

Investor Day Presentation

OCTOBER 2024



Forward Looking Statement

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, Inc. (“Climb Bio”); the anticipated benefits of the acquisition of Tenet Medicines, Inc.; expectations regarding budoprutug’s therapeutic benefits, clinical potential and clinical development; the trial design for planned clinical trials of budoprutug; plans to optimize the administration of budoprutug; the anticipated timelines for initiating clinical trials of budoprutug; 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 acquisition of Tenet Medicines, Inc.; 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 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 of budoprutug; competing successfully with other companies that are seeking to develop treatments for systemic lupus erythematosus, immune thrombocytopenia and membranous nephropathy and other immune-mediated diseases; maintaining or protecting intellectual property rights related to budoprutug and/or its other product candidates; managing expenses; and raising the substantial additional capital needed, on the timeline necessary, to continue development of budoprutug 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.

Introduction & Corporate Update

Aoife Brennan, MD | President & CEO



Together We Can Reach Higher Ground

At Climb Bio, we believe elevating relationships leads to more meaningful insights, better answers, and ultimately, to more inspired medicines for patients living with immune-mediated diseases



More than 2.5 million Americans suffer from a B-cell mediated disease



Committed to enhancing the patient experience



Driven to becoming a leader in development for immune-mediated diseases

Team Highlights

Building a highly-credentialed and experienced development organization focused on execution



Aoife Brennan
President and CEO



Jan Hillson
Senior Clinical Advisor



William Bonificio
Interim CBO



Janaki M. Subramanyam
VP, Regulatory Affairs



Nishi Rampal
SVP, Clinical Development



Emily Pimblett
CAO



Brett Kaplan
COO



Stephen Thomas
Director & Interim CSO

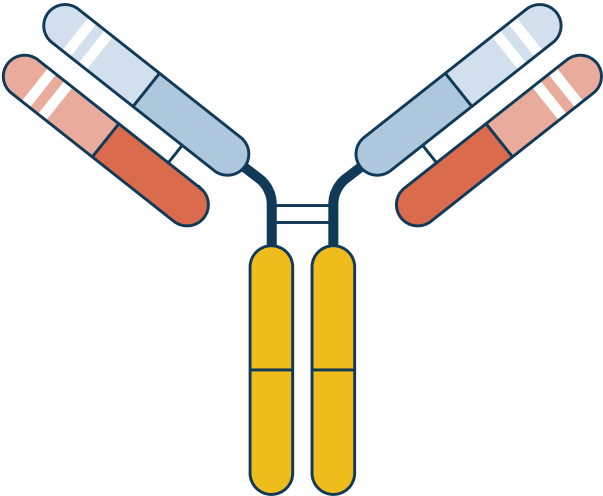


Jay Mitchell
VP, Clinical Operations



Kate Hecht
SVP, Program Management

Budoprutug: Anti-CD19 mAb Designed to Treat a Broad Range of B-Cell Mediated Diseases



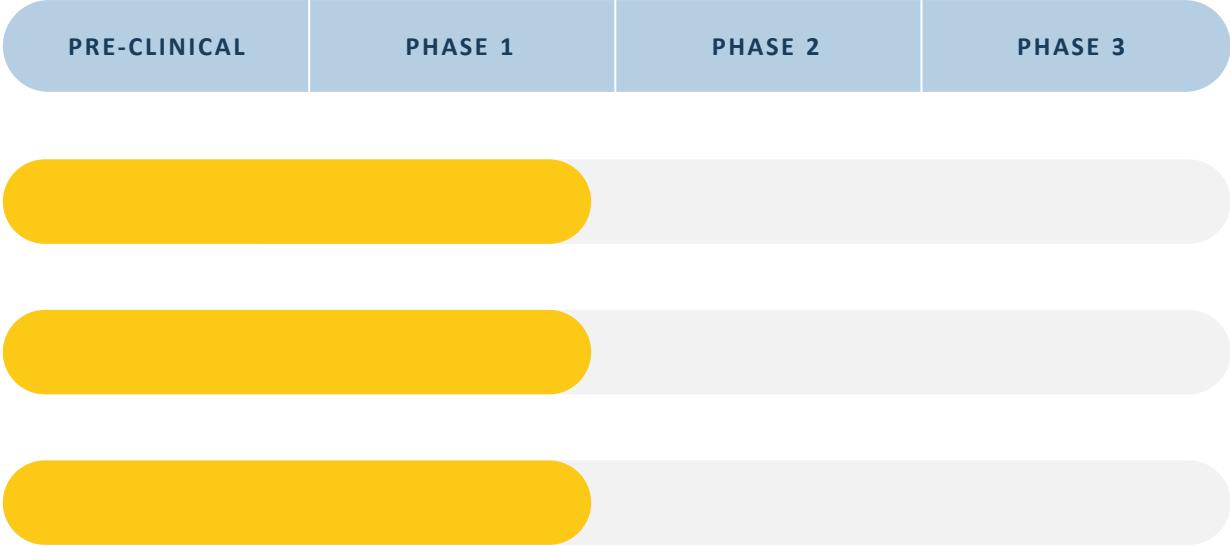
Budoprutug
Anti-CD19
Fc⁺, mAb

INDICATION(S)

Membranous Nephropathy

Systemic Lupus Erythematosus*

Immune Thrombocytopenia*



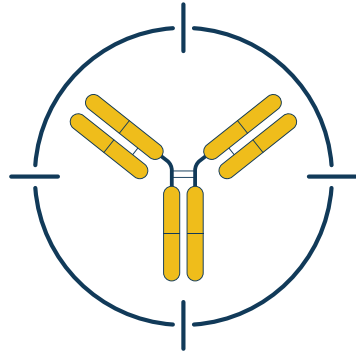
CD19 is a promising target antigen for AAb-mediated diseases as a clinically-validated MoA

Additional potentially addressable indications across multiple therapeutic areas

Aiming to advance potentially best-in-class mAb to late-stage clinical trials

Corporate Strategy & Vision

Climb is well-positioned to advance budoprutug across three distinct opportunity sets



Primarily IgG4-Mediated

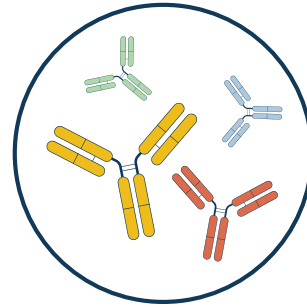
Clear pathophysiology supporting targeting of CD19-expressing B-cells

