R&D Spotlight: Budoprutug and the CD19 Opportunity

MAY 5, 2026

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Forward Looking Statements

This presentation contains “forward-looking statements” within the meaning of the Private Securities Litigation Reform Act of 1995, including without limitation statements regarding: future expectations, plans and prospects for Climb Bio; expectations regarding the therapeutic benefits, clinical potential and clinical development of budoprutug and CLYM116; the anticipated timelines for reporting initial data from Climb Bio’s ongoing and planned clinical trials of budoprutug and CLYM116; the anticipated timeline for initiating Climb Bio’s parallel clinical trial of budoprutug in patients with systemic lupus erythematosus (SLE) in China; the potential commercial opportunity and limited competitive landscape for budoprutug; the expected patient populations in primary membranous nephropathy (pMN), immune thrombocytopenia (ITP), and systemic lupus erythematosus (SLE); the expected benefits of budoprutug’s Fast Track Designation and Orphan Drug Designation in primary membranous nephropathy (pMN); the sufficiency of Climb Bio’s cash resources for the period anticipated; and other statements containing the words “anticipate,” “believe,” “continue,” “could,” “estimate,” “expect,” “intend,” “may,” “plan,” “potential,” “predict,” “project,” “should,” “target,” “would,” “will,” “working” and similar expressions. Forward-looking statements are based on management’s current expectations of future events and are subject to a number of risks and uncertainties that could cause actual results to differ materially and adversely from those set forth in, or implied by, such forward-looking statements. Climb Bio may not actually achieve the plans, intentions or expectations disclosed in these forward-looking statements, and you should not place undue reliance on these forward-looking statements. These risks and uncertainties include, but are not limited to, important risks and uncertainties associated with: the ability of Climb Bio to timely and successfully achieve or recognize the anticipated benefits of its technology transfer and exclusive license agreement with Beijing Mabworks Biotech Co., Ltd.; 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 institutional 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 (pMN), immune thrombocytopenia (ITP), systemic lupus erythematosus (SLE), IgA nephropathy (IgAN) 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.

Webcast Agenda

Corporate Strategy & Budoprutug Opportunity

Aoife Brennan, M.B., Ch.B.

President and CEO, Climb Bio

CD19: A Key Driver of B-Cell Mediated Autoimmune Disease

David Jayne, M.D.

Professor of Clinical Autoimmunity at the University of Cambridge

Director of the Vasculitis and Lupus Service at Addenbrooke's Hospital

Budoprutug: A Differentiated CD19 mAb with Broad Autoimmune Potential

Edgar Charles, M.D.

Chief Medical Officer, Climb Bio

Q&A session

including Dr. Jayne and management

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 Designations granted for pMN
- **CLYM116 -** anti-APRIL mAb in development for IgAN

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

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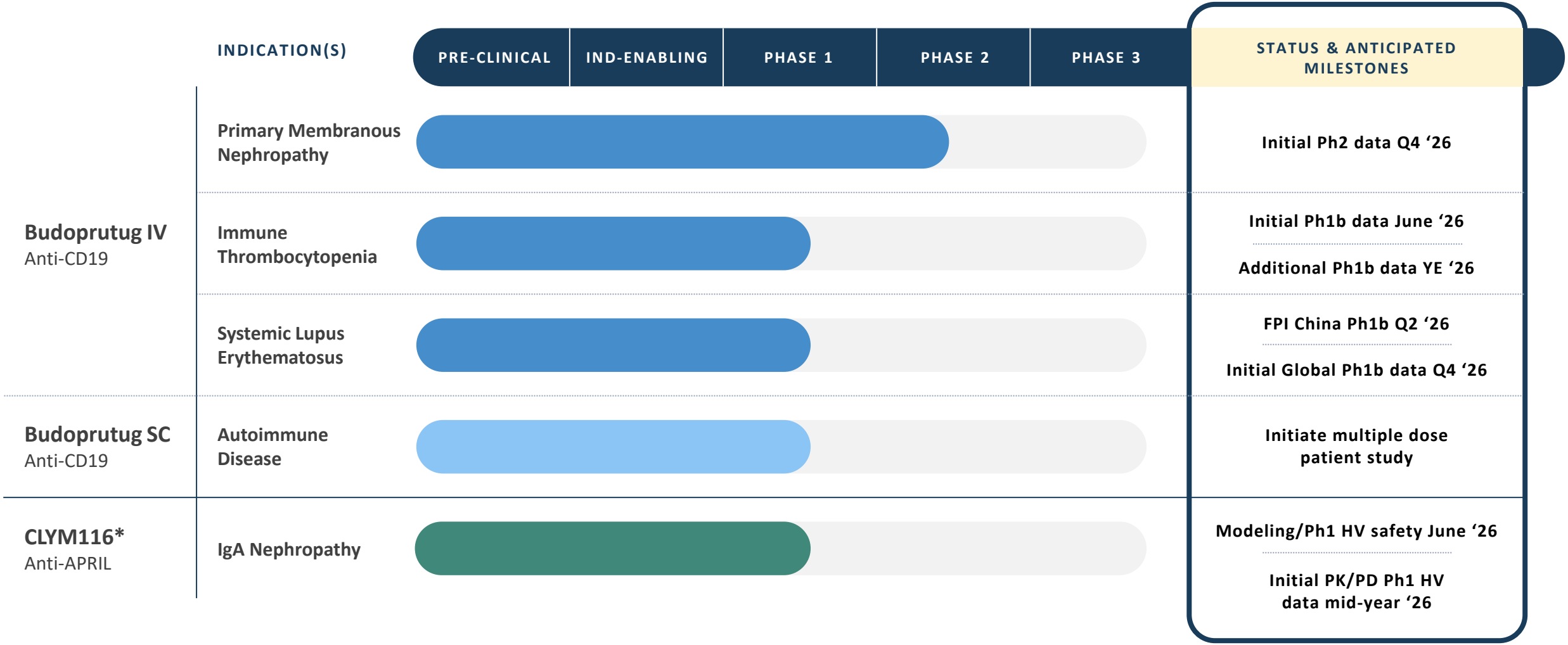
* Does not include the impact of proceeds from the April PIPE financing

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

Pipeline of Highly Differentiated mAbs

Anticipating initial readouts from all ongoing trials in 2026



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

*Climb Bio has worldwide rights outside Greater China (defined as mainland China, Hong Kong, Macau, and Taiwan); Partner: Beijing Mabworks Biotech Co., Ltd.

APRIL = a proliferation-inducing ligand, IV = intravenous, FPI = first patient in, mAbs = monoclonal antibodies, PD = pharmacodynamic, PK = pharmacokinetic, SC = subcutaneous

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CD19 mAb Targeting Remains a White Space

Low competitive intensity for an established modality translates into opportunity for budoprutug

CD19 Autoimmune Competitive Landscape[1]

B-Cell Depleting Approaches

Rationale for a mAb Approach

- Established therapeutic class with understood safety profile and scalability
- Straightforward manufacturing and broad community access
- Targeted immunomodulation without lymphodepletion

Budoprutug Opportunity

Limited competition in CD19 mAb space relative to TCEs and cell therapies | Administration profile intended to support outpatient and community use | Potential to address multiple B-cell mediated autoimmune diseases

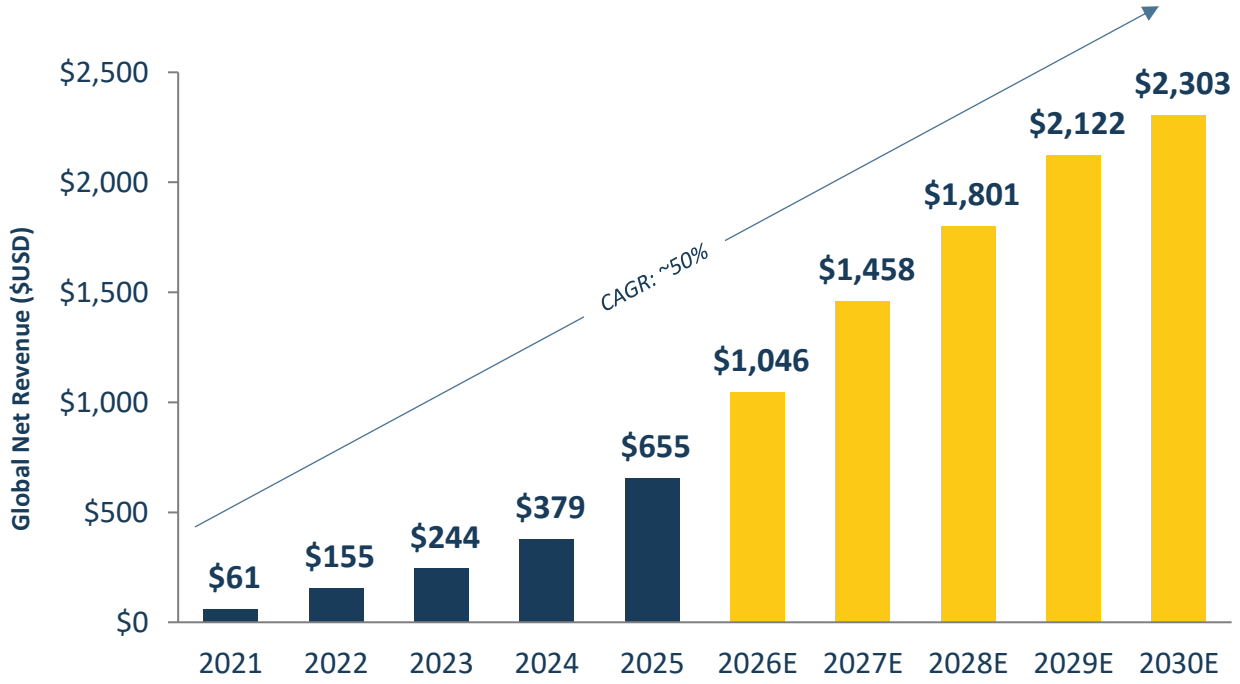
mAb = monoclonal antibody; TCEs = T cell engagers

[1] ClinicalTrials.gov, April 2026; includes clinical stage programs and approved programs (inebilizumab)

Opportunity for a CD19 B-Cell Depleting mAb is Significant

Inebilizumab's commercial success in rare neurological diseases supports the viability of a CD19 mAb approach

Inebilizumab Consensus Sales Estimates[1]



NMOSD approval, Jun 2020; IgG4-RD approval, Apr 2025; gMG approval, Dec 2025

Significant potential opportunity for budoprutug in B-cell mediated diseases beyond inebilizumab's neuro focus

gMG = generalized myasthenia gravis, IgG4-RD = immunoglobulin G4-related disease, mAb = monoclonal antibody, NMOSD = neuromyelitis optica spectrum disorder

[1] Wall Street estimates, April 2026

Budoprutug Target Indications Represent a Robust Opportunity Set

Pursuing development in diseases with large addressable populations and significant unmet need

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

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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Targeting CD19 in autoimmunity

David Jayne, M.D.

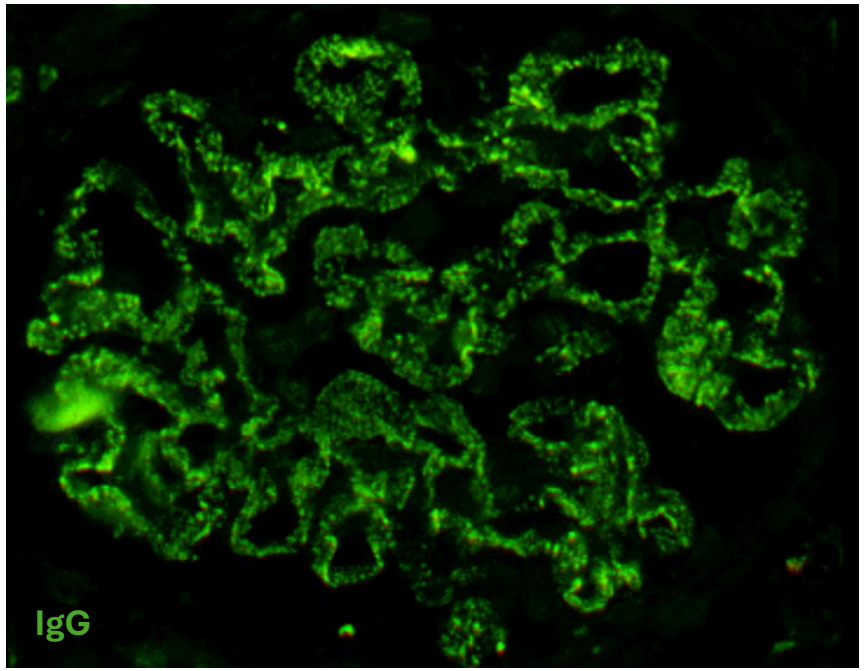
University of Cambridge, UK

Disclosures: Consultancy or lecture fees from Abbvie, Alentis, Amgen, Astra-Zeneca, Climb Bio, CSL Vifor, Fate, GSK, Novartis and Otsuka, and grants from CSL Vifor, GSK, Roche/Genentech and UCB

Functions of B cells in autoimmunity

- Develop into plasmablasts and plasma cells that generate pathogenic antibodies (e.g. pMN, ITP, SLE)
- Support T cell autoreactivity (e.g. RA, MS)
- Typically targeted by anti-CD20 monoclonal antibodies (e.g. rituximab, ocrelizumab)

