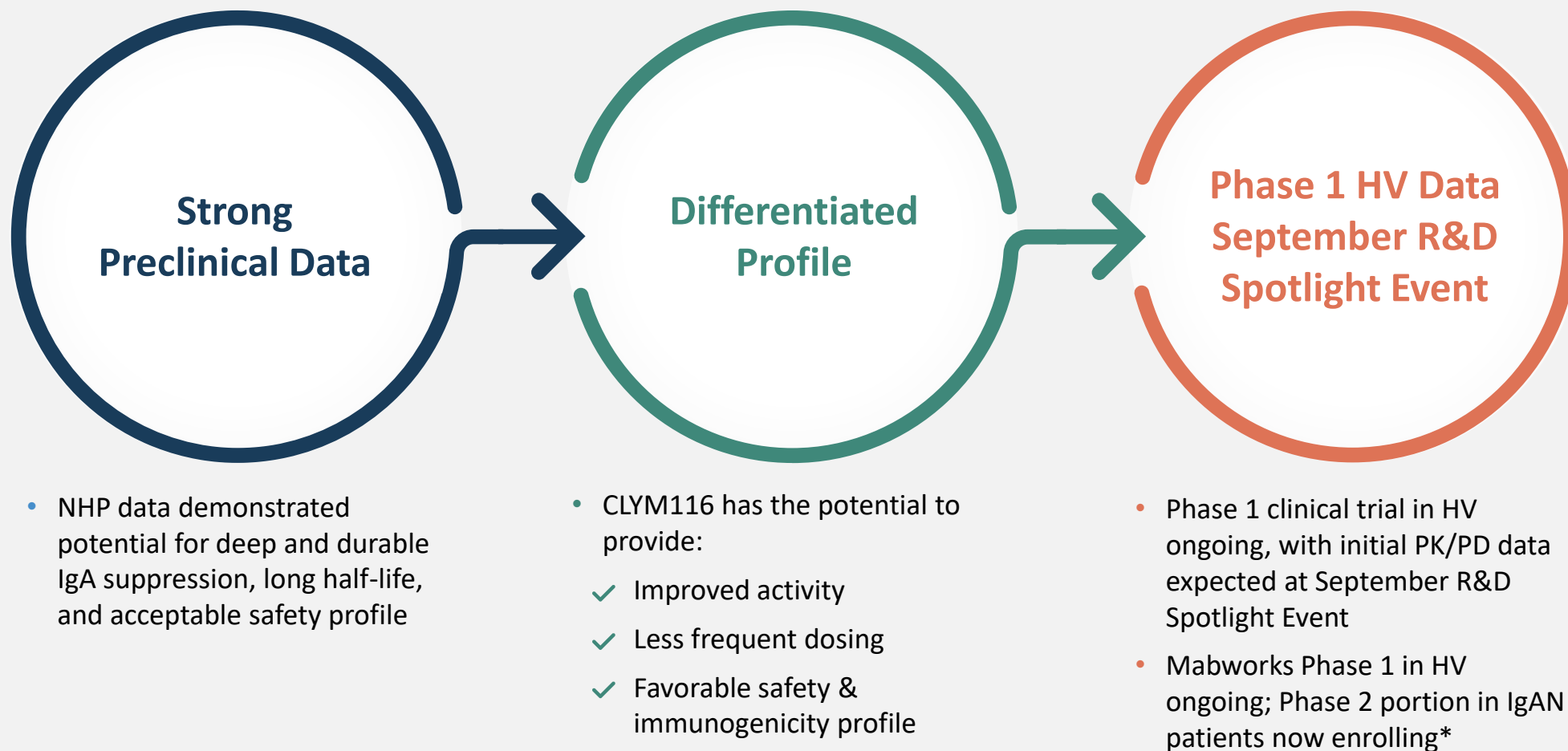


CLYM116

Anti-APRIL mAb

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

Initial PK/PD data in HVs expected in Sept; FPI in Phase 2 trial in IgAN patients anticipated in Q3 '26



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 workshop⁶
- **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