Lead indication

Membranous Nephropathy

Opportunity to Differentiate

Potential for “immune reset”, improved efficacy



Complex Systemic

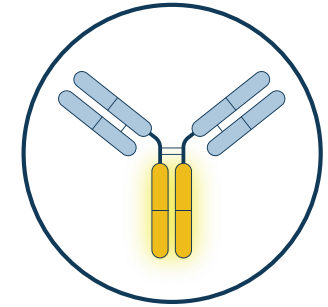
Multi-organ, systemic diseases with heterogenous patient populations

Lead indication

Systemic Lupus Erythematosus

Opportunity to Differentiate

Improve efficacy while balancing safety, tolerability, and convenience



Primarily Single Organ IgG1 - 3

Orphan diseases with compelling clinical proof-of-concept using B-cell depletion

Lead indication

Immune Thrombocytopenia

Opportunity to Differentiate

Demonstrate efficacy in relapsing and/or refractory patients

R&D Day Agenda

October 15, 2024 | 12.00 – 02.00 ET | Virtual Webcast



Introduction & Corporate Update

Aoife Brennan, President and CEO



Scientific Background

Stephen Thomas, Director and Interim CSO



IgG4-Mediated Disease Opportunity

Frank Cortazar, KOL, PI



Complex Rheumatologic Disease Opportunity

Jan Hillson, Senior Clinical Advisor



Validated Rare Disease Opportunity

Nishi Rampal, SVP, Clin Dev



Corporate Outlook

Brett Kaplan, COO



Q&A

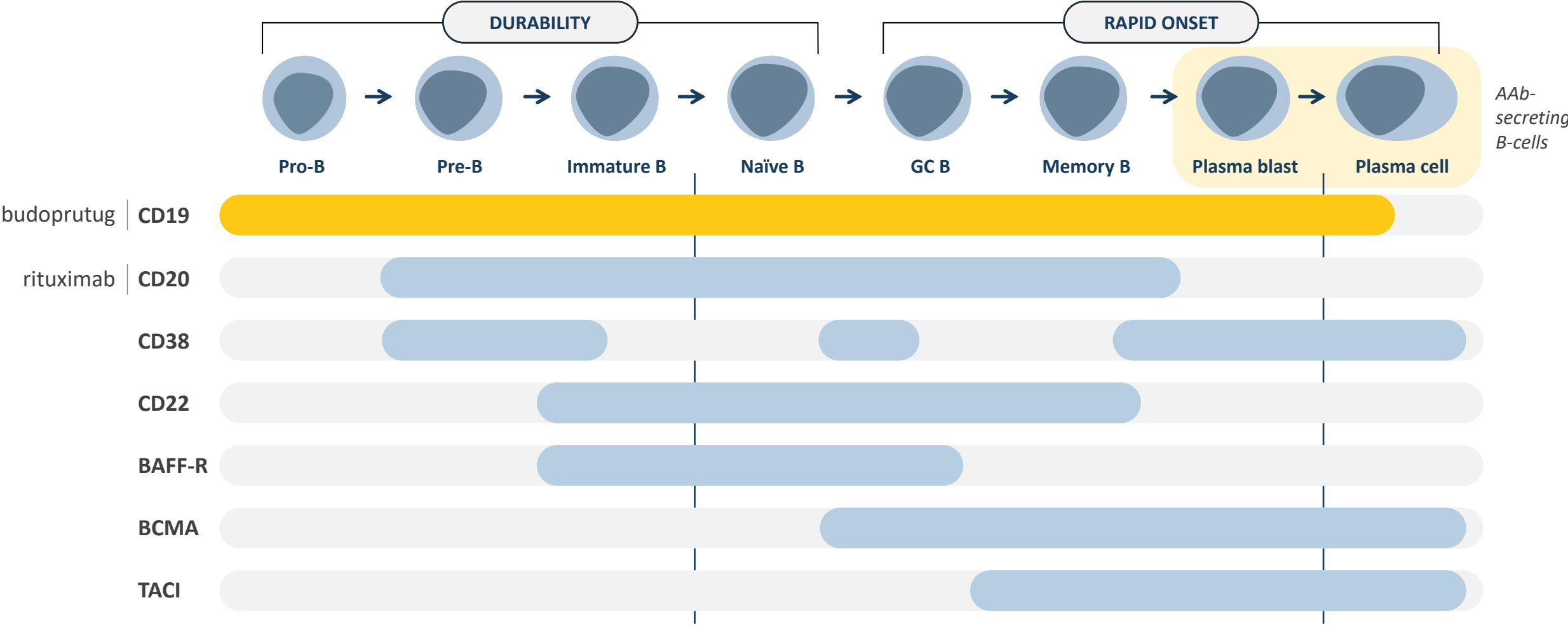
Moderated by Aoife

Scientific Background

Stephen Thomas, PhD | Director and Interim CSO

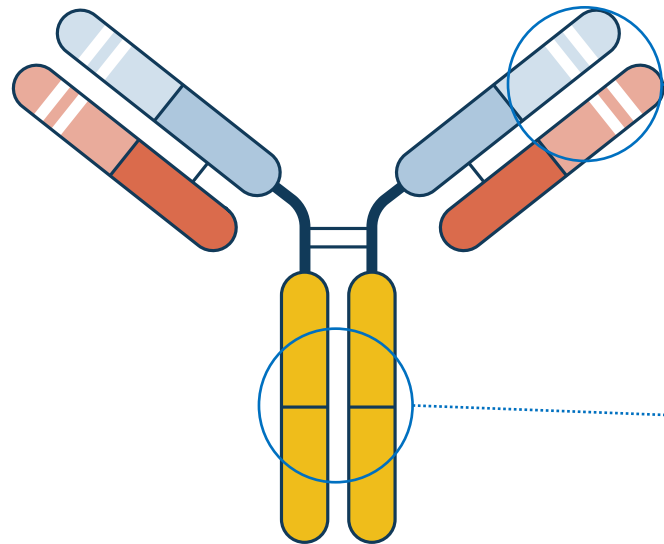
CD19 Expression on Autoantibody-Secreting Cells (ASCs)

CD19-targeted therapy potentially enables rapid onset and durability



Budoprutug: Fc-Enhanced Anti-CD19 mAb

Designed to treat immune-mediated diseases



Budoprutug is a highly potent anti-CD19 mAb containing a low-fucosylated Fc region, leading to enhanced effector function and highly potent ADCC