Kidney biopsy of a patient with pMN



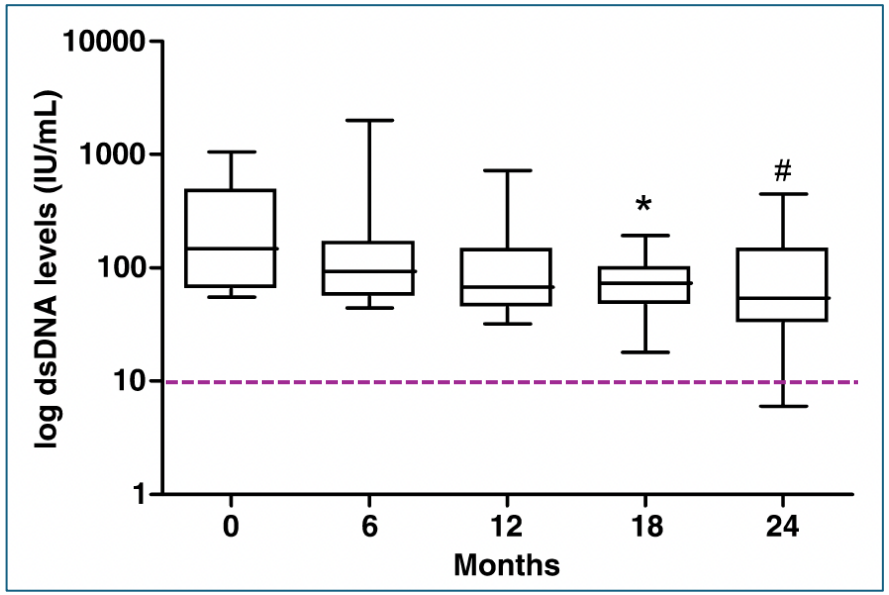
pMN = primary membranous nephropathy, ITP = immune thrombocytopenia, SLE = systemic lupus erythematosus, RA = rheumatoid arthritis, MS = multiple sclerosis

Limitations with targeting CD20

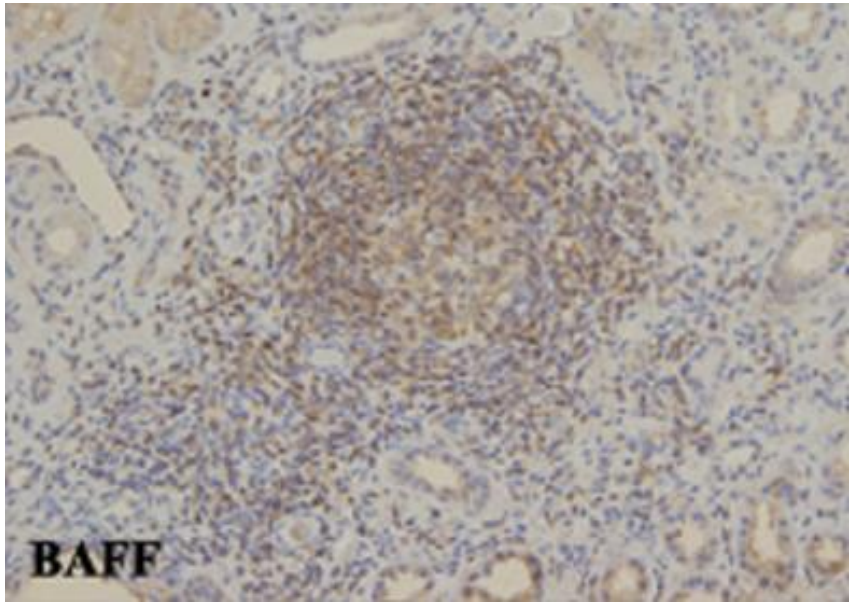
- Failure to remove circulating autoantibodies
- Incomplete removal of tissue resident B cells

} Only partial efficacy and high relapse rate

SLE: anti-DNA antibodies after rituximab



SLE: B cells in the kidney drive poor prognosis

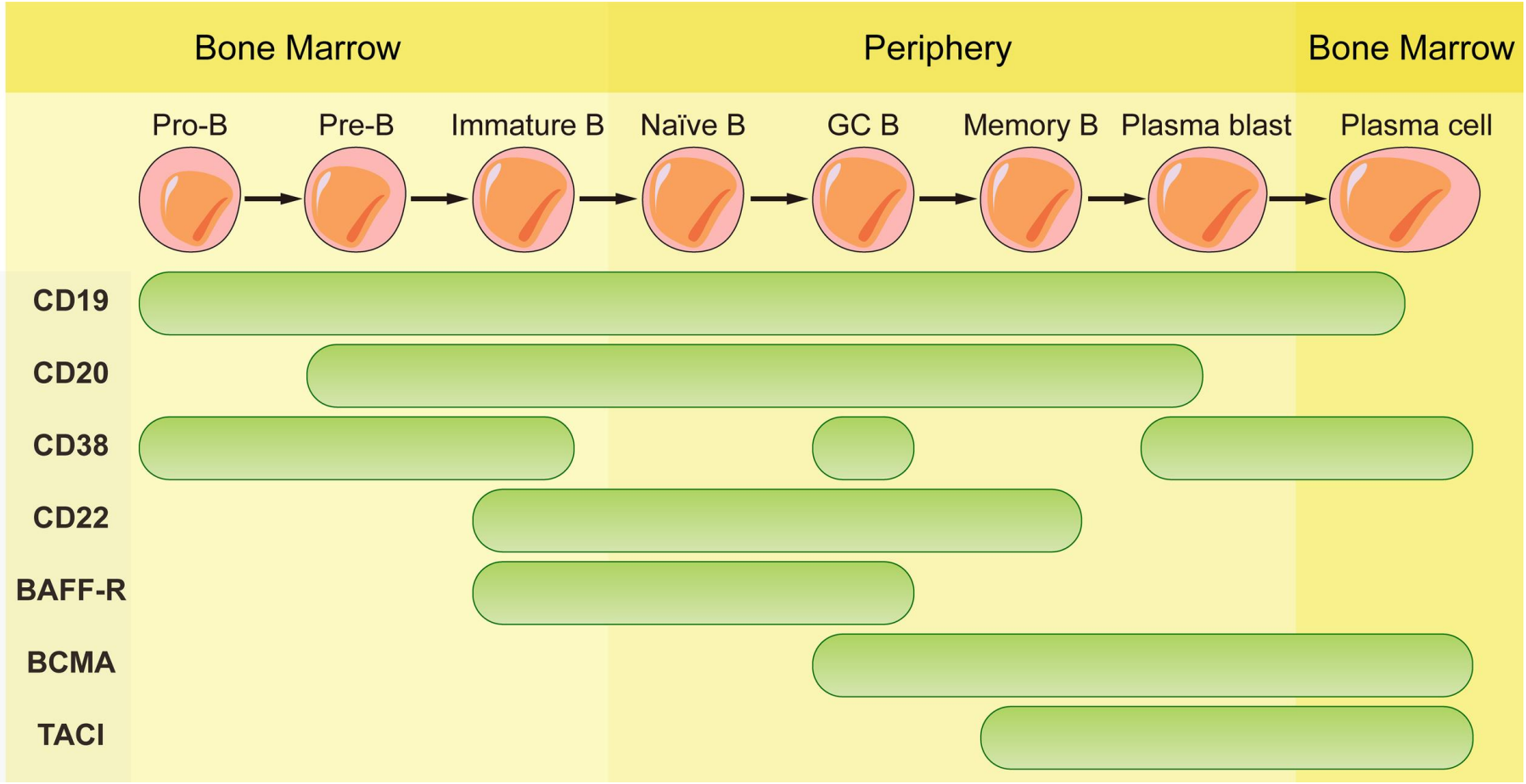


Catapano F et al, Nephrol Dial Transpl 2010. Chang et al, J Immunol 2011

Why target CD19 ?

- CD19 promotes the growth, activation, proliferation, and signaling of B lymphocytes
- $CD19^{+}$ cells play an important role in the pathogenesis of autoimmune disorders
- CD19 has a broad distribution within B cell and plasma cell development

Rationale for Use of CD19 Strategies



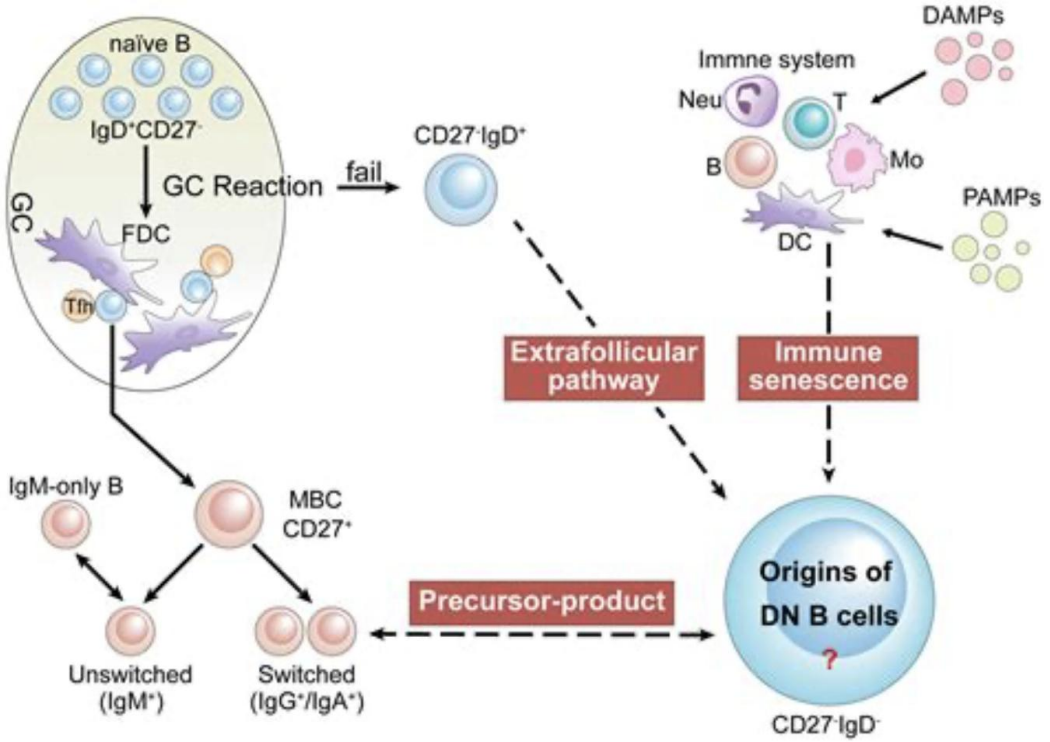
Unlike CD20, CD19 is expressed on pro-B cells, plasmablasts and many plasma cells

Zhang Z et al, Frontiers in Immunology (14) 2023

CD19 targets other pathogenic B cell subsets

“Double-Negative” (DN) B Cells

- precursors of autoantibody-secreting cells
- expanded in autoimmune diseases like SLE
- Defined by:
- CD19+ IgD- CD27-CXCR5- CD24lo



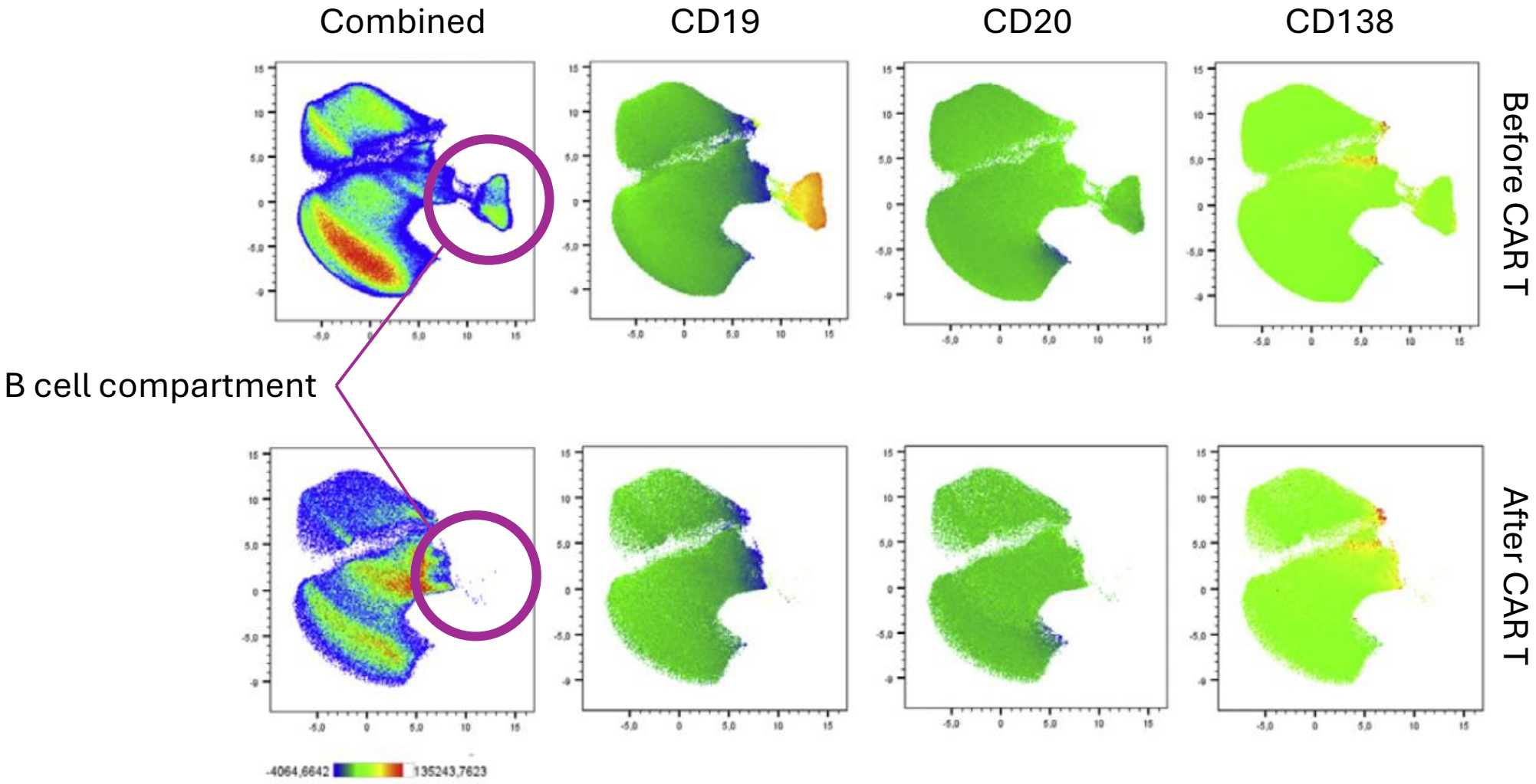
Yuzi L et al, Clin Exp Immunol 2023

Other key roles of CD19 in pathogenesis

- Autoantibody-secreting surface Ig+ CD20– CD19+ CD38+ plasma cells in rituximab-refractory autoimmune patients
- Clinical remission of rituximab-refractory autoimmune disease induced by anti-CD19 chimeric antigen receptor T cells
- CD19+ plasma cells are not short-lived plasmablasts
- Autoantigen-binding B cells and CD19+ plasma cells serve a critical role in perpetuating T cell activation in autoimmune disease