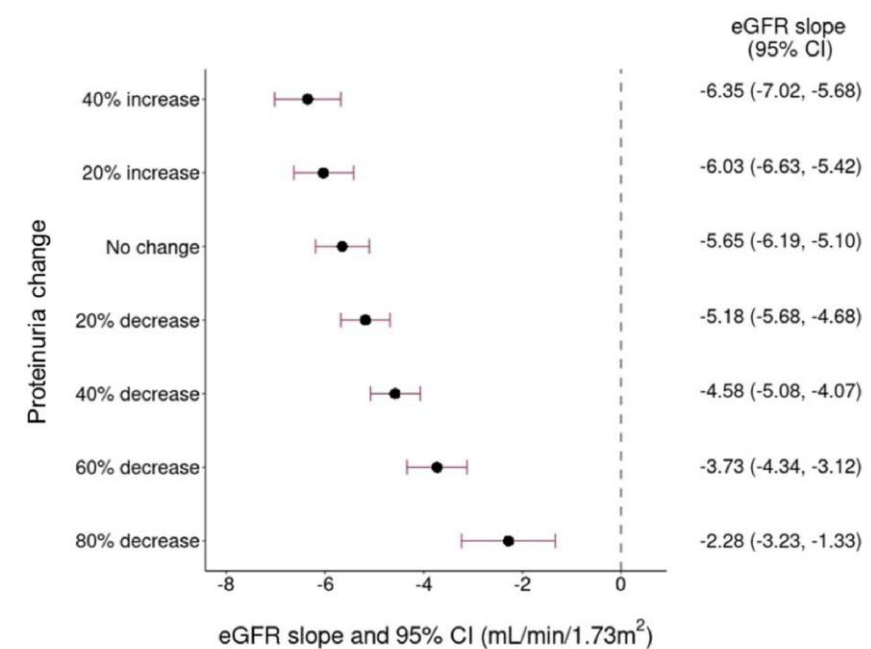
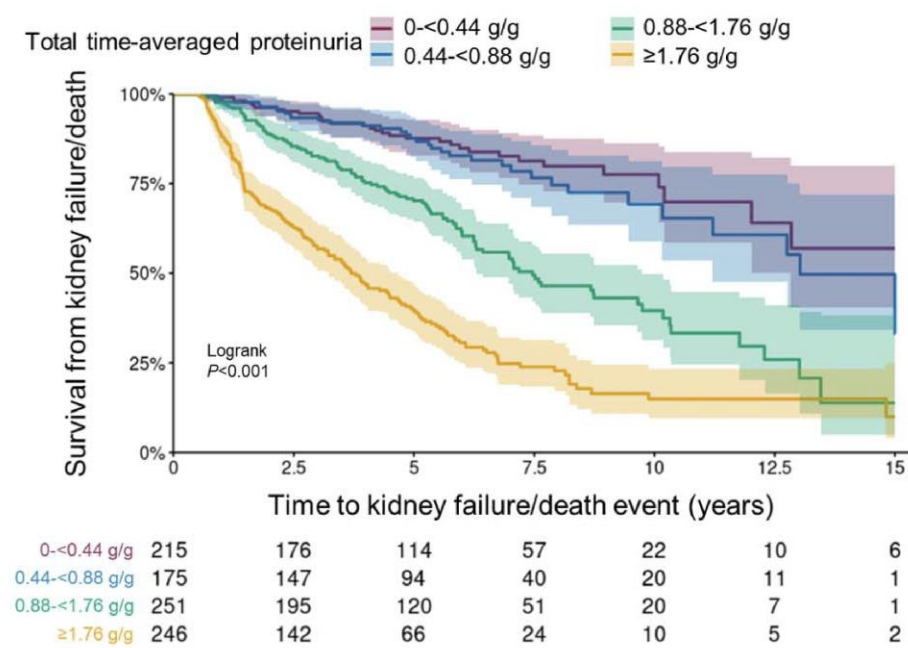
THE CLIMB SOLUTION

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

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)