Unique attributes driving differentiation & positioning

HIGH AFFINITY

18 pM

binding affinity to CD19 to counter low antigen density

ADCC-ENHANCED

>100x potency

vs. wild-type IgG1 to drive deep & durable B cell depletion

HIGH CONCENTRATION

≥175 mg/mL

with low viscosity for low volume, SC injection

✓ **Potential for best-in-class efficacy**

Rapid, deep, and durable B cell depletion at doses as low as 100 mg

✓ **Opportunity for patient-tailored approach to treatment**

Potential to provide IV and/or SC offerings where favorable to patient and point-of-care

✓ **Optimized dosing and tolerability**

Potential for induction and maintenance dosing paradigm with favorable safety, tolerability profile

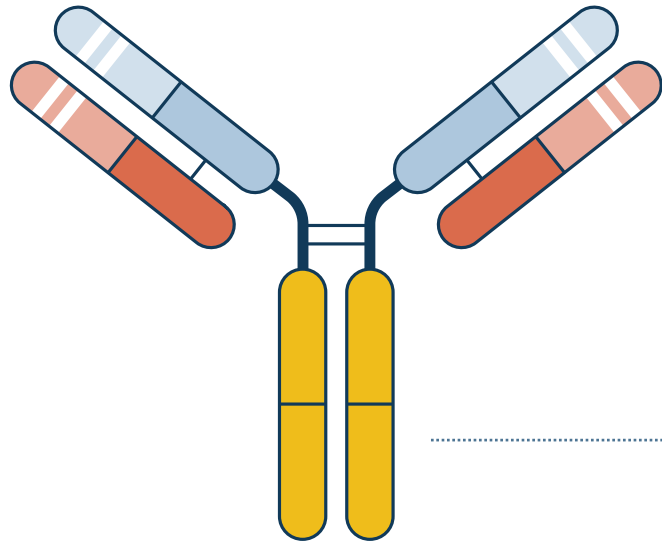
✓ **Pipeline-in-a-molecule potential**

3 distinct opportunity sets:

IgG4-Mediated, Complex Systemic, & Primarily Single Organ IgG1 – 3

Why a Monoclonal Antibody?

Key aspects of budoprutug potentially support a differentiated target product profile



Budoprutug is a highly potent anti-CD19 mAb containing a low-fucosylated Fc region, leading to enhanced effector function and highly potent ADCC

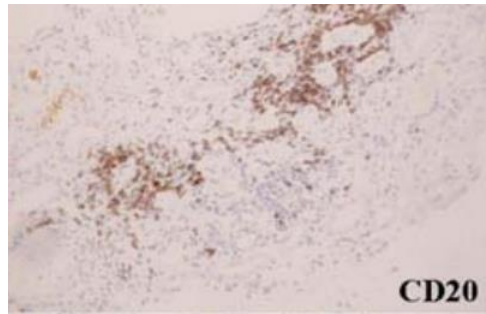
- ✓ **Manufacturability: *The Right Construct***
Monoclonal antibodies typically have well-established manufacturing and supply chains, favorable cost-of-goods, & scalability
- ✓ **Efficacy: *The Right Target***
CD19-targeted therapies have demonstrated impressive efficacy in controlled trials and case reports of patients with autoimmune diseases
- ✓ **Safety & Tolerability: *The Right Modality***
Anti-CD19 mAbs have demonstrated a favorable safety and tolerability profile to date, comparable to anti-CD20s
- ✓ **Patient-Tailored: *The Right Dosing Regimen***
Opportunity for induction and maintenance, treat-to-target approach is highly differentiated from typical biologics in I&I

Naked mAbs Have Demonstrated Tissue-Level B-cell Depletion

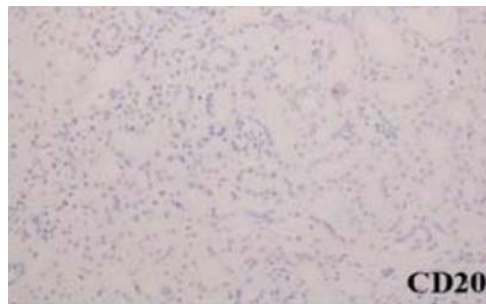
Rituximab (RTX) depleted B-cells in patient tissues, though left behind CD20-negative B-cells