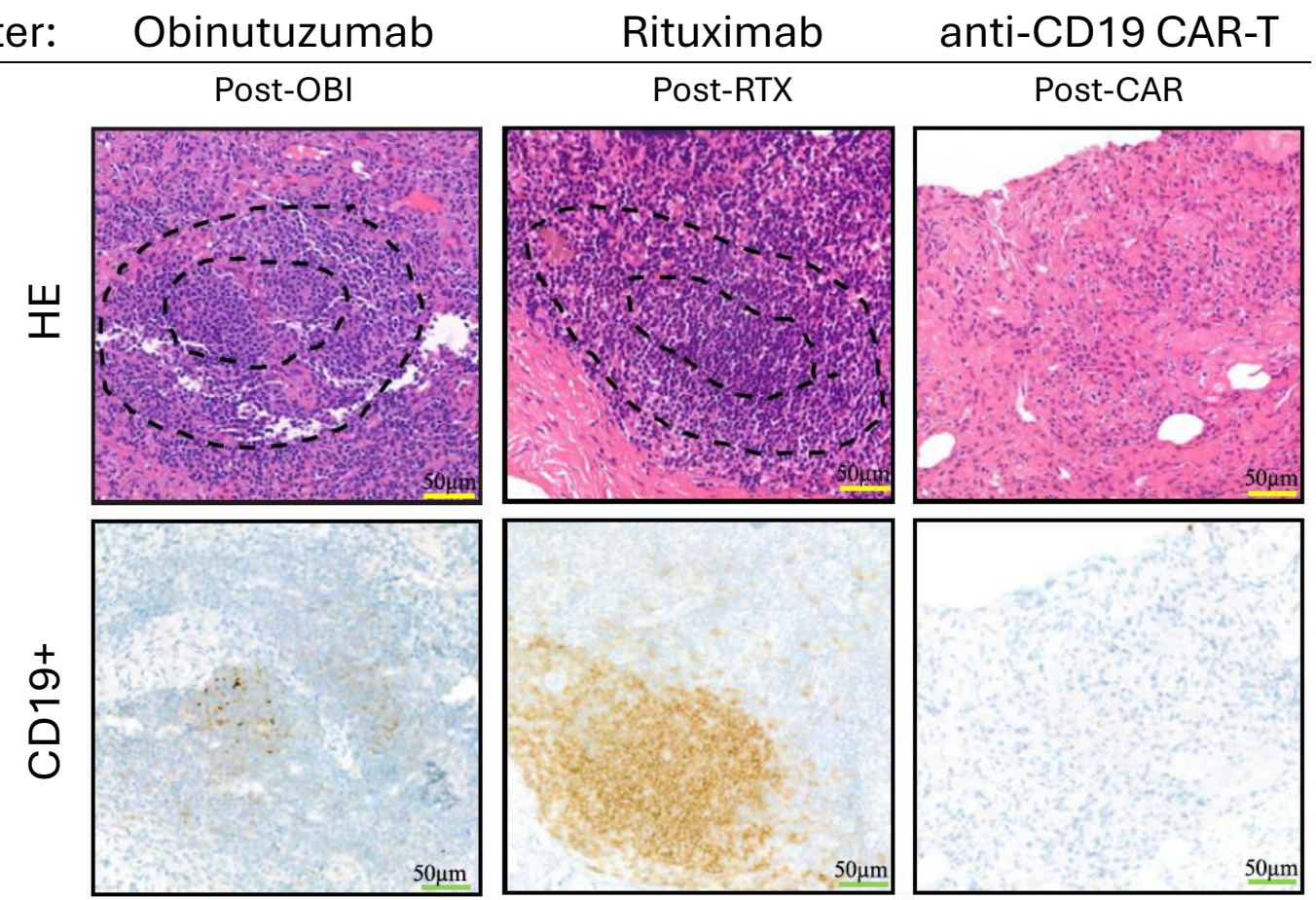
Suan D et al, J Immunol 2025

CD19 CAR-T in vasculitis: B and plasma cell depletion



Ioanna Minopoulou et al, Ann Rheum Dis 2024

Tissue depletion with anti-CD20 monoclonals



Tur C et al, Ann Rheum Dis 2025

Conclusions

- Durable remissions are associated with a B cell reset and deep depletion of B cells and plasma cells
- Targeting CD19 rather than CD20 offers potential for more complete B cell depletion, which can potentially be achieved with a monoclonal antibody approach
- This is likely to be particularly important in removing pathogenic autoantibodies, which is relevant for the treatment of many autoimmune diseases

Budoprutug: A Differentiated CD19 mAb with Broad Autoimmune Potential

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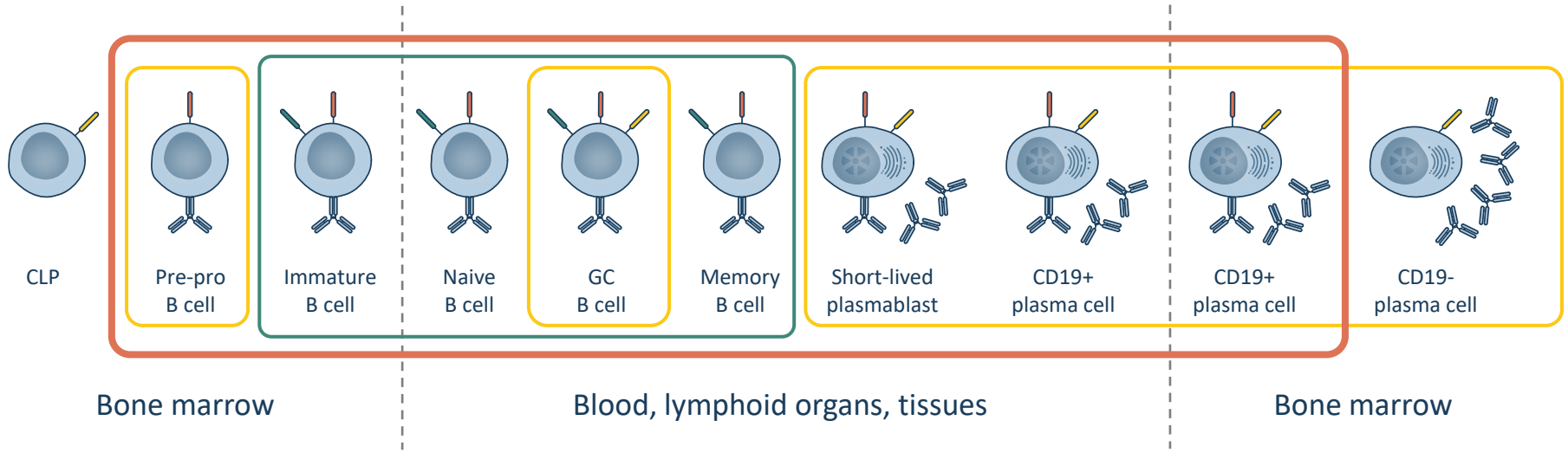
CD19 is Emerging as a Preferred Pan-B-Cell Target

Broad B-cell expression profile with potential for achieving deeper and more durable B-cell depletion

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



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

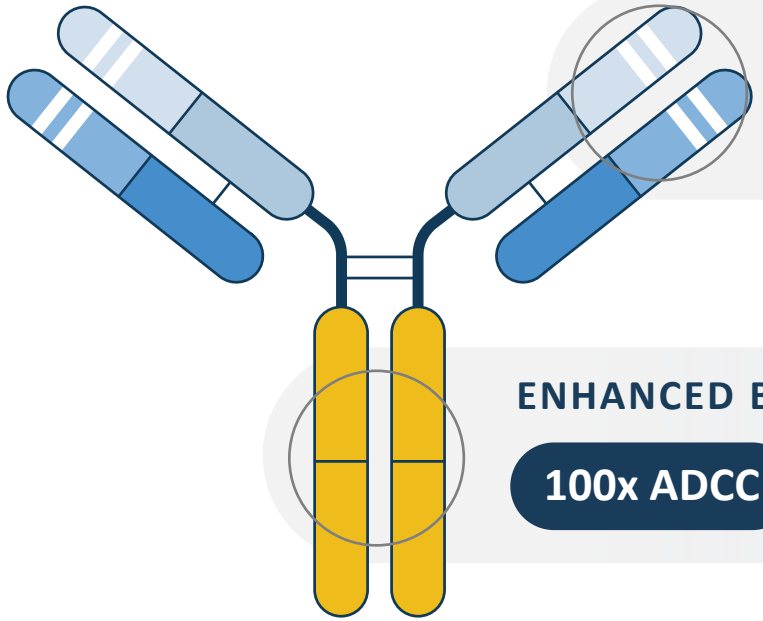
CLP = common lymphoid progenitor, GC = germinal center, mAbs = monoclonal antibodies

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

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

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

KEY FEATURES



STRONG TARGET BINDING

18 pM binding affinity to CD19 counters low antigen density

ENHANCED B CELL DEPLETION

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

SUBCUTANEOUS DOSING POTENTIAL

≥175 mg/mL

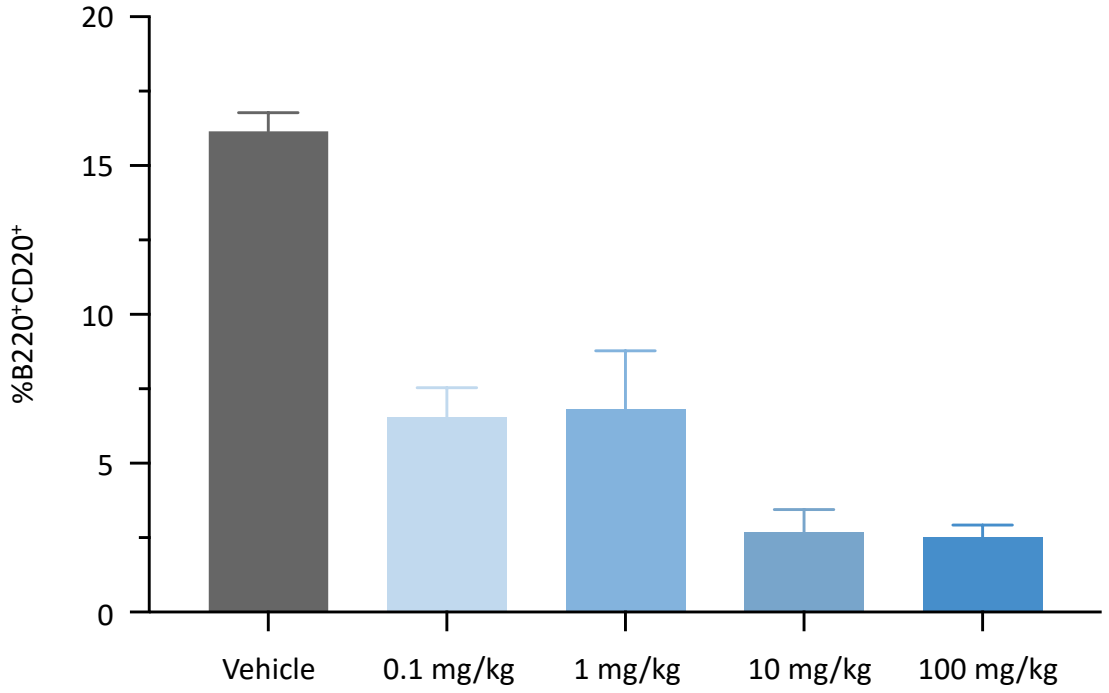
High concentration formulation with low viscosity

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

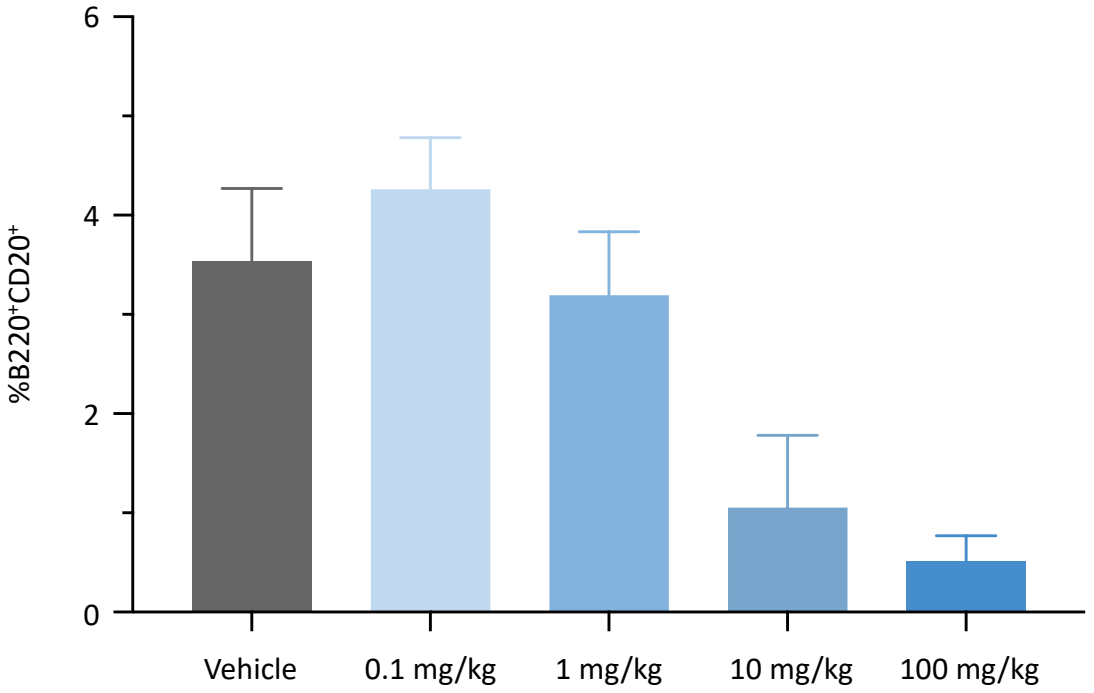
Budoprutug Has the Potential to Deliver Deep B-Cell Depletion