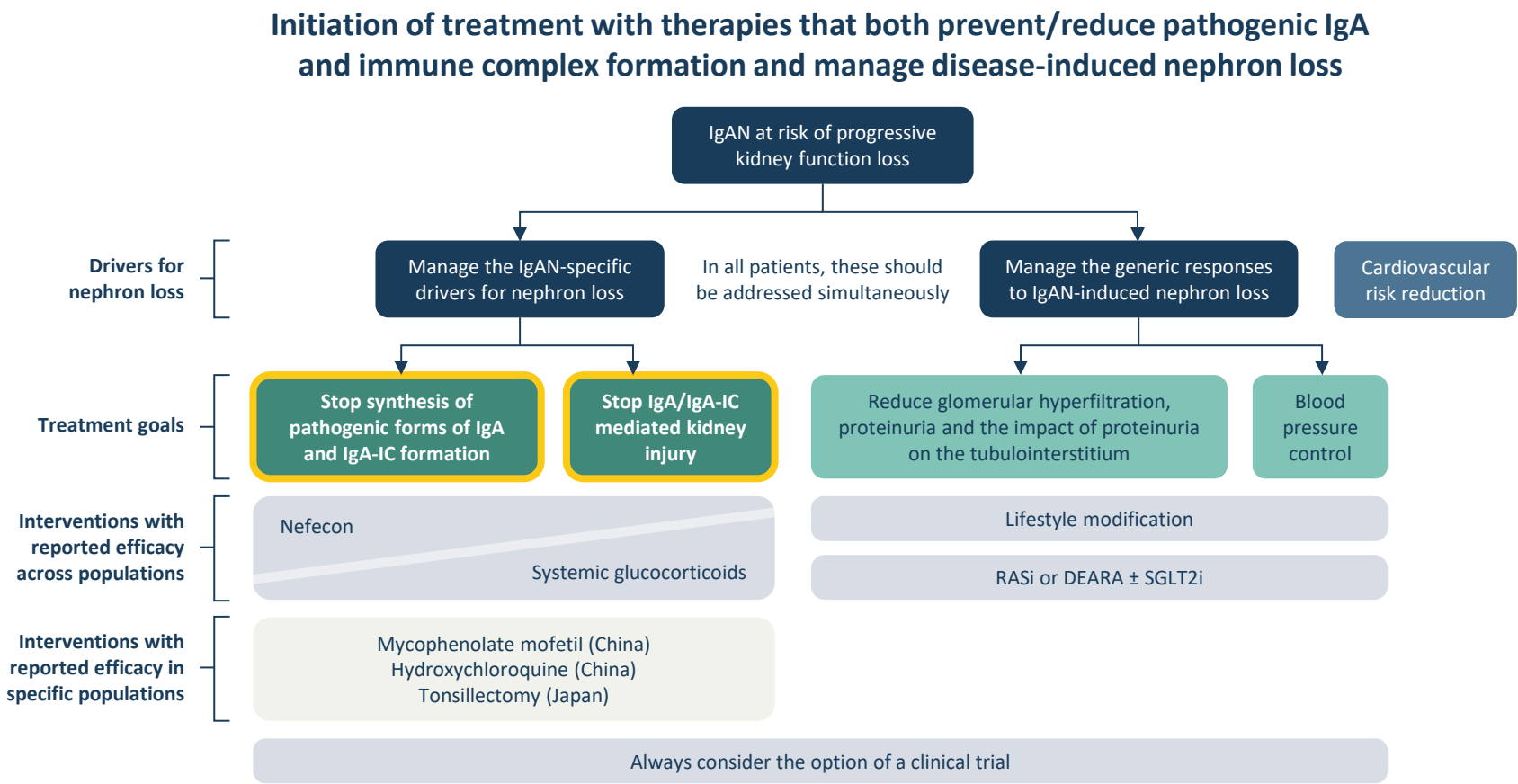


KDIGO 2025 Guideline Highlights Role for IgA Reduction in IgAN

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



Selective APRIL Inhibition Has Been Clinically Validated in IgAN

In Phase 3, sibeprenlimab matched or outperformed BAFF/APRIL agents on proteinuria (UPCR) reduction and achieved statistically significant eGFR improvement at 2 years

	SELECTIVE APRIL INHIBITION Sibeprenlimab	DUAL BAFF/APRIL INHIBITION Atacicept	DUAL BAFF/APRIL INHIBITION Povetacicept
UPCR reduction at 9 months (placebo-adjusted)	51.2% ¹ p<0.0001	42% ² p<0.0001	49.8% ³ p<0.0001
eGFR*	Stabilization of eGFR through 2 years in Phase 3 ⁴	Stabilization of eGFR at 36 weeks in Phase 2 ⁵	Stable eGFR through 48 weeks observed in Phase 1/2 ⁶

- 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

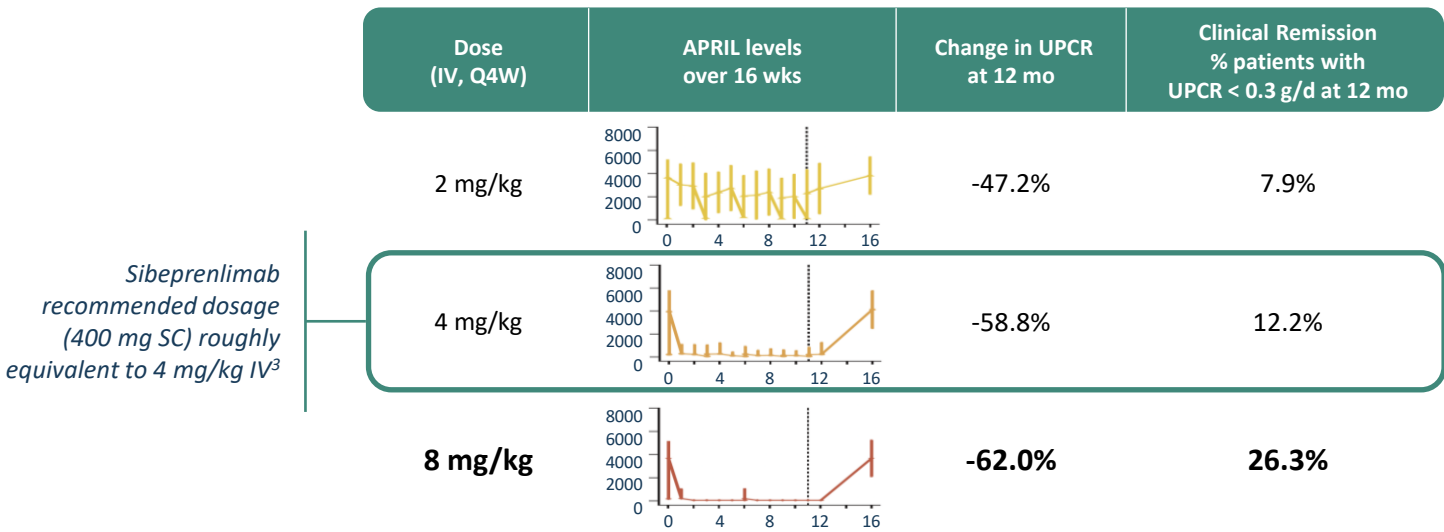
Table above reflects cross-trial comparisons and not data from head-to-head studies; differences exist between trial designs and participant characteristics, and caution should be exercised when comparing data across trials.