KIDNEY

Before RTX

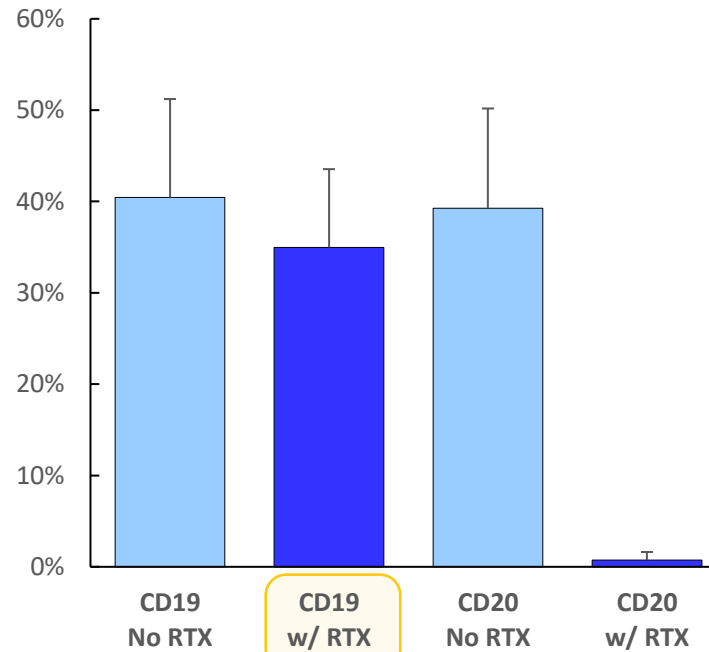


After RTX



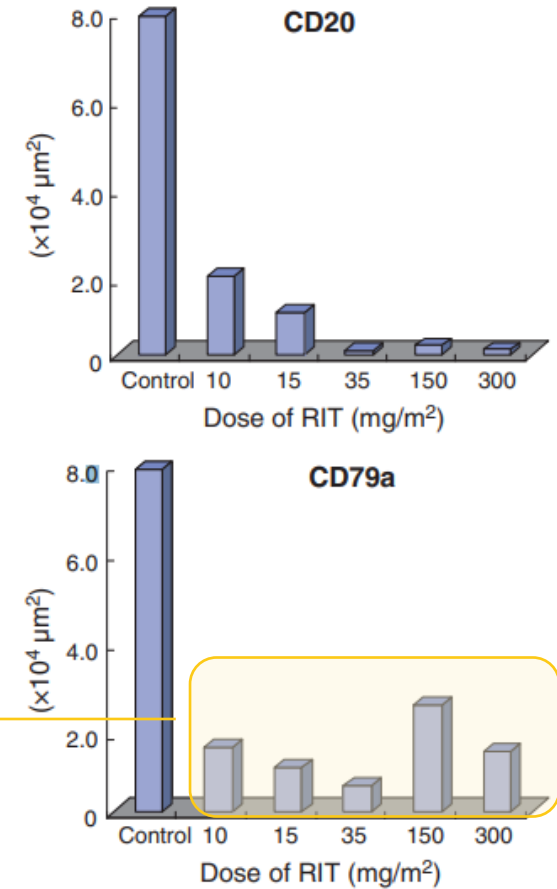
Lack of CD20⁺ B-cell staining in tissue after treatment with RTX

LYMPH NODE



CD19⁺ and CD79a⁺ B-cells are not effectively cleared from tissue after RTX, potentially due to low or no CD20 expression

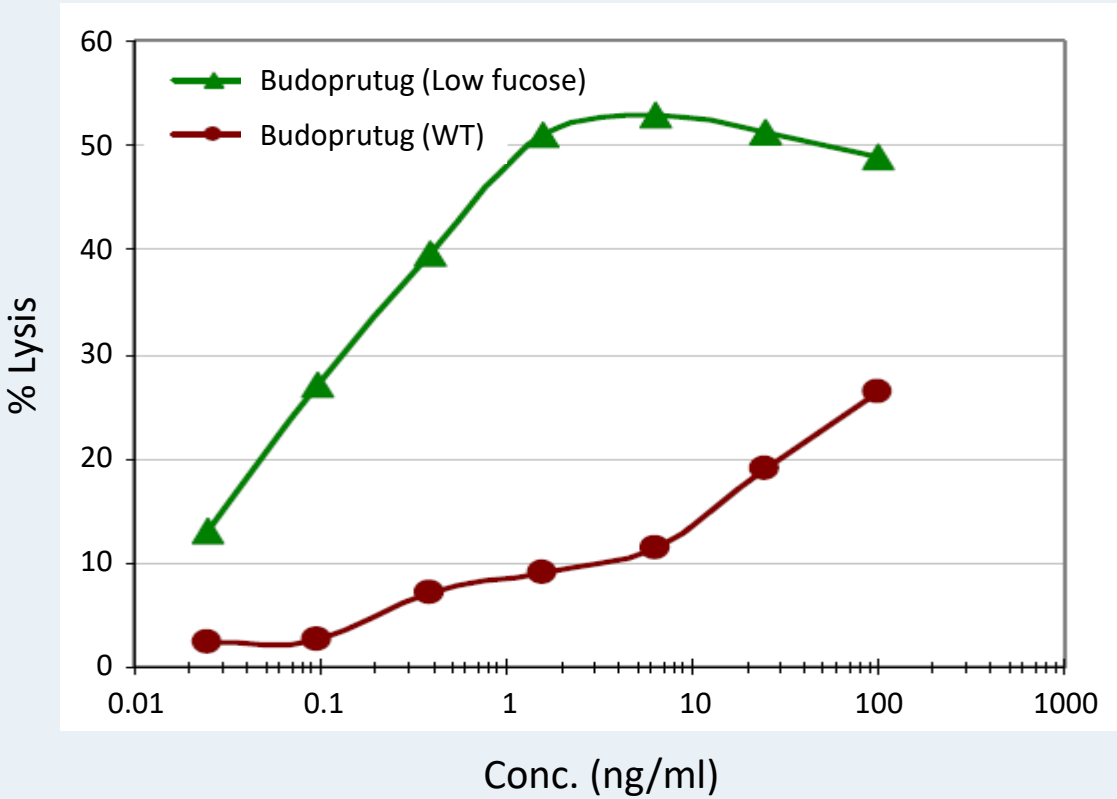
SPLEEN



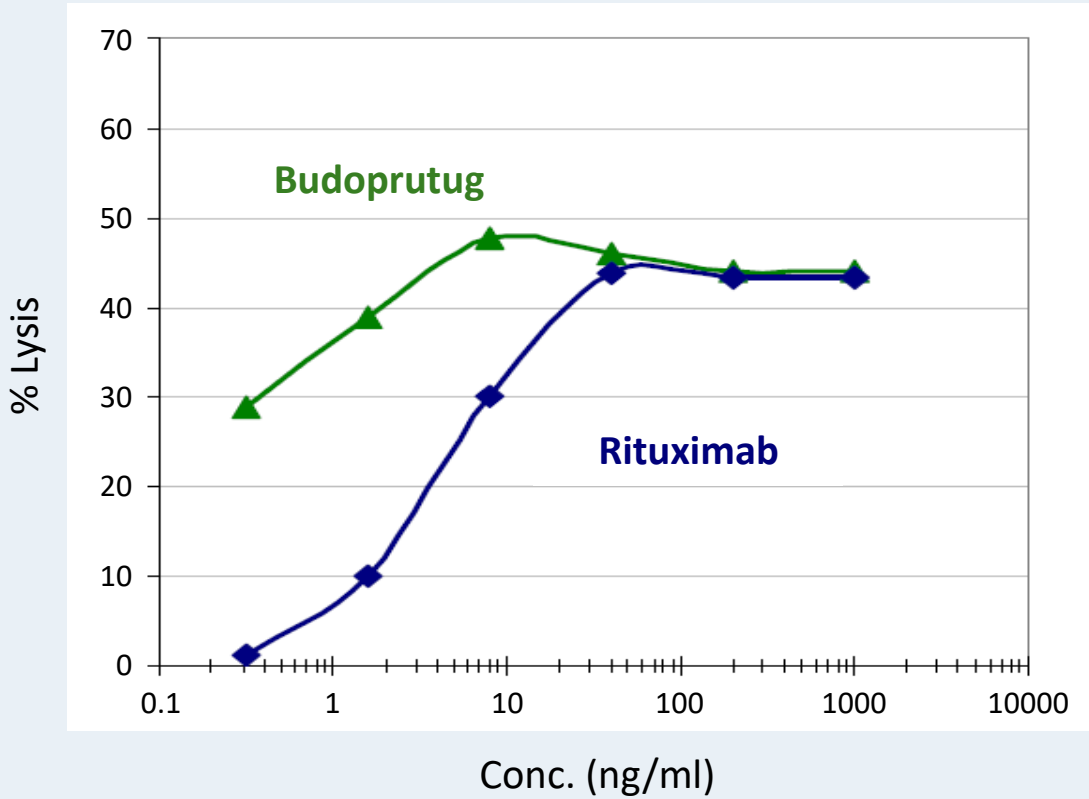
Low Fucosylation Dramatically Enhances Potency