Budoprutug demonstrated depletion of tissue-resident B cells in a human CD19 transgenic mouse model

Lymph Node



Bone Marrow



Data expressed as % of CD45⁺ live cells, mean (SEM)

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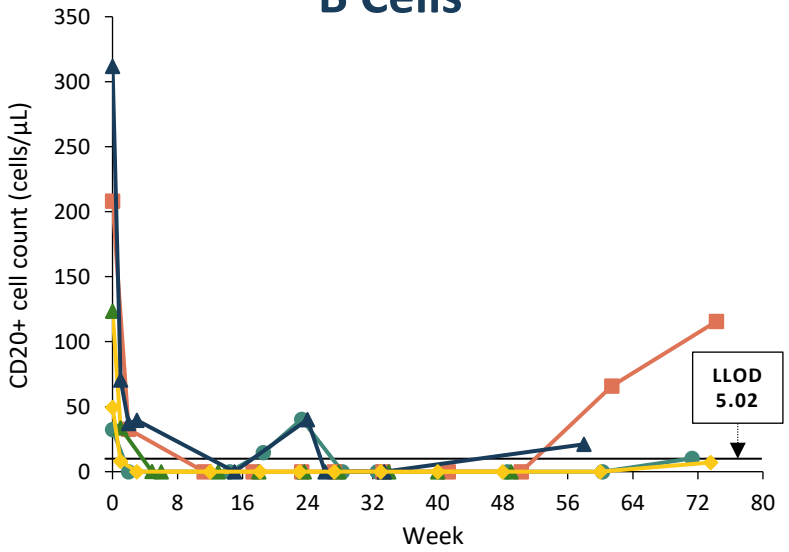
Company data on file. huCD19 transgenic C57BL/6J mice treated with budoprutug IV or Vehicle IV QW x 4. B-cell measurements taken at sacrifice 7-8 days post-last dose of budoprutug

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

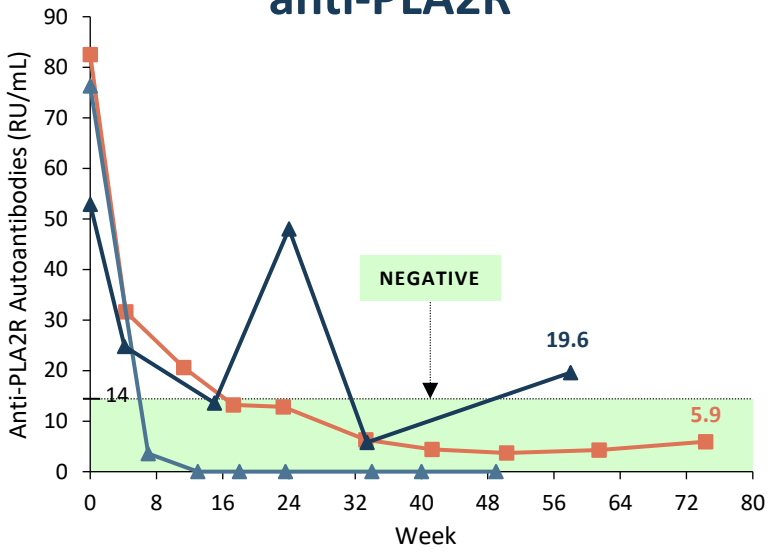
Budoprutug administration resulted in durable B-cell depletion, serologic remission, and clinical remission (as measured by proteinuria)

B Cells[1]



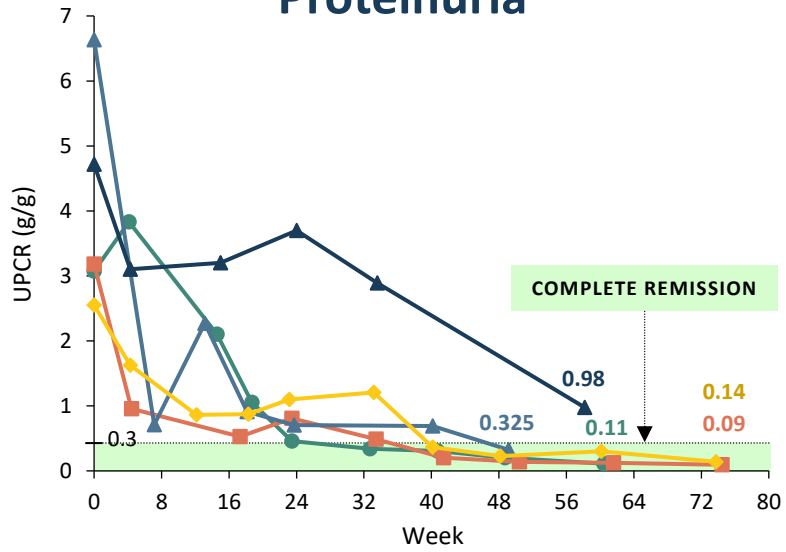
- **Rapid and complete** circulating **B cell depletion** observed in **100% (5/5) patients** at doses of 100-200 mg
- **Sustained reductions** out to 1 year + after two dose cycles

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]

No deaths, no drug-related SAEs, no discontinuations due to AEs, no DLTs

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AEs = adverse events, DLTs = dose limiting toxicities, LLOD = lower limit of detection, 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

Developing Budoprutug Across Multiple Diseases

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

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

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

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Seferiana

Living with primary membranous nephropathy

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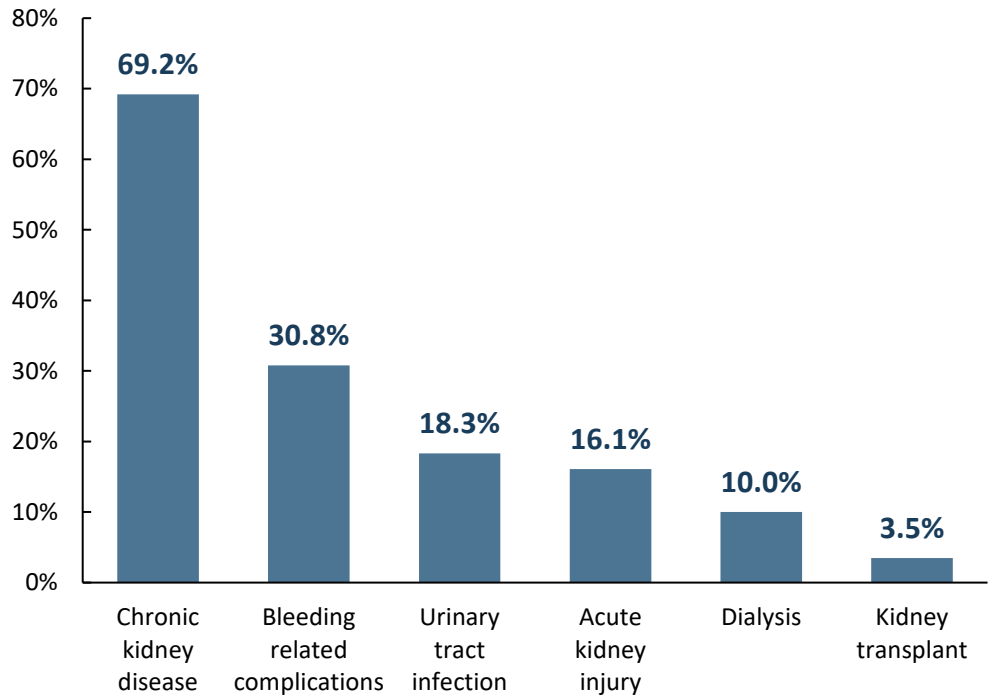
pMN is a Progressive Renal Disease With Poor Outcomes

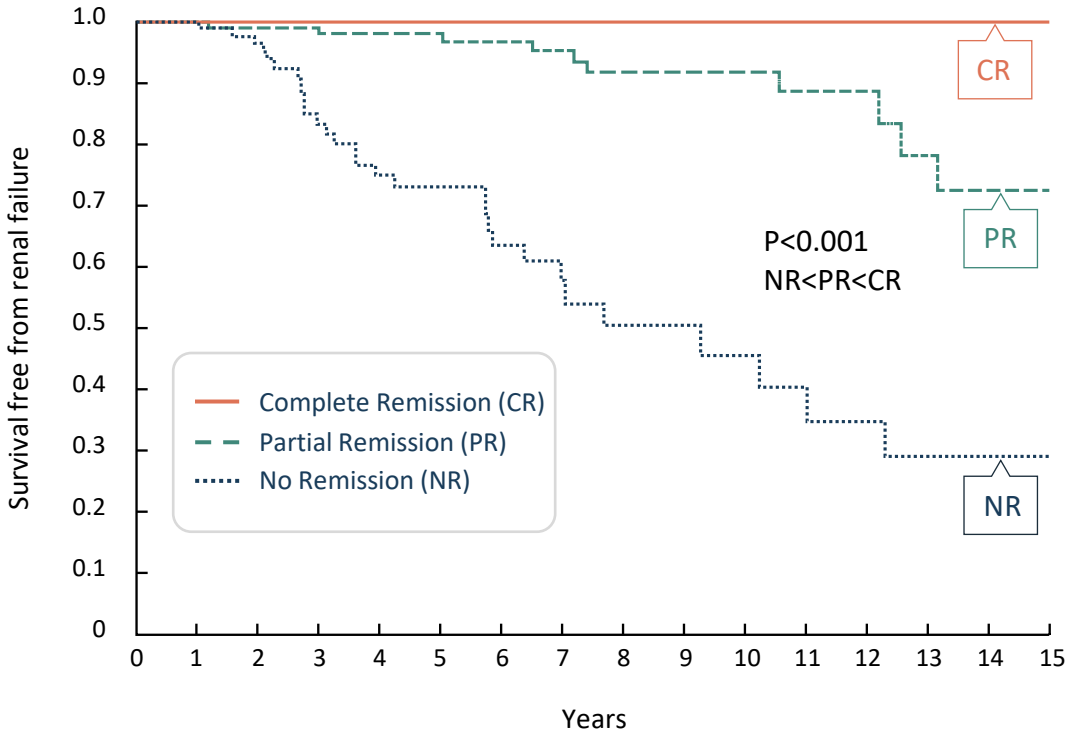
Patients with uncontrolled pMN suffer from kidney failure and other comorbidities

Patients with uncontrolled disease suffer from major comorbidities[1]

(% patients experiencing complication over 1 year period, n=2,689)



Goal is to induce complete remission to prevent declining kidney function and eventual kidney failure[2]

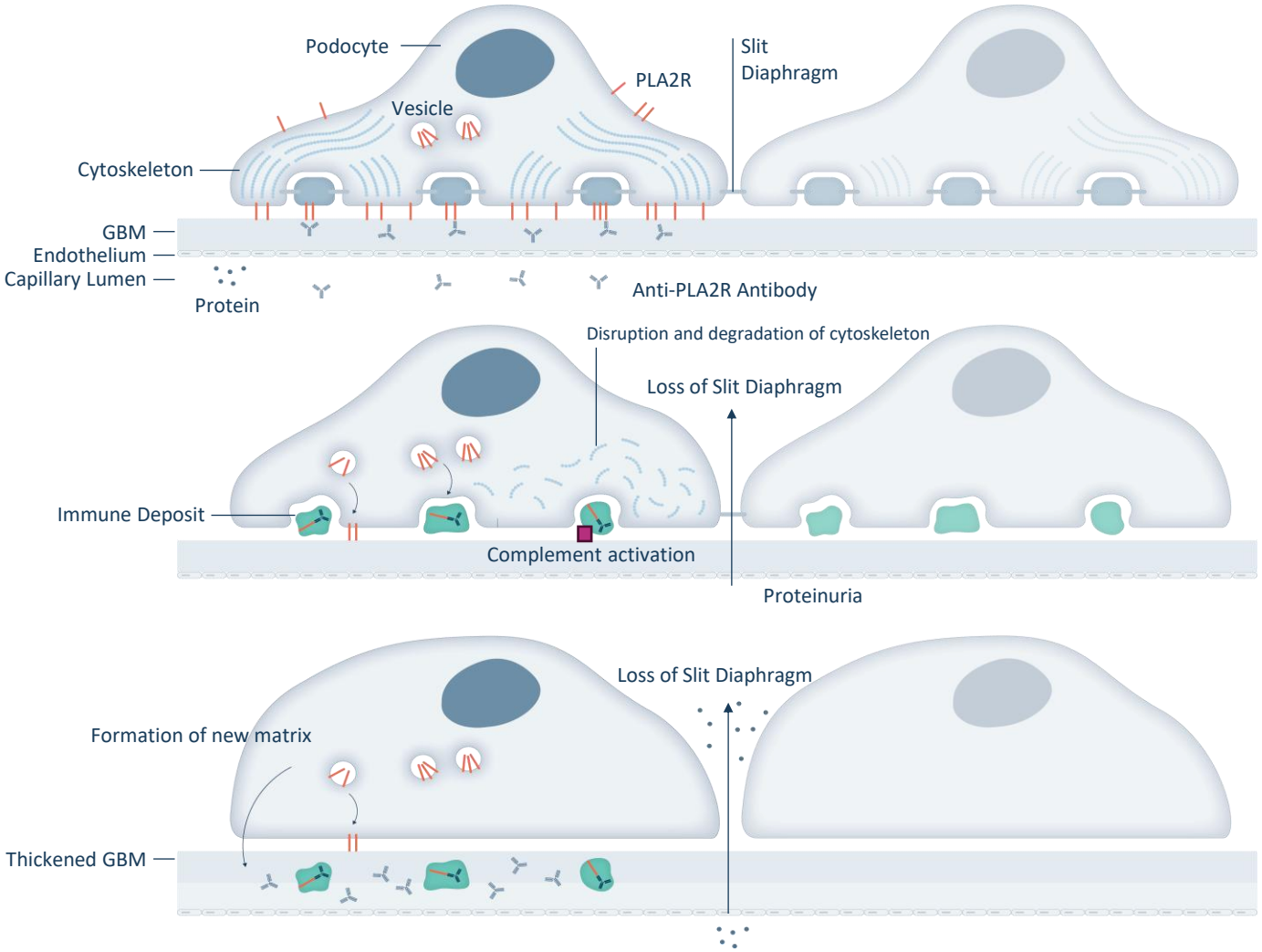


pMN = primary membranous nephropathy

[1] Adapted from Nazareth J Mang Care Spec Pharm 2019, [2] Adapted from Troyanov Kid Intl 2004