In patients with IgAN, depth of proteinuria reduction has been correlated with better long-term kidney outcomes, including slower eGFR decline and reduced risk of kidney failure⁷

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 data¹ suggest that sibeprenlimab recommended dosage² may not completely suppress APRIL or provide optimal proteinuria control



Immunogenicity was observed in the Phase 3 sibeprenlimab study, which resulted in an impact on drug exposure and proteinuria reductions²

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

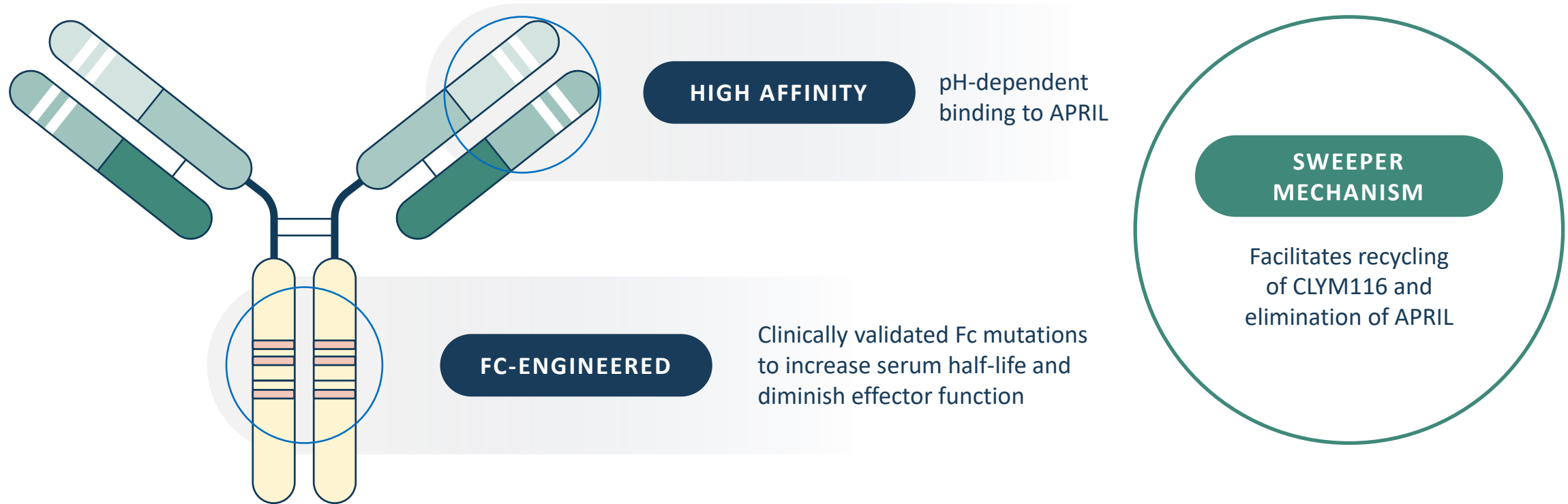
Less frequent dosing: reduced injection frequency through more prolonged APRIL suppression

Favorable safety profile, with reduced immunogenicity: supporting chronic administration