Budoprutug demonstrates potent ADCC, with no detectable CDC in cell-based models

Comparison of in vitro ADCC of budoprutug with low-fucose vs wild-type (WT) IgG1



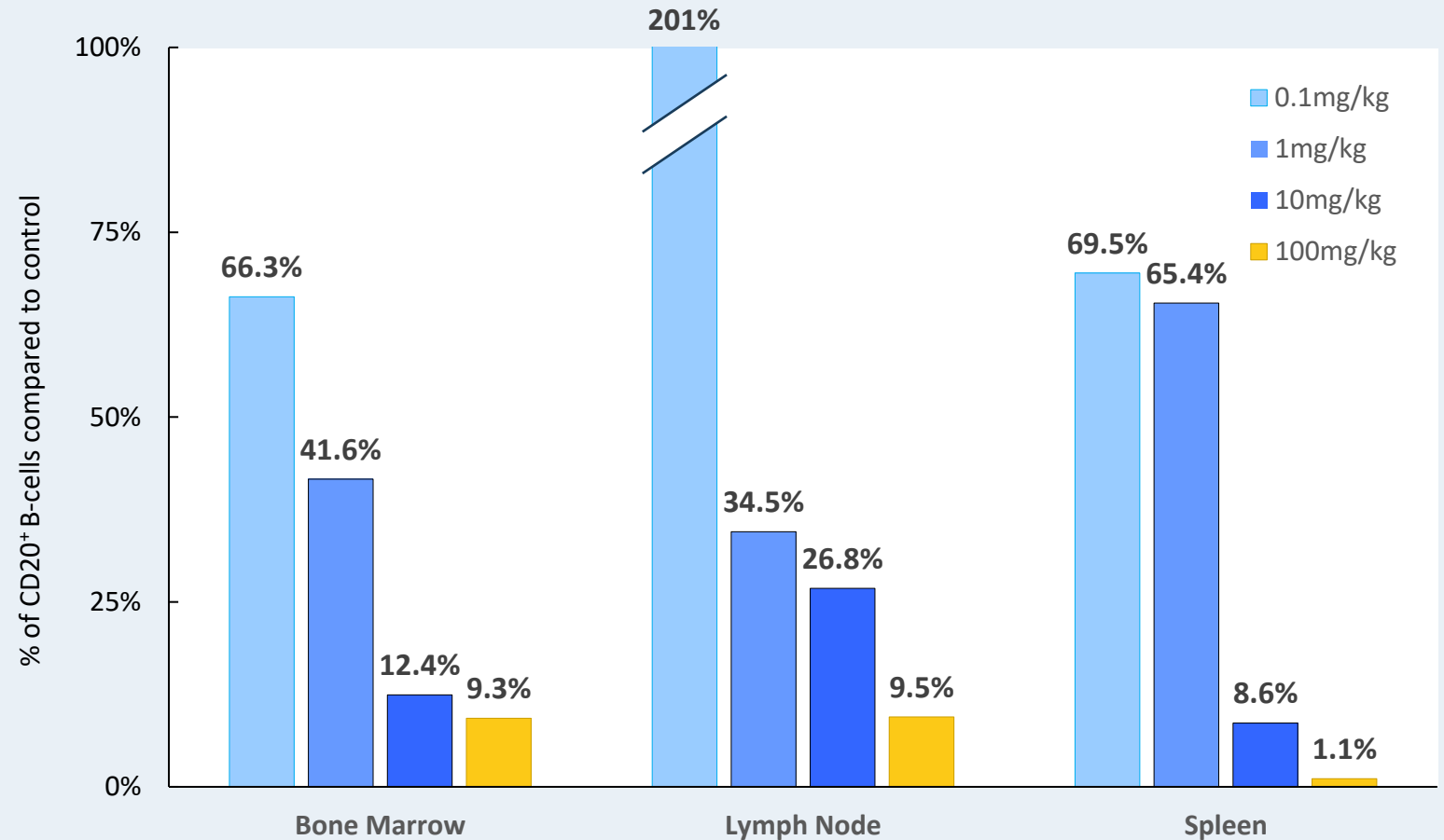
Comparison of in vitro ADCC of budoprutug vs. rituximab against Daudi lymphoma cells



Budoprutug Achieved Depletion of Tissue-Resident B-cells In Vivo

huCD19 transgenic mice treated with weekly doses (QWx4) of budoprutug for 28 days

- B-cell measurements in tissue were taken 7 days post-dosing
- Dose-dependent B-cell depletion in all tissues analyzed at Day 28
- >90% depletion in all tissues at 100 mg/kg



Budoprutug Biomarker Strategy

Opportunity to help accelerate proof-of-concept, benchmark within competitive landscape

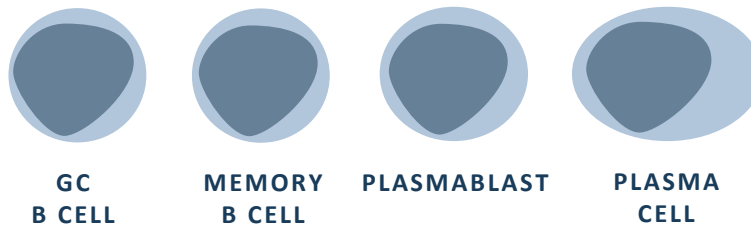
Autoantibody Reduction

- Level, duration and breadth of disease-related autoantibody reduction



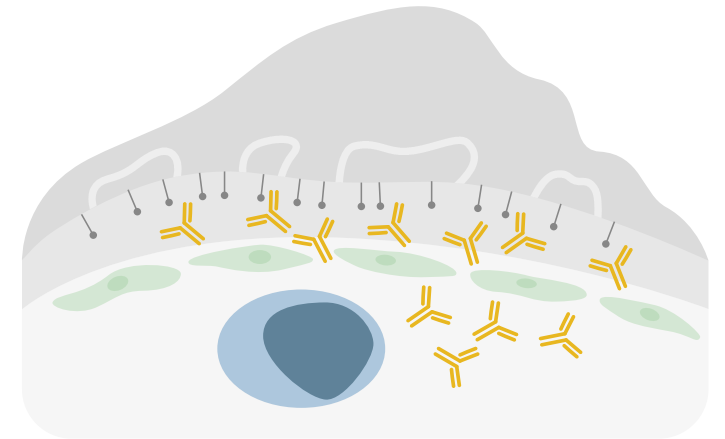
Peripheral B-cell Depletion (& Recovery)