Clear Pathophysiology Supporting Targeting of CD19 in pMN

Pathogenic antibodies are IgG4 isotype and are secreted by CD19+ B cells



- B cells secrete anti-PLA2R antibodies that bind PLA2R on podocytes, forming subepithelial immune complexes.
- This leads to complement-mediated podocyte injury, disruption of the glomerular filtration barrier, proteinuria, and the development of nephrotic syndrome
- Plasmablasts and plasma cells are the source of the pathogenic autoantibodies
- Disappearance of pathogenic antibodies precedes clinical remission
- Rituximab MENTOR trial established B-cell depletion as a therapeutic option in pMN

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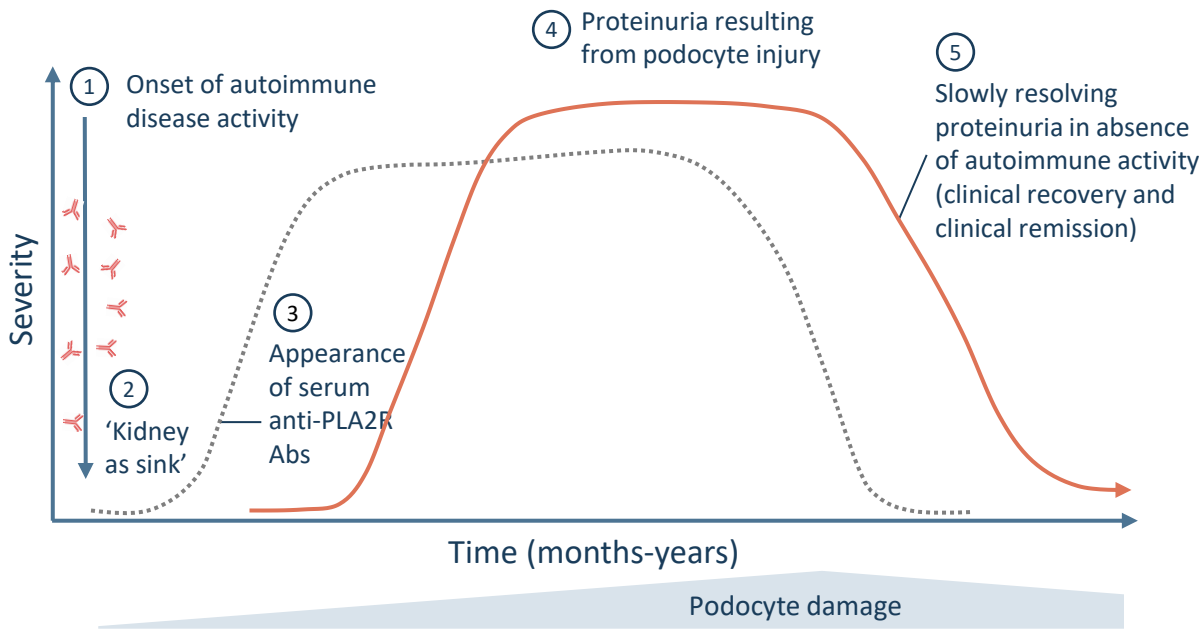
GBM = glomerular basement membrane, PLA2R = phospholipase A2 receptor, pMN = primary membranous nephropathy
Adapted from Ronco Nature Rev Disease Primers 2021, MENTOR: Fervenza NEJM 2019

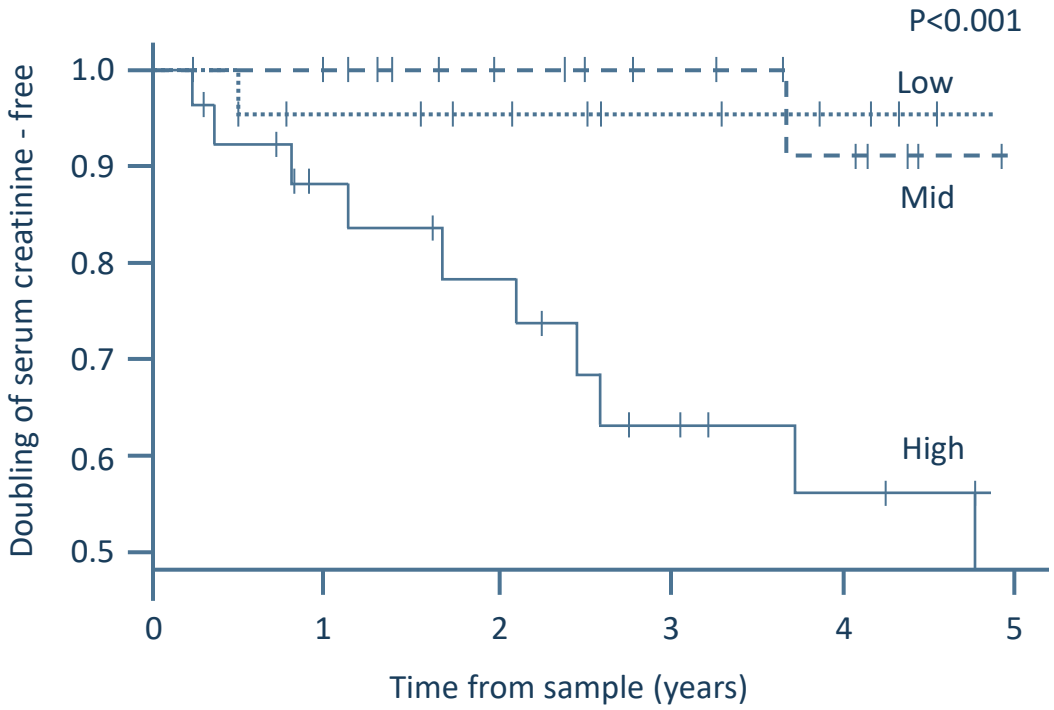
Anti-PLA2R is a Predictive and Prognostic Biomarker in pMN

Immunologic recovery precedes proteinuria resolution; titer has been associated with long-term risk of disease progression

Anti-PLA2R reflects active autoimmune disease; immunologic recovery precedes resolution of proteinuria[1]



Anti-PLA2R titer associated with risk of kidney disease progression[2]



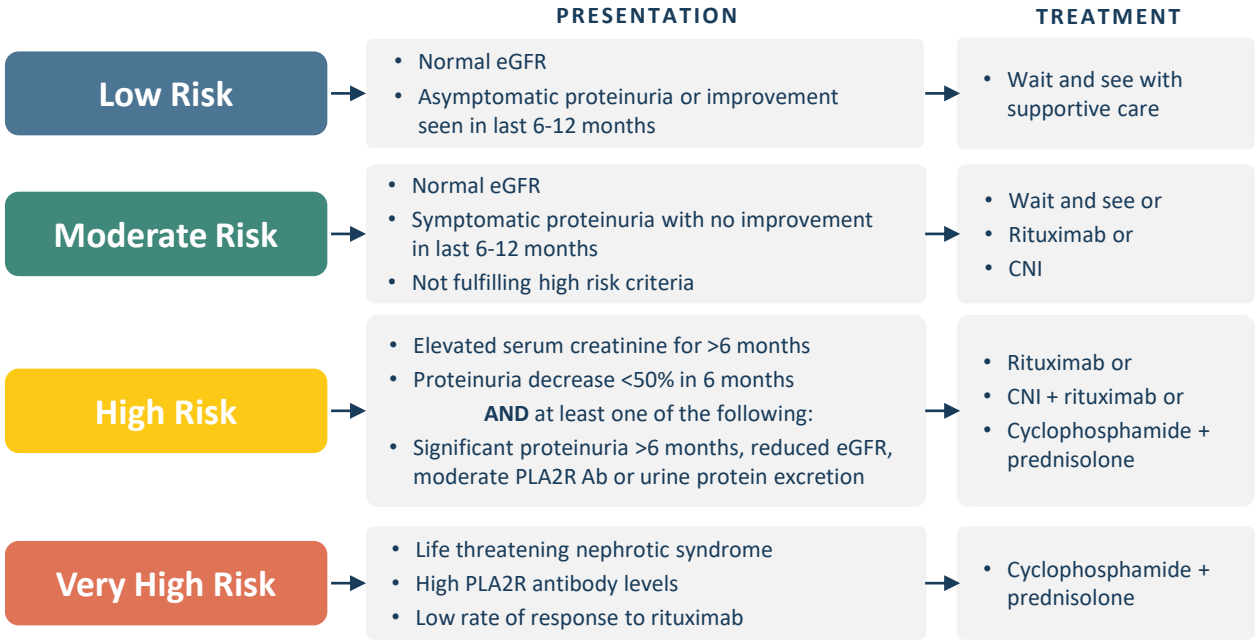
Abs = antibodies, PLA2R = phospholipase A2 receptor, pMN = primary membranous nephropathy

[1] Adapted from Lener KI 2021, [2] Adapted from Kanigcherla KI 2013

No Approved Therapies for pMN, Unmet Need Remains High

KDIGO Guideline recommends off-label use of rituximab or CNIs, but remission rates are low, highlighting the need for disease-modifying therapies that deliver complete remission of proteinuria

KDIGO Guideline recommends treatment with rituximab, cyclophosphamide or CNI-based therapy based on risk estimate[1]



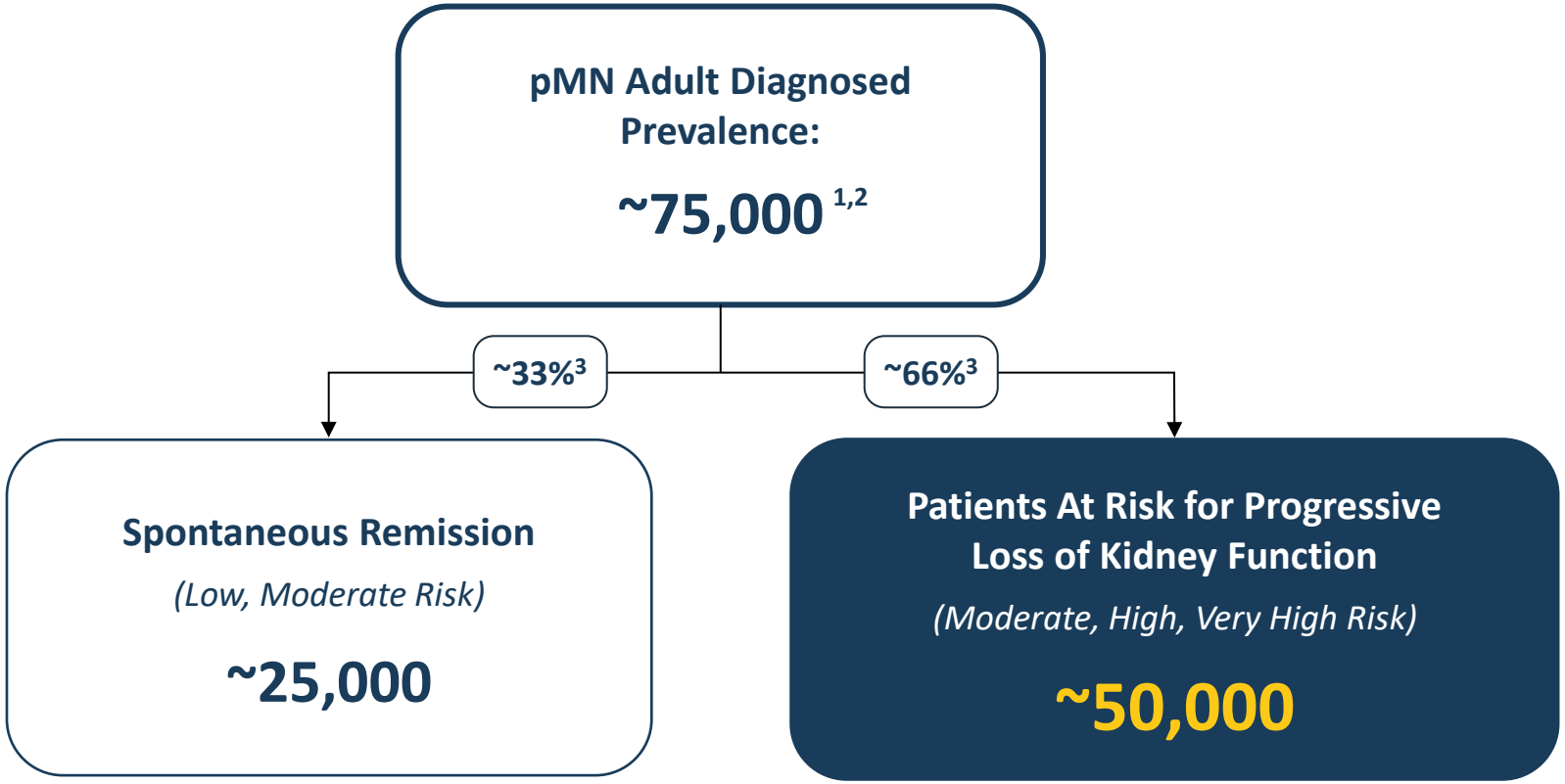
MENTOR demonstrated benefit of rituximab vs. CNI, but only 35% patients achieved CR at 2 years[2]

	Complete Remission (CR) Rate	
	1 year	2 years
Rituximab	14%	35%
CNI	1.5%	0%

Ab = antibody, eGFR = estimated glomerular filtration rate, KDIGO = Kidney Disease Improving Global Outcomes, CNI = calcineurin inhibitor, PLA2R = phospholipase A2 receptor, pMN = primary membranous nephropathy [1] Adapted from KDIGO 2021 Guideline for the Management of Glomerular Diseases, [2] Fervenza NEJM 2019

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] KIDGO 2021 Guideline for the Management of Glomerular Diseases