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

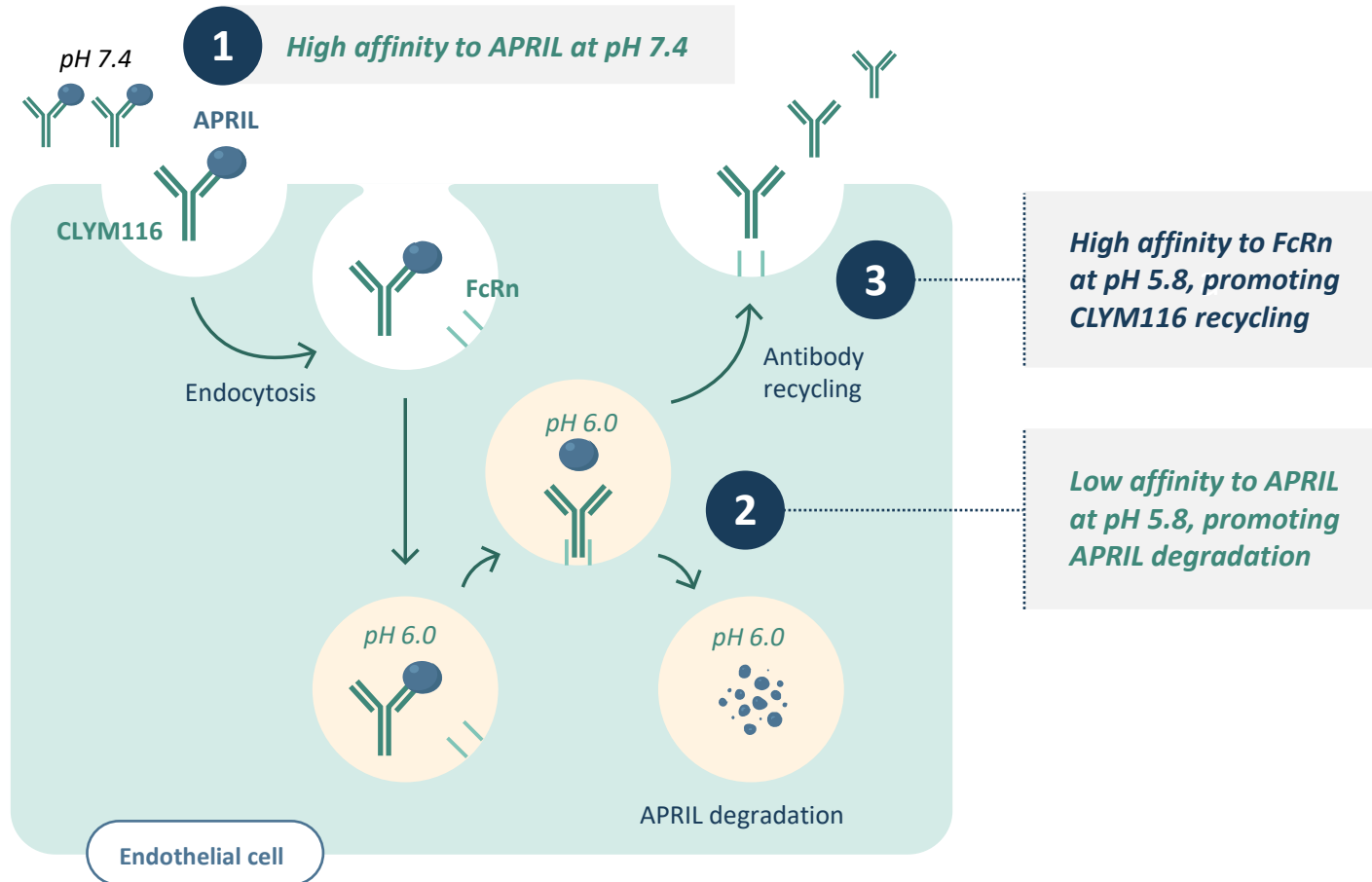
Potential best-in-class anti-APRIL mAb, designed for improved activity, less frequent dosing, and favorable safety profile

KEY FEATURES



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**

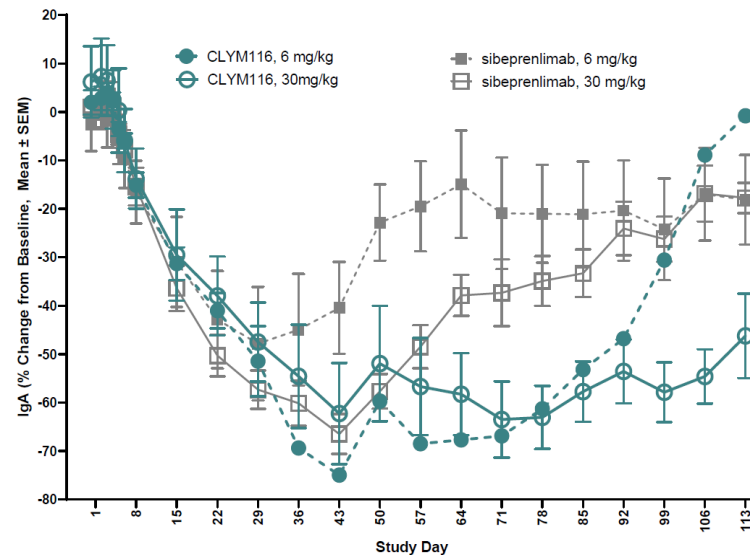
Efficient antibody recycling 3 reduces clearance of CLYM116, resulting in potentially longer half-life