- Level, duration and breadth of peripheral B-cell depletion
- Time to peripheral B-cell recovery (with immunophenotyping)



Tissue B-Cell Depletion

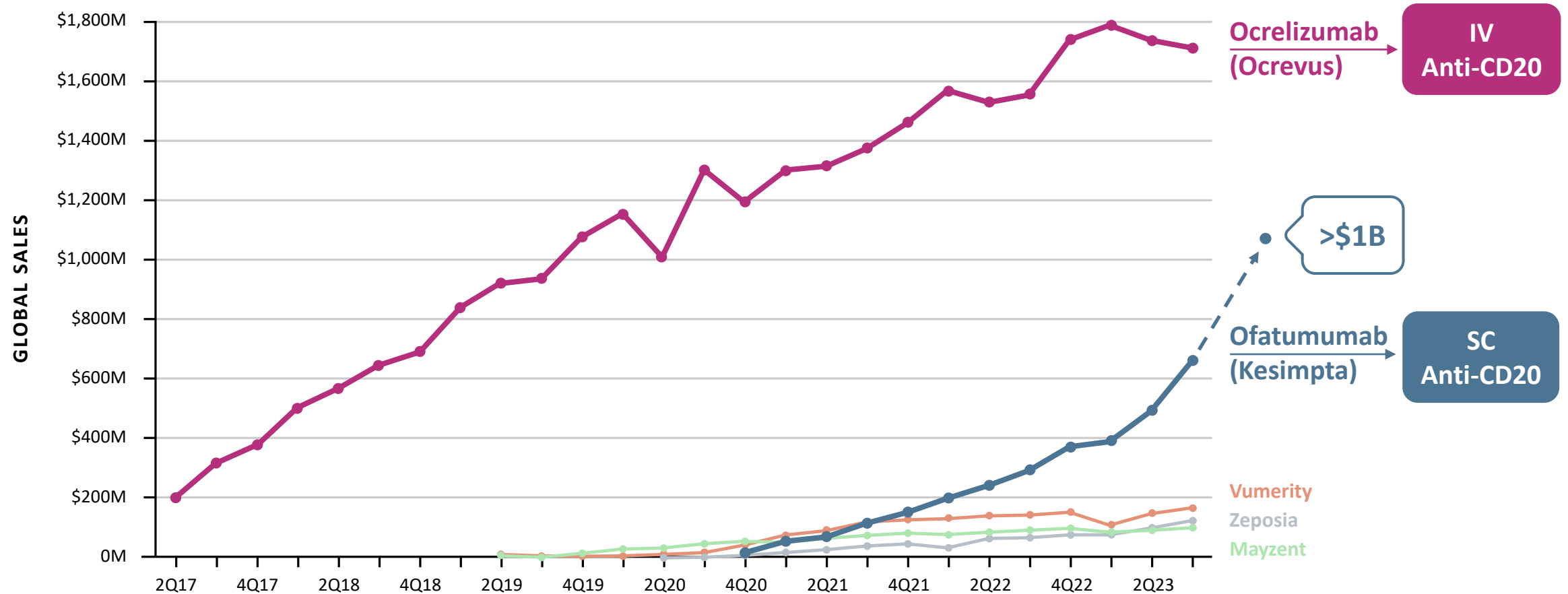
- Level, duration and breadth of tissue-level (i.e., lymph node, spleen, kidney) B-cell depletion



Subcutaneous Dose Form May Enable Further Differentiation

SC dosing presents the opportunity for product line extension and patient-tailored solution(s)

Real-world example showing SC dose form gaining appreciable market share relative to IV within same modality & MoA