Budoprutug Has a Highly Competitive Profile in pMN

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

	Budoprutug (based on completed pMN Ph1b)	**Rituximab**	**Obinutuzimab**	**Povetacicept**	**Felzartamab**
Target	CD19	CD20	CD20	BAFF/APRIL	CD38
Serologic remission (anti-PLA2R negativity)	✓ **100%** (3/3)[1]	64–95%[2] (titer decrease)	90-92%[6-7]	100% (4/4)[9]	23% (6/26)[10]
Complete or partial clinical remission	✓ **100%** (5/5)[1]	60% (39/65)[2]	85% (50/59) to 95% (20/21)[6-7]	100% (5/5)[9]	35% (9/26)[10]
Complete remission	✓ **60%** (3/5)[1] *UPCR ≤0.3 g/g*	14-41%[2-5] *UPCR ≤0.3 g/g*	29-38%[6-8] *UPCR ≤0.3 g/g*	40% (2/5)[9] *UPCR <0.5 g/g*	0% (0/26)[10] *UPCR <0.5 g/g*
Dosing	✓ 2 IV doses administered 14 days apart, then q6m	2 x 1000 mg IV doses administered 7 days apart, then q6m	2 x 1000 mg IV doses administered 14 days apart, then q6m	80 mg SC every 4 weeks	9 IV doses over 6 months

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

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

IV = intravenous, PLA2R = phospholipase A2 receptor, pMN = primary membranous nephropathy, q6m = every 6 months, 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 Kid Int 2008, [4] Fervenza CJASN 2010, [5] Roccatello Autoimmun Rev 2016, [6] Hu CJASN 2024, [7] Su KI Reports 2024, [8] Lin Kid Med 2024, [9] Madan KI Reports 2025, [10] Rovin KI Reports 2024

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PrisMN: Budoprutug Phase 2 pMN Study

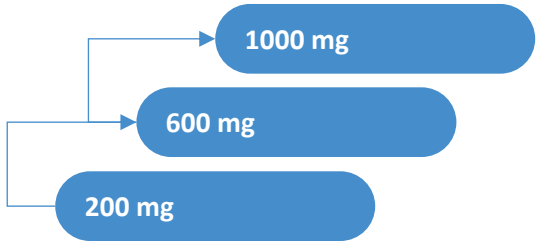
Global, open-label, dose escalation study enrolling patients with moderate to severe pMN

PrisMN
A NEW LENS ON PMN

Dose escalation starts at previous Phase 1b dose and escalates to 1000 mg

OPEN-LABEL, DOSE ESCALATION STUDY N = 45

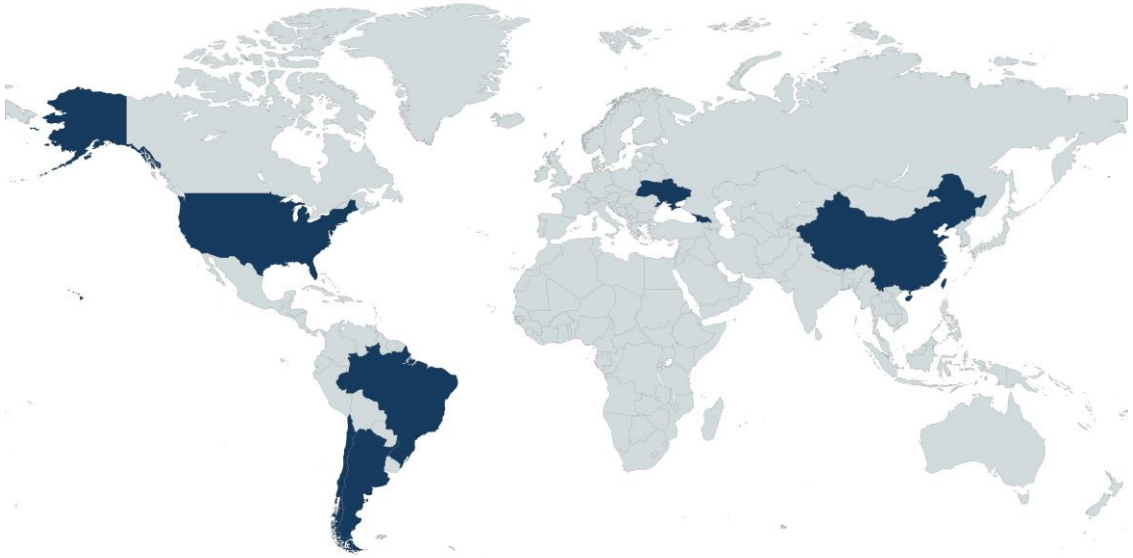
15 patients per cohort, enrolled sequentially



Population

- 18-70 years of age
- Confirmed diagnosis of pMN, no secondary causes of MN
- UPCR ≥ 2.0 g/g
- PLA2R antibody positive
- Adequate trial of ACEi/ARB, with stable dose for ≥ 4 weeks
- eGFR ≥ 35 mL/min/1.73m^2
- ≥ 6 months since any prior B cell depleting agent

Global footprint - ~45 sites across 8 countries



- USA
- Argentina
- Brazil
- Chile
- China
- Georgia
- Taiwan
- Ukraine

NCT07096843

ACEi = angiotensin converting enzyme inhibitor, ARB = angiotensin receptor blocker, eGFR = estimated glomerular filtration rate, MN = membranous nephropathy, PLA2R = phospholipase A2 receptor, pMN = primary membranous nephropathy, UPCR = urine protein creatinine ratio

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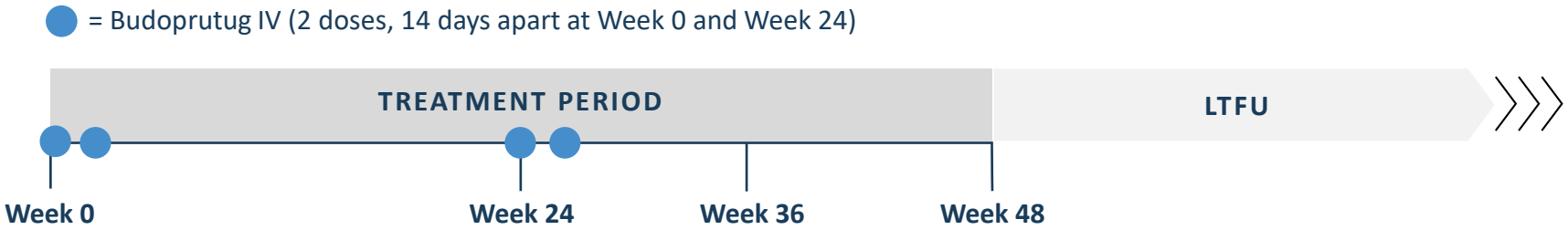
PrisMN: Budoprutug Phase 2 pMN Study

Biomarker endpoints provide potential to rapidly identify a Phase 3 dose

PrisMN
A NEW LENS ON PMN

Biomarker data will drive Phase 3 dose identification, while long-term follow-up will evaluate potential for disease remission



Initial B-cell depletion and anti-PLA2R data from the low dose cohort (200mg at 12-24 weeks) anticipated Q4 2026

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)

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

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John

Living with immune thrombocytopenia

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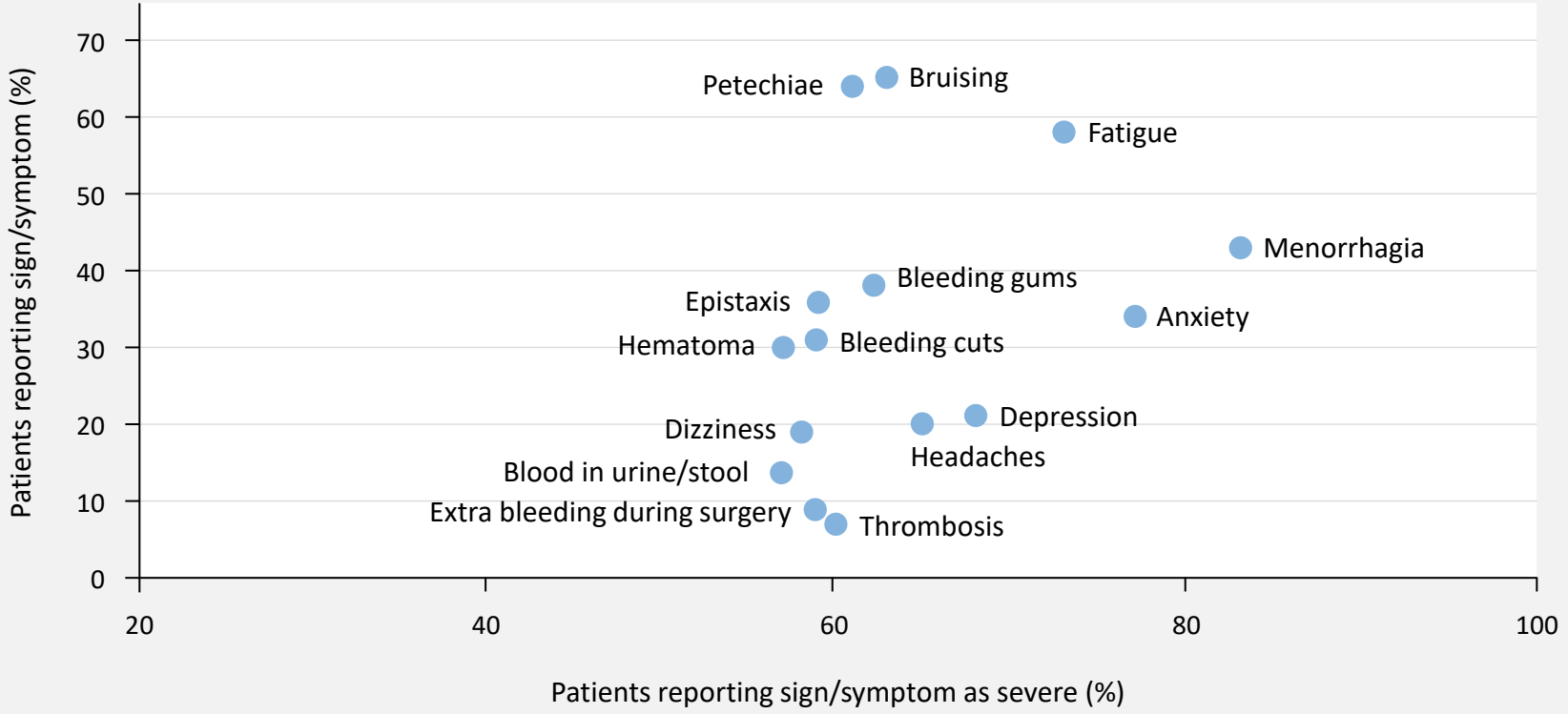
ITP is a Chronic Bleeding Disorder with Multiple Comorbidities

Autoantibody-mediated disease characterized by low platelet counts, leading to bruising, bleeding episodes, hemorrhage, and fatigue

Patient report of ITP symptoms at diagnosis[1]

(I-WISh Survey, n=1,507)



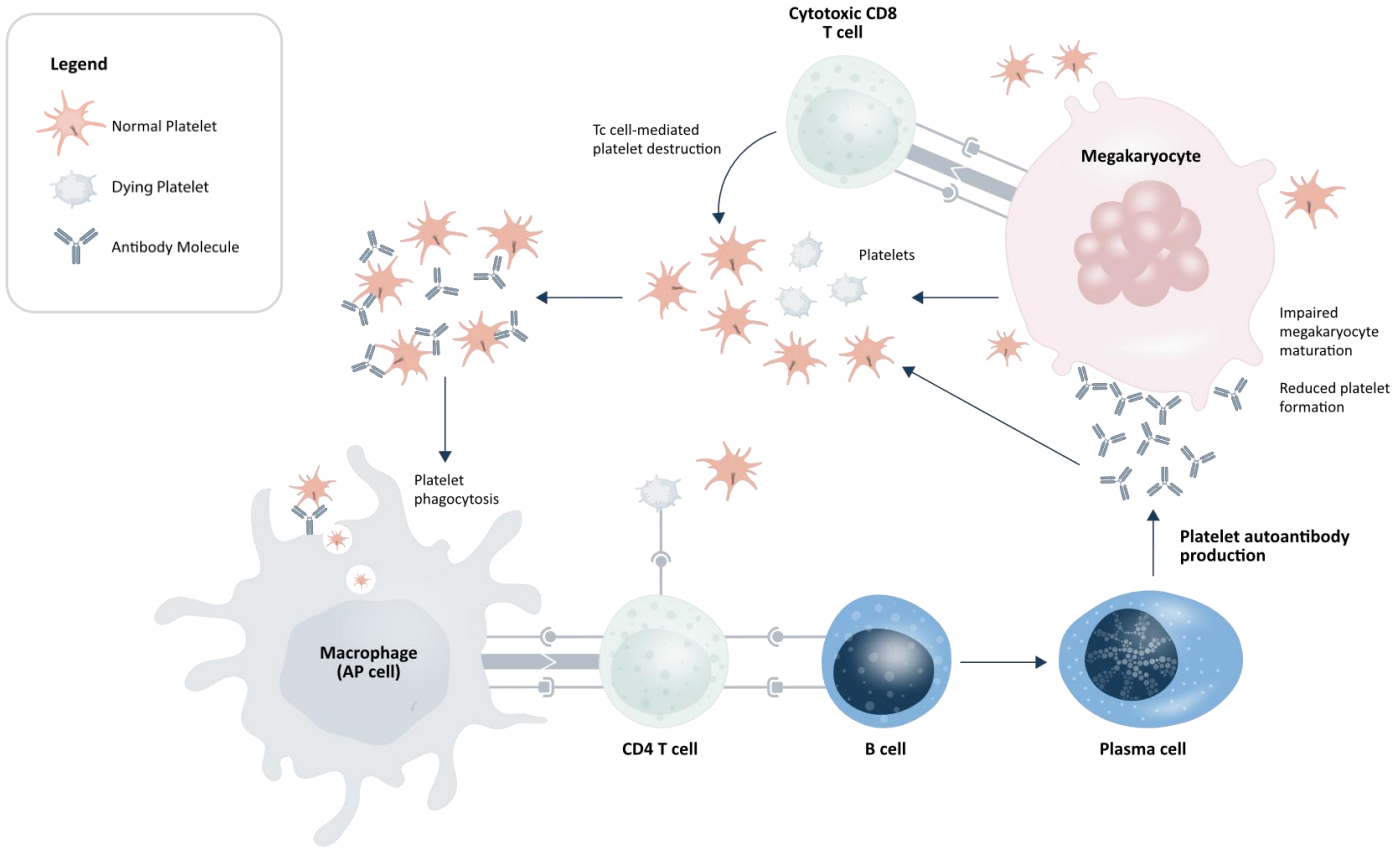
- Typically diagnosed ~55 yrs, increased incidence with age[2,3]
- High clinical burden, driven by steroid dependence and bleeding-related hospitalizations[4]
- Patients experience easy bruising, fatigue, functional limitations, and emotional impact[5]
- Physicians prioritize bleeding prevention, QoL improvement, and normalization of platelet counts[1]