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*



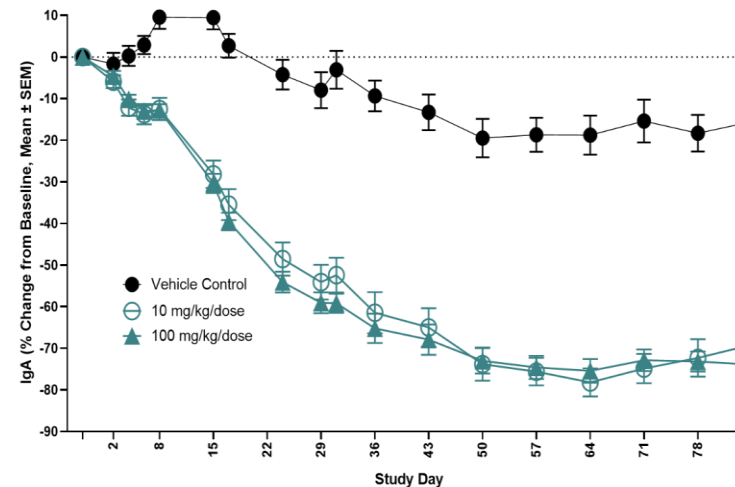
Confirmed ADA+ animals were excluded from the analysis

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

*Data from a head-to-head study in nonhuman primates, 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

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



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

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

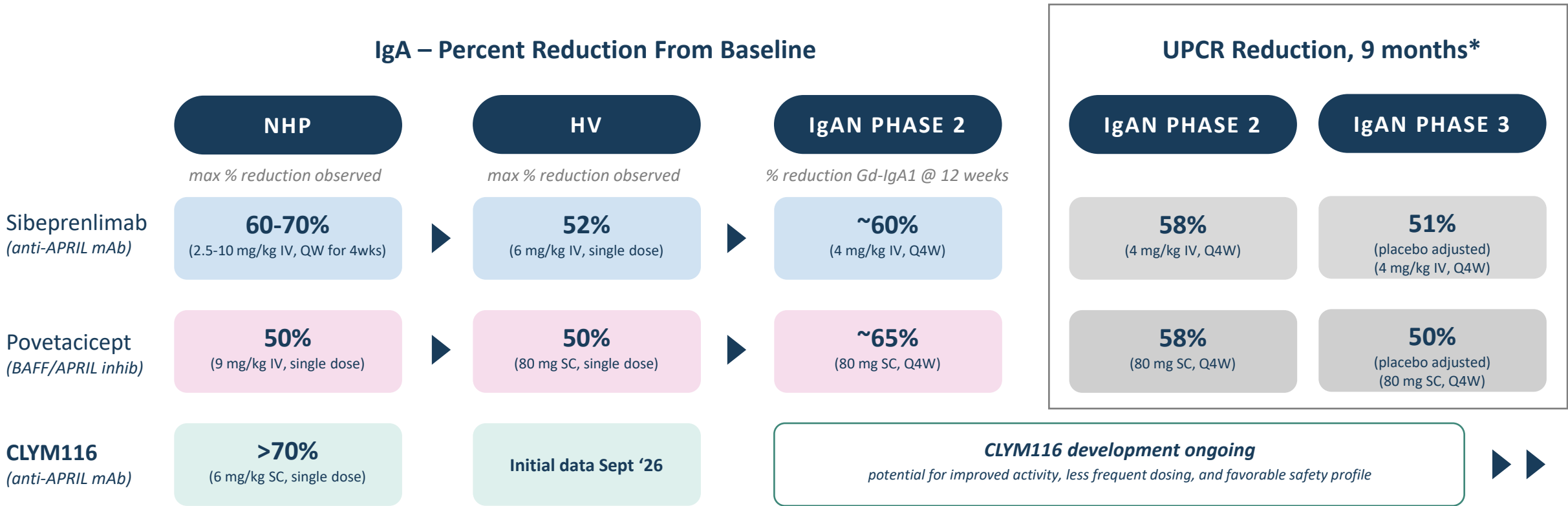


Table above reflects cross-study and cross-trial comparisons and not data from head-to-head studies; differences exist between trial designs and participant characteristics and caution should be exercised when comparing data across trials.

*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.