Targeting CD19 in autoantibody-mediated glomerular disease

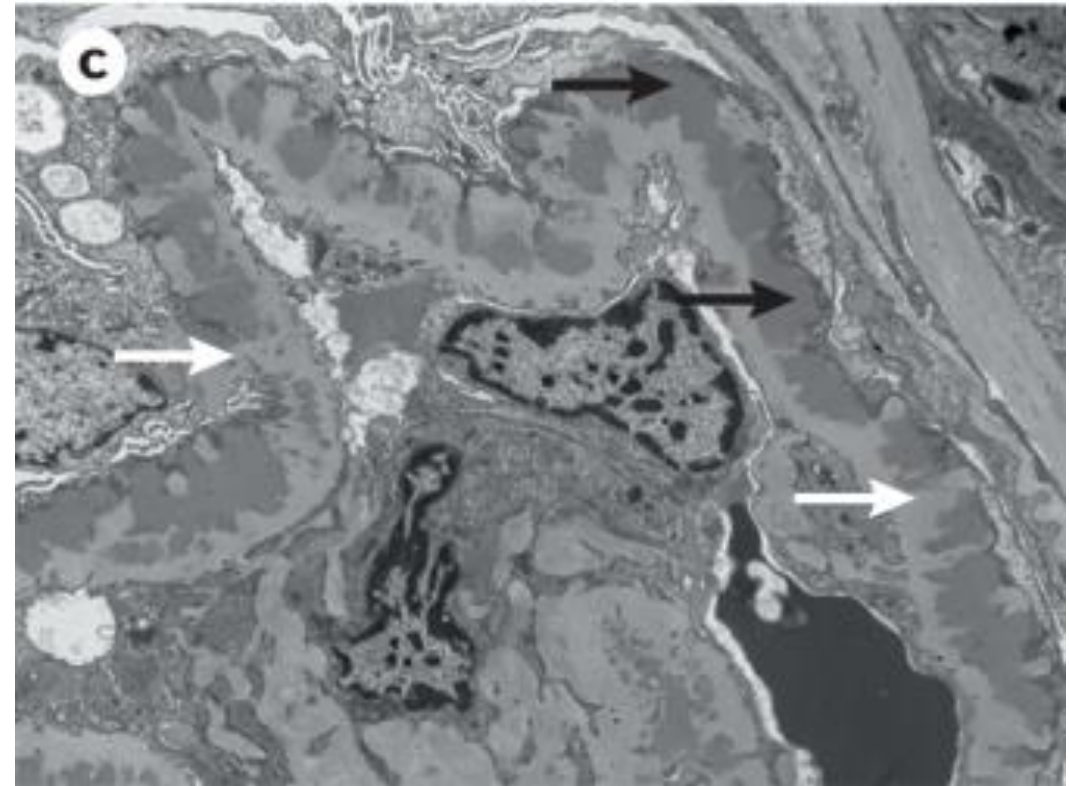
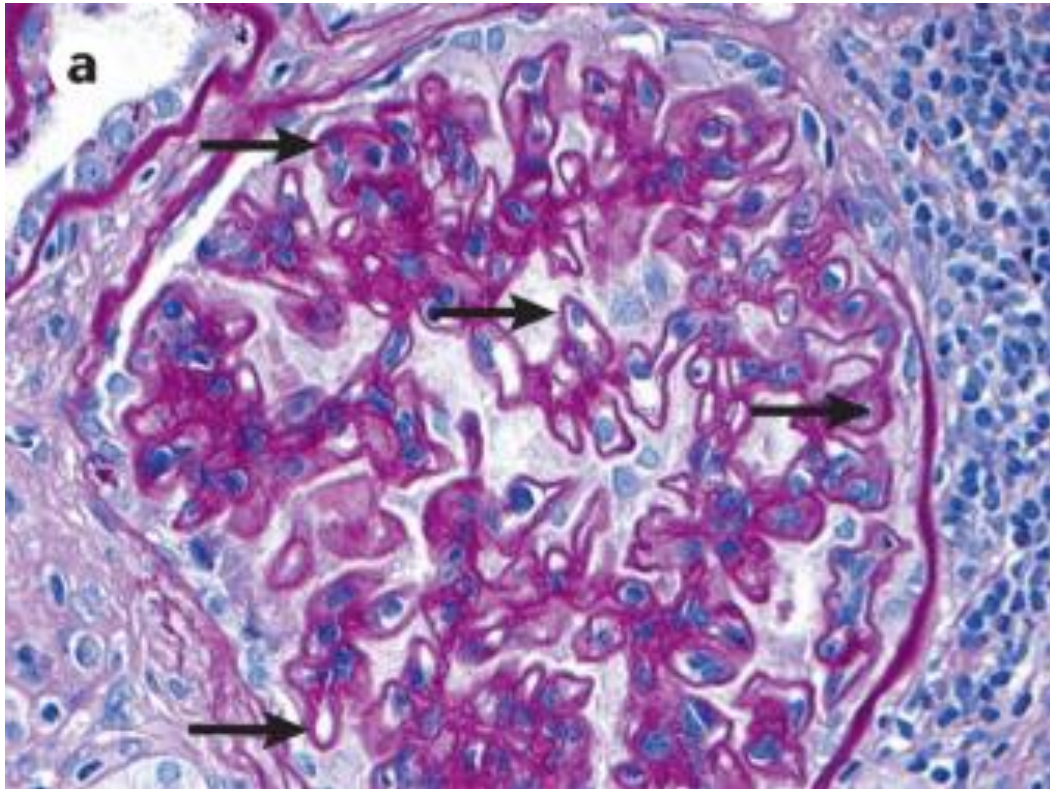
Frank B Cortazar, MD



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Membranous Nephropathy



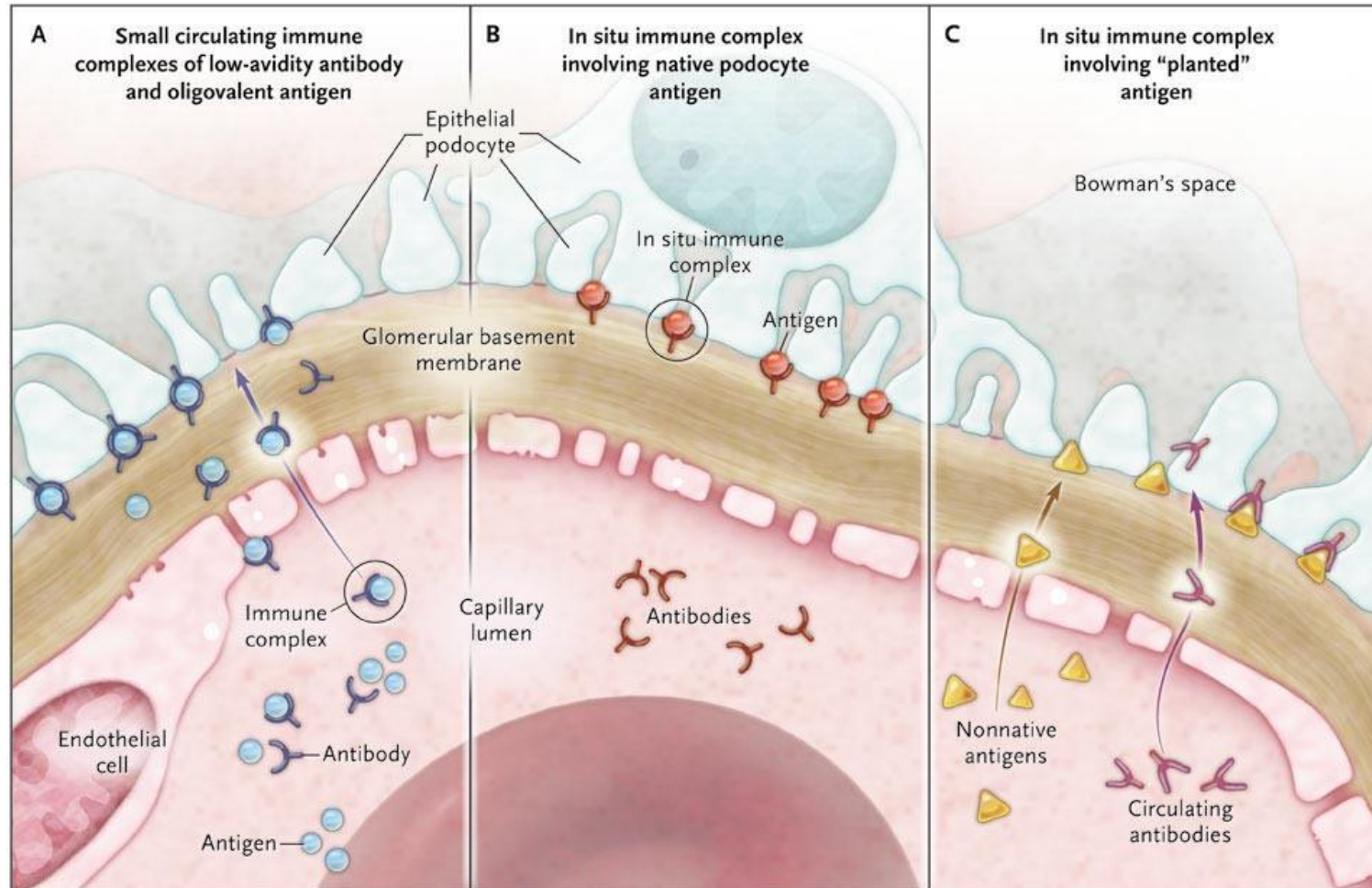
Nat Rev Dis Primers 7, 69 (2021).