ITP = immune thrombocytopenia , QoL = quality of life

[1] Adapted from Cooper 'Perceptions' AJH 2020, [2] Michel Eur J Haematol 2009, [3] Lawrie Br J Haematol 2023, [4] Kuter ASH 2024, [5] Cooper 'HRQoL' AJH 2020

Anti-CD19 is a Potentially Disease Modifying Approach in ITP

Targeting CD19 has potential to eliminate pathogenic, autoantibody-producing B cells, which are a critical disease driver in ITP



- B cells secrete anti-platelet autoantibodies which bind to platelets and mark them for destruction by macrophages
- Autoantibodies also bind megakaryocytes, impairing platelet production
- Rituximab established B-cell depletion as a therapeutic option in ITP

ITP = immune thrombocytopenia

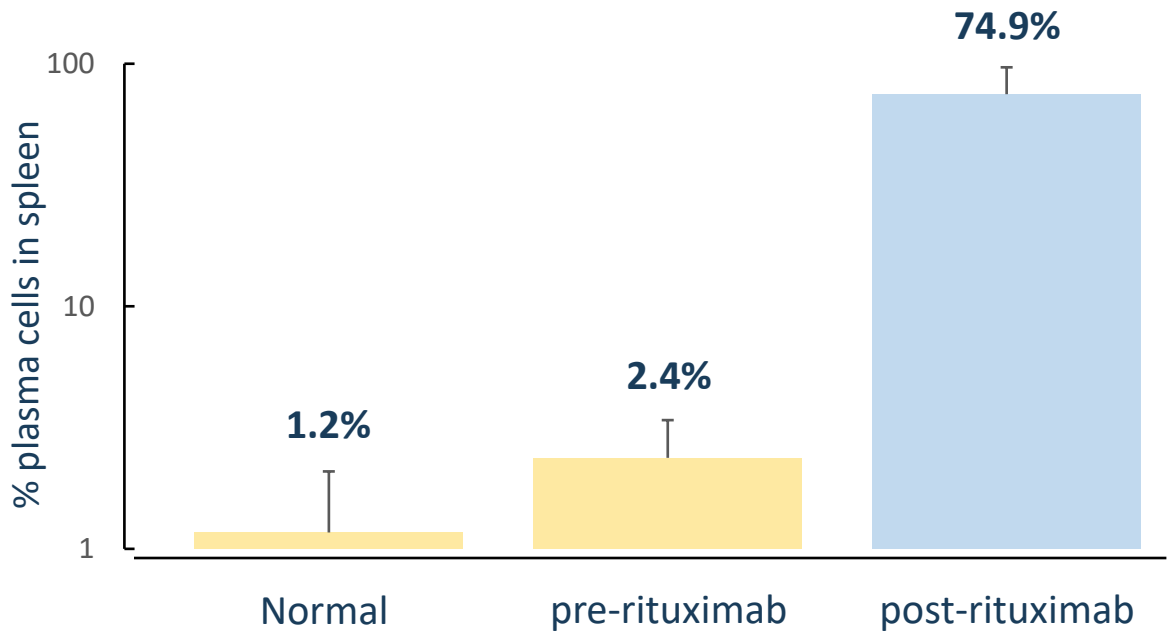
Adapted from Lopez Blood Reviews 2025

Potential for Sustained Elimination of Pathogenic B Cells with CD19

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

- 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]



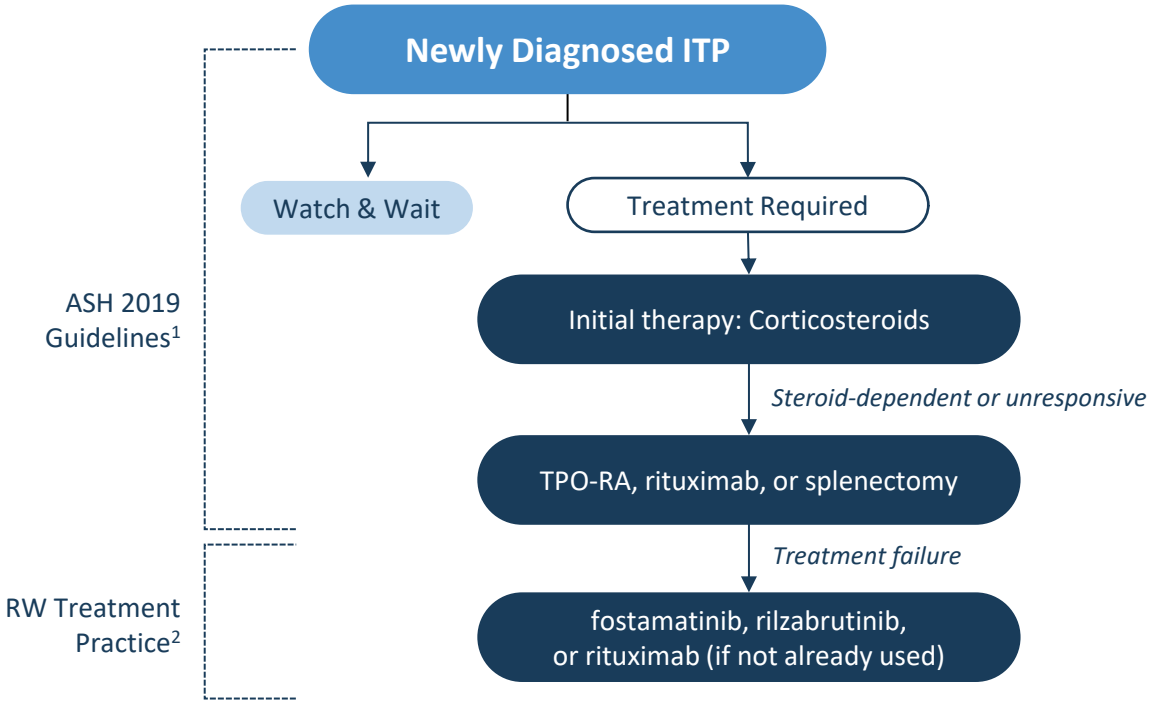
ITP = immune thrombocytopenia, mAbs = monoclonal antibodies

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

ITP Remains Poorly Controlled After Steroid Failure

Limited effective options, with a need for therapies that deliver durable platelet responses with improved safety and lower treatment burden

Corticosteroids remain first line, followed by TPO-RAs or off-label rituximab[1]; SYK/BTK typically used after second line failure[2]



Durable responses remain low (~20%), with meaningful gaps in safety and treatment burden

	Fostamatinib[3] *Approved in 2018*	Rilzabrutinib[4] *Approved in 2025*
MOA	SYK inhibitor	BTK inhibitor
Platelet Response	37-48%	64%
Durable Response	16-18%	23%
Warnings	Hypertension Hepatotoxicity Diarrhea Neutropenia	Hepatoxicity
Dosing	Oral, BID	Oral, BID

Study duration: 24 weeks; Definitions of platelet response and durable response varied across studies

Fostamatinib prior therapy: 94% corticosteroids, 53% immunoglobulins, 48% TPO-RAs

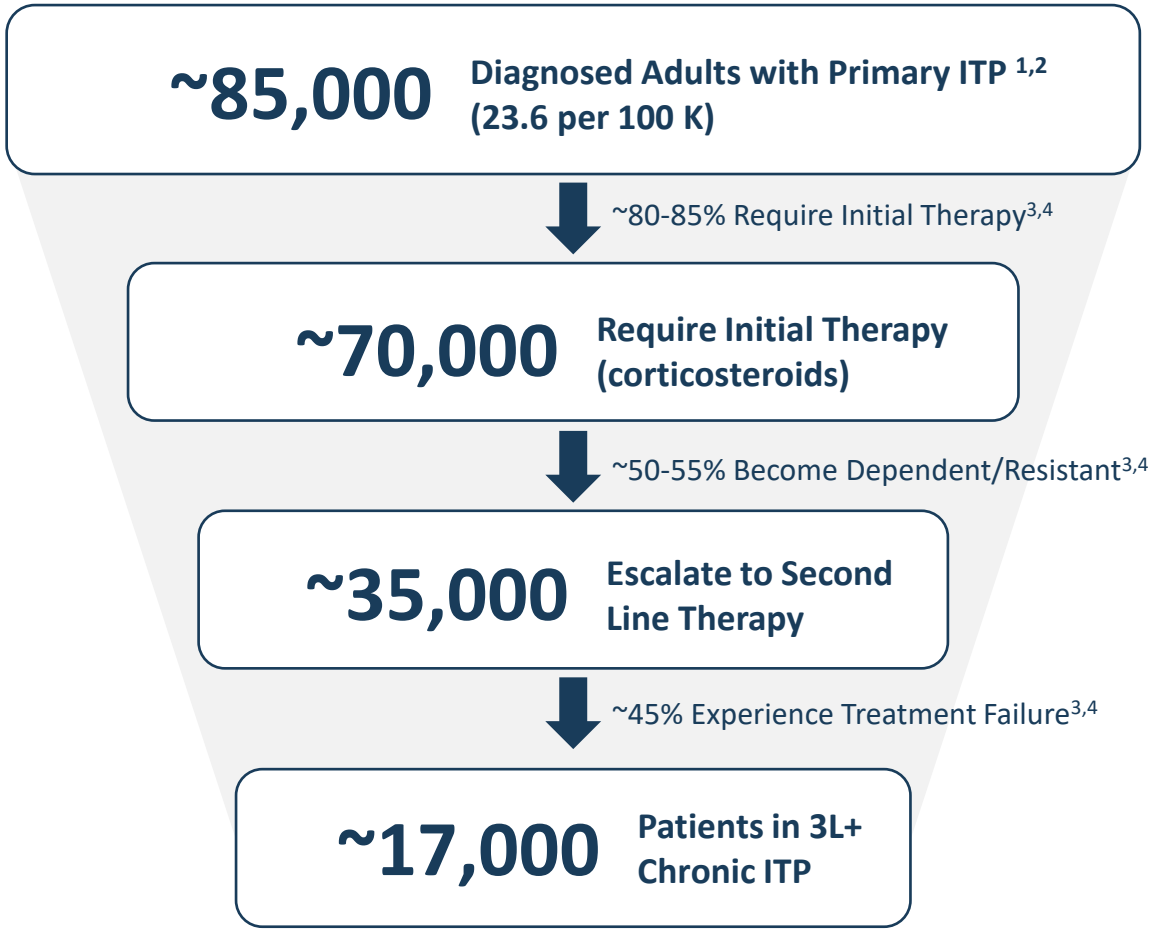
Rilzabrutinib prior therapy: 96% corticosteroids, 55% immunoglobulins, 69% TPO-RAs, 35% rituximab

ASH = American Society of Hematology, BID = twice daily, BTK = Bruton's tyrosine kinase, ITP = immune thrombocytopenia, MOA = mechanism of action, RW = real world, SYK = spleen tyrosine kinase, TPO-RAs = thrombopoietin receptor agonists

[1] ASH 2019 Guidelines for ITP (Neunert Blood Advances 2019), [2] Climb Market Research 2026, [3] fostamatinib prescribing information, [4] rilzabrutinib prescribing information

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

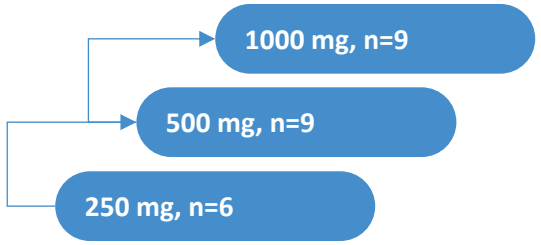
Budoprutug Phase 1b/2a ITP Trial

Multi-center, open-label, dose escalation study enrolling previously treated patients with ITP

Evaluation of 3 doses, ranging from 250-1000 mg

OPEN-LABEL, DOSE ESCALATION STUDY

Up to 9 patients per cohort, enrolled sequentially



Population

- ≥ 18 years of age
- Insufficient response to 1 or more prior therapies
- Platelet count < 30,000/µL; confirmed on 2 occasions
- ≥ 24 weeks since any prior B-cell depleting agent

Recruitment ongoing in 20 sites across 5 countries

- Bulgaria
- Greece
- Serbia
- Spain
- Ukraine

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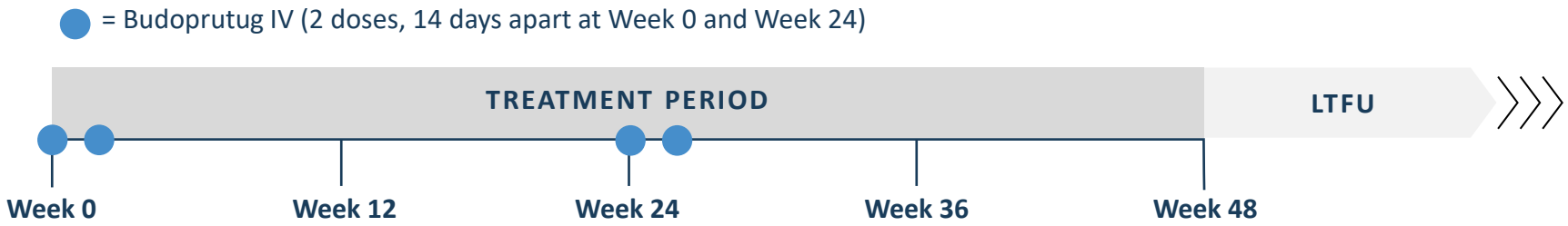
NCT07043946

ITP = immune thrombocytopenia

Budoprutug Phase 1b/2a ITP Trial

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

Platelet response will be assessed during the treatment period, and long-term follow-up will evaluate potential for disease remission