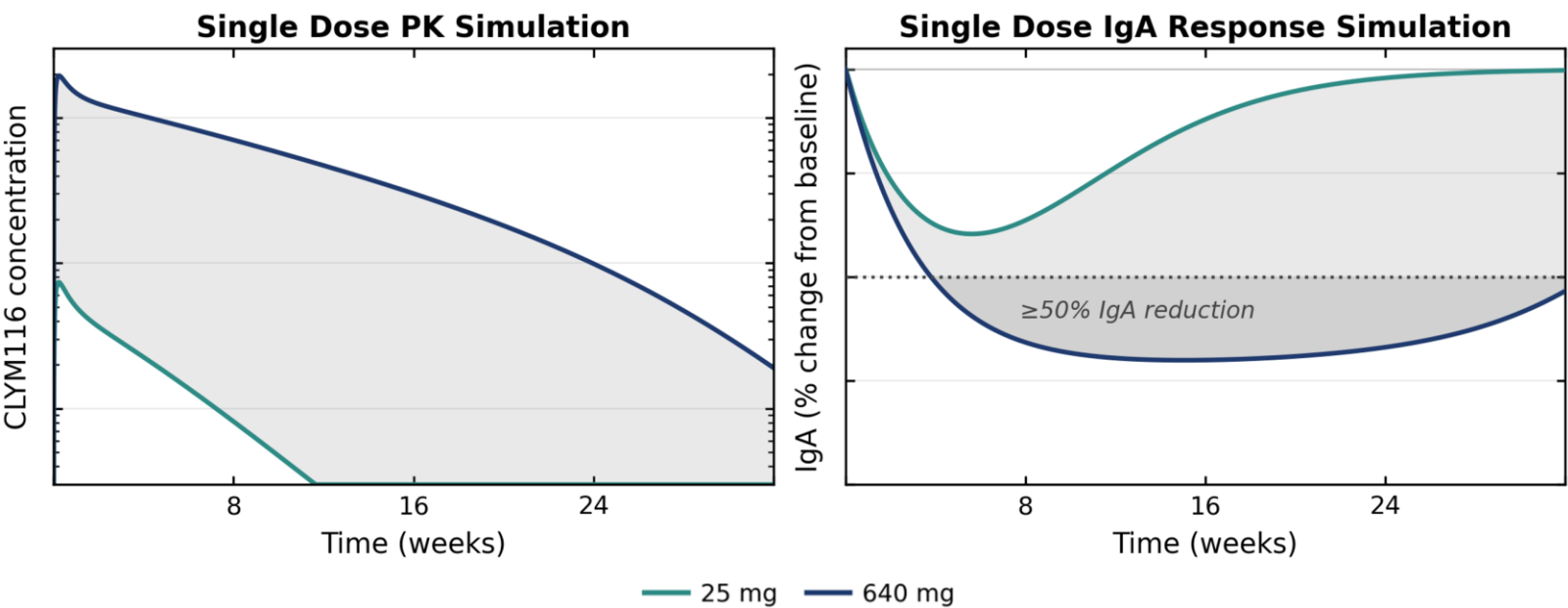
Translational Model Projects Dose-Dependent IgA Suppression in HIV

CLYM116-specific PK/PD modeling derived from NHP data projected HIV 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



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 at September R&D Spotlight Event

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

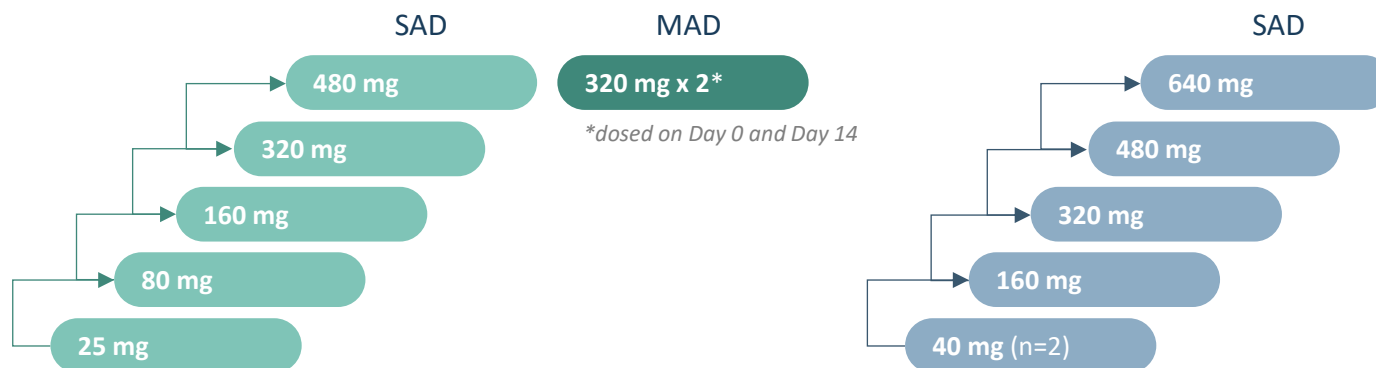
- Pharmacokinetic profile
- Effect on immunoglobulins (IgA, IgM, and IgG) and APRIL levels (pharmacodynamic response)

Australia Phase 1

China Phase 1

ASCENDING DOSE COHORTS, N ~80

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





Safety

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

Ongoing Studies in Australia and China: Preliminary Safety Data from Single Doses up to 320 mg (n=49)*

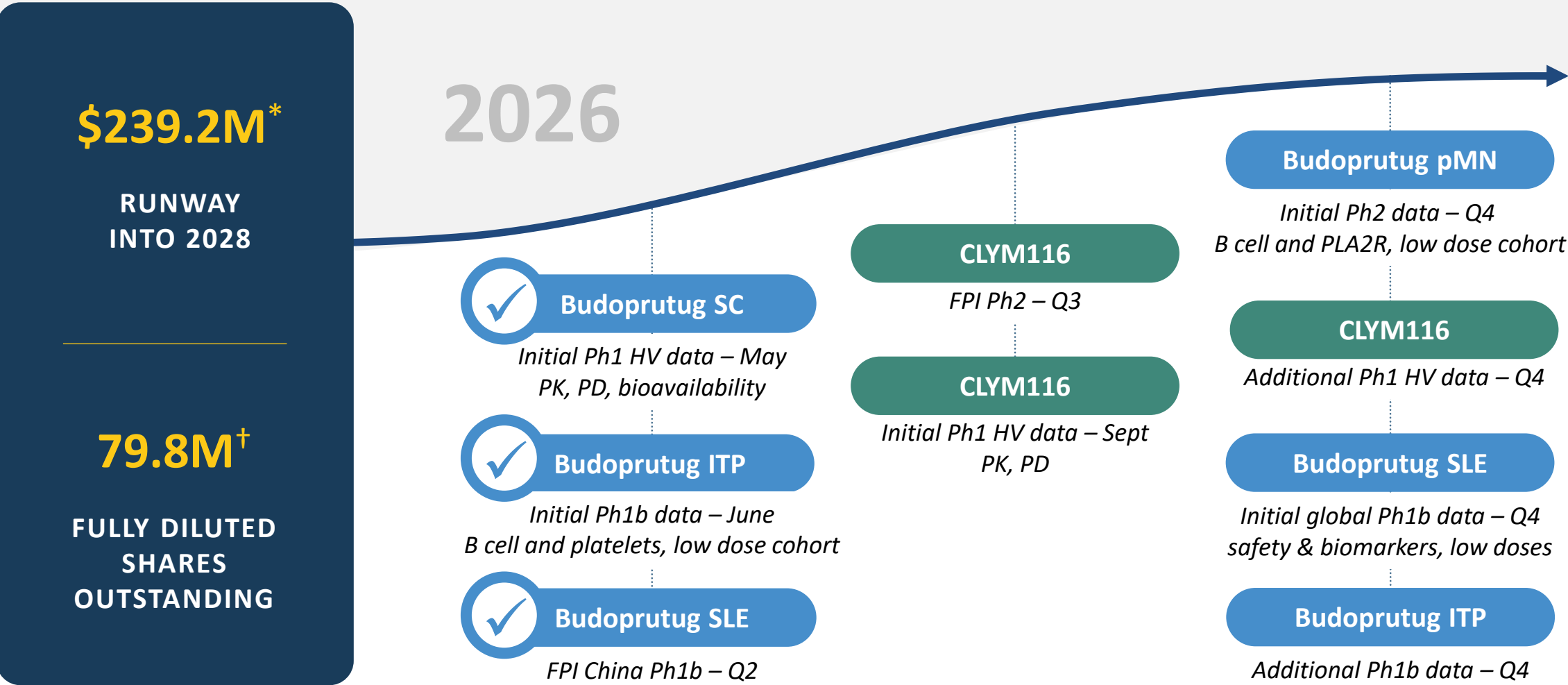
- ✓ No dose-limiting toxicities, serious adverse events, Grade ≥ 3 adverse events, or AE-related discontinuations
- ✓ All AEs mild to moderate (Grade 1-2), transient, and self-resolving
- ✓ Injection site reactions observed in 2 patients, both Grade 1, resolved without intervention

**China Phase 1 study is placebo-controlled and remains blinded. The reported population (n=49) includes participants who received CLYM116 (n=39) or placebo (n=10)*

Looking ahead

Climb Bio: Data-Rich 2026

Continuing the ascent with readouts anticipated from all ongoing trials



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 the second half of 2028**