Initial B-cell depletion and platelet data from the low dose cohort (250mg at 24 weeks) anticipated June 2026

Additional data expected by year end

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, IV = intravenous, LTFU = long term follow-up

Marisa

Living with systemic lupus erythematosus

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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 Ab approved for LN[2], and rituximab used off-label[3]

***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[4]

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

mAb approach can overcome key CAR T-cell challenges

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

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

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

Parallel Global and China SLE Studies

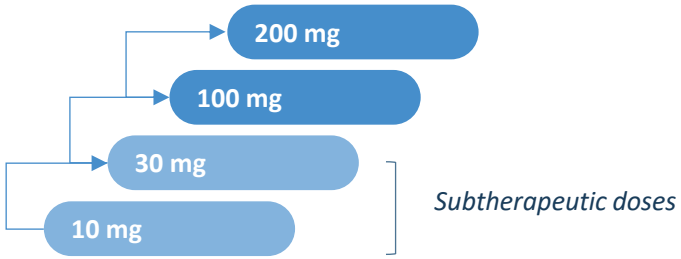
Designed to assess B-cell depletion and kinetics of re-repopulation of B-cell subsets

Global Phase 1b Study

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

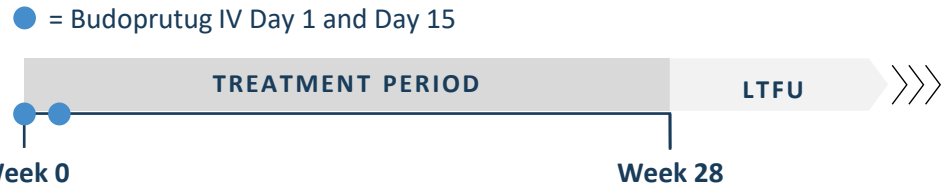
SINGLE ASCENDING DOSE COHORTS, N ~30

Up to 6 patients per cohort enrolled sequentially



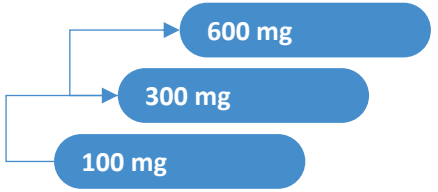
China Phase 1b/2a Study

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



DOSE ESCALATION, N ~18

Up to 6 patients per cohort enrolled sequentially



DOSE EXPANSION, N ~12

Up to 12 patients at dose identified in escalation period

Dose selected; administered as 2 doses, 14 days apart

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

Global study: NCT07011043 China study: NCT07564596

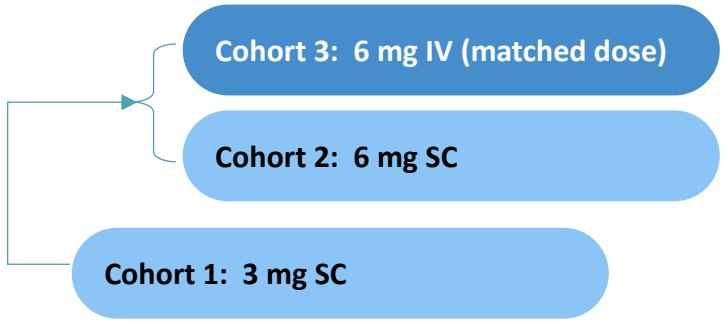
FPI = first patient in, IV = intravenous, LTFU = long term follow-up, SLE = systemic lupus erythematosus, SLEDAI = SLE disease activity index

Budoprutug – Subcutaneous (SC) Formulation

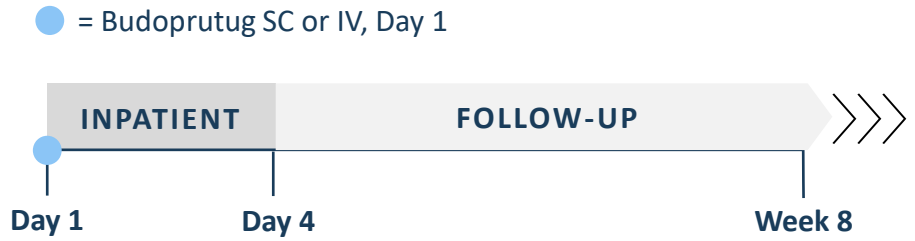
Budoprutug SC Formulation Phase 1 Trial Completed

Evaluation of pharmacodynamic effects of budoprutug SC and IV in healthy volunteers

SINGLE ASCENDING DOSE COHORTS



- 8 subjects per SC cohort (6 budoprutug: 2 placebo)
- 14 subjects in the IV cohort (12 budoprutug: 2 placebo)



Low doses of budoprutug were chosen to achieve non-complete (~50%) peripheral B cell depletion in HV

NCT07090655

HV = healthy volunteers, IV = intravenous, SC = subcutaneous

Follow-up continued for subjects whose B cell count was not above 50% of baseline or above lower limit of normal by week 8

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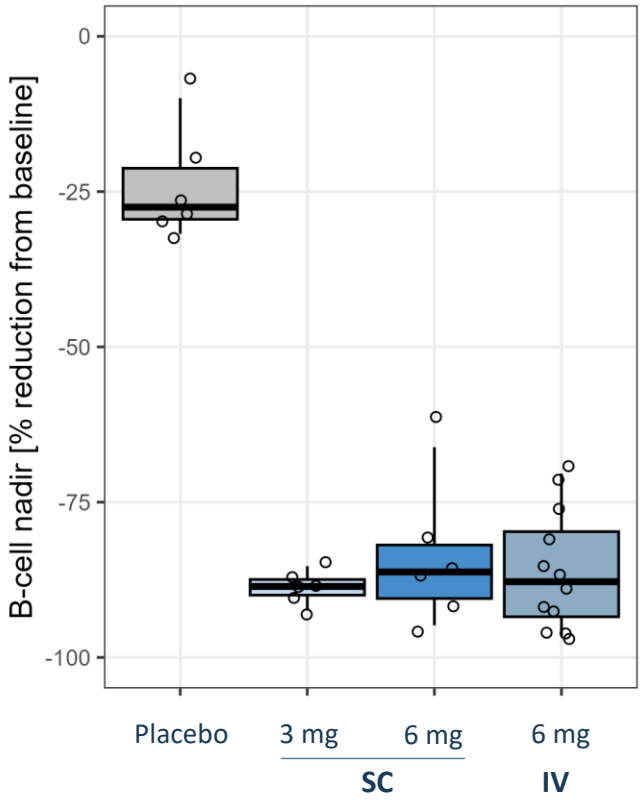
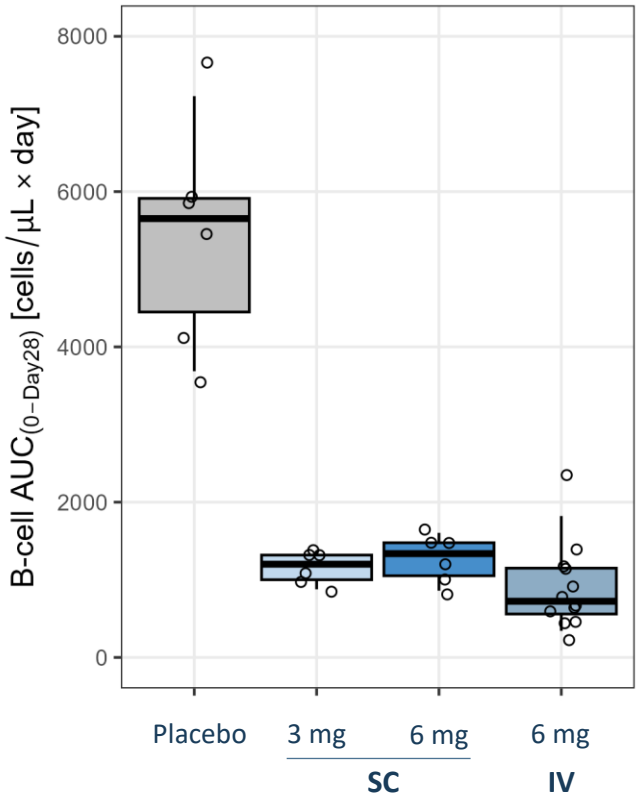
Robust B-cell Depletion Observed with Budoprutug SC in HV

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



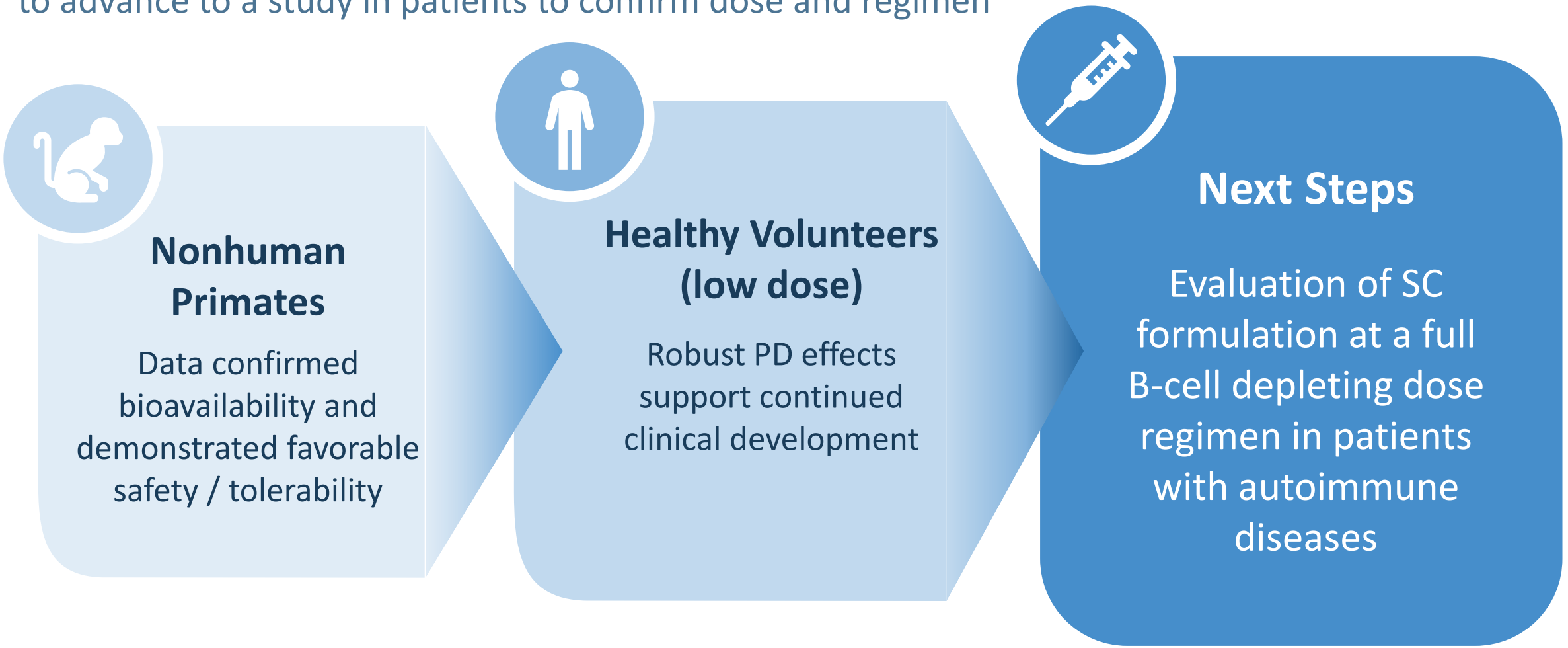


Budoprutug SC was well-tolerated; safety profile consistent between SC and IV

AUC = area under the curve, HV = healthy volunteers, IV = intravenous, SC = subcutaneous
Company data on file

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



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

Laying the Foundation for Budoprutug's Long-Term Potential

Emerging data will further inform safety and the ability to achieve deep B-cell depletion, the optimal dose in renal and non-renal indications, and the potential for long-term disease control

Range of budoprutug doses assessed across studies will inform dose selection for future trials

Dose	pMN	ITP	GL SLE	CH SLE
10 mg			◆	
30 mg			◆	
100 mg			◆	◆
200 mg	◆		◆	
250 mg		◆		
300 mg				◆
500 mg		◆		
600 mg	◆			◆
1000 mg	◆	◆		

Anticipated 2026 Data Readouts

- ✓ SC data in healthy volunteers
- ITP, B cell & platelets, low dose – June
- pMN, B cell & PLA2R, low dose – Q4
- Global SLE, B cell & Ab, low doses – Q4
- ITP, additional data – YE

Anticipated Study Milestones & Next Steps

- China SLE – FPI Q2 '26
- SC – Initiate multiple dose patient study

Ab = antibody, CH = China, FPI = first patient in, GL = global, ITP = immune thrombocytopenia, PLA2R = phospholipase A2 receptor, pMN = primary membranous nephropathy, SC = subcutaneous, SLE = systemic lupus erythematosus

Budoprutug: A Differentiated CD19 mAb with Expansive Potential

Validated CD19 Target
CD19 is broadly expressed across the B-cell lineage, including early B cells and antibody-producing cells—populations not fully addressed by an anti-CD20 approach

Monoclonal Antibody Design
Potential to combine potency with the scalability and clinical familiarity characteristic of monoclonal antibodies

Core Indications Informed by Biology and Market Opportunity
Targeting pMN, ITP, and SLE, diseases united by B-cell pathology and high unmet medical needs

Market White Space
CD19 monoclonal antibodies remain underrepresented in immune-mediated diseases, creating a compelling opportunity for leadership

Meaningful Near-Term Milestones
Upcoming data expected to define dose, durability, and clinical profile for budoprutug

ITP = immune thrombocytopenia; mAb = monoclonal antibody, pMN = primary membranous nephropathy, SLE = systemic lupus erythematosus

Q&A Session

Aoife Brennan, M.B., Ch.B.
President and CEO, Climb Bio

Edgar Charles, M.D.
Chief Medical Officer, Climb Bio

David Jayne, M.D.
Professor, University of Cambridge

Perrin Wilson, Ph.D.
Chief Business Officer, Climb Bio

Susan Altschuller, Ph.D., MBA
Chief Financial Officer, Climb Bio