Membranous Nephropathy Epidemiology

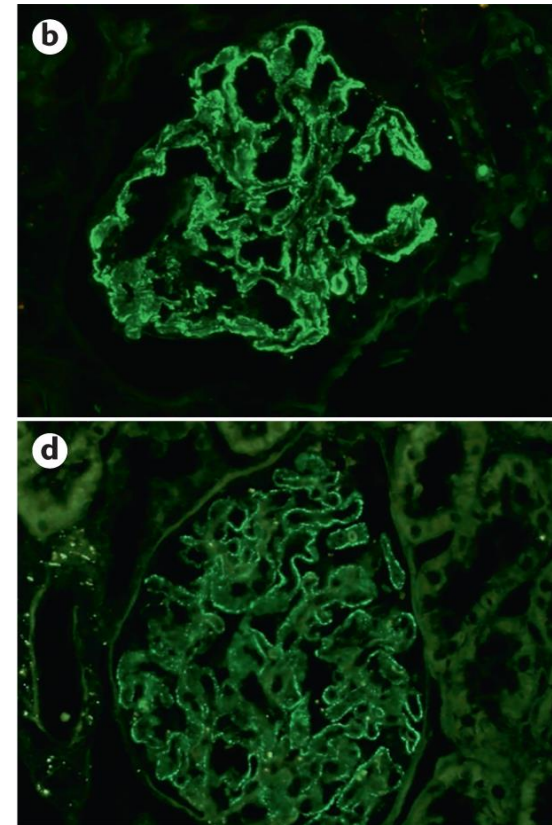
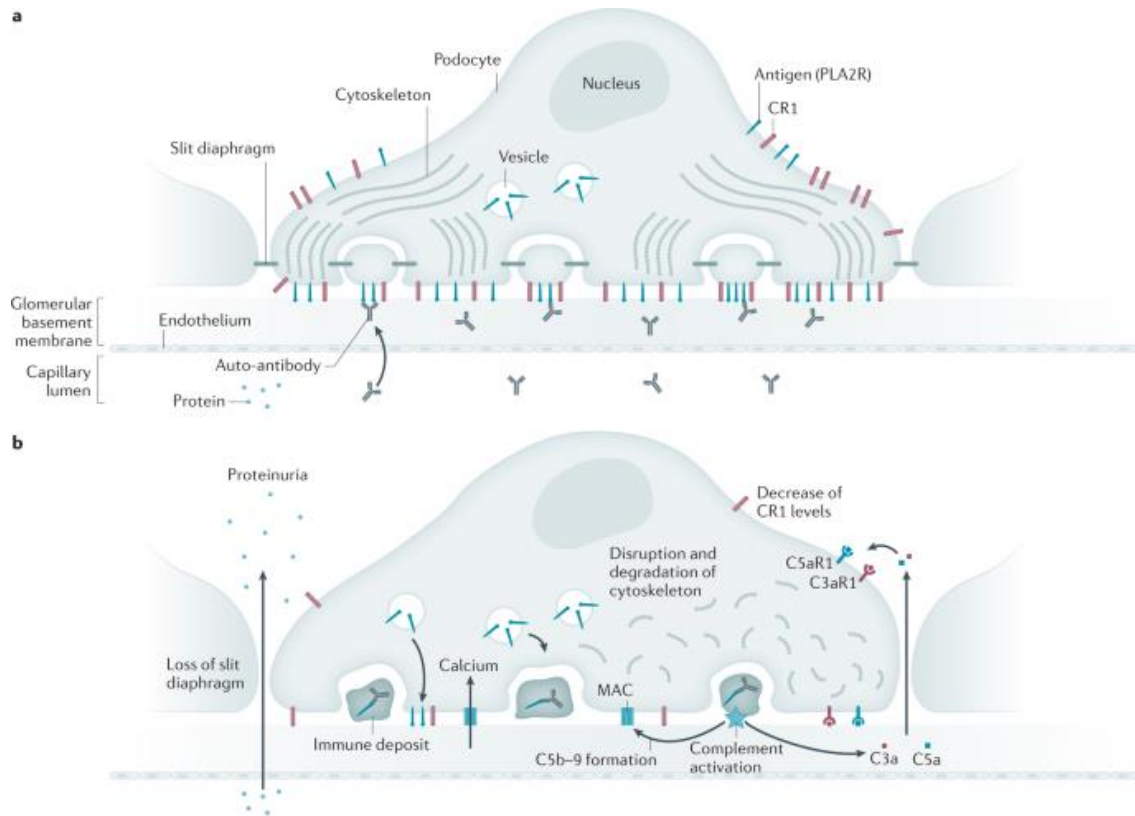
- Most common cause of nephrotic syndrome in non-diabetic adults
 - Incidence 8-10 cases/million
- **“Primary” Membranous (80%)**
 - Phospholipase-A2 receptor (PLA2R): 70-80%
 - Thrombospondin type 1 domain-containing 7a (THSD7A): 2-5%
 - Others
- **Secondary Membranous (20%)**
 - Autoimmune
 - Infection
 - Drugs
 - Malignancy



Immune Complex Formation



PLA2R-associated MN



Nat Rev Dis Primers 7, 69 (2021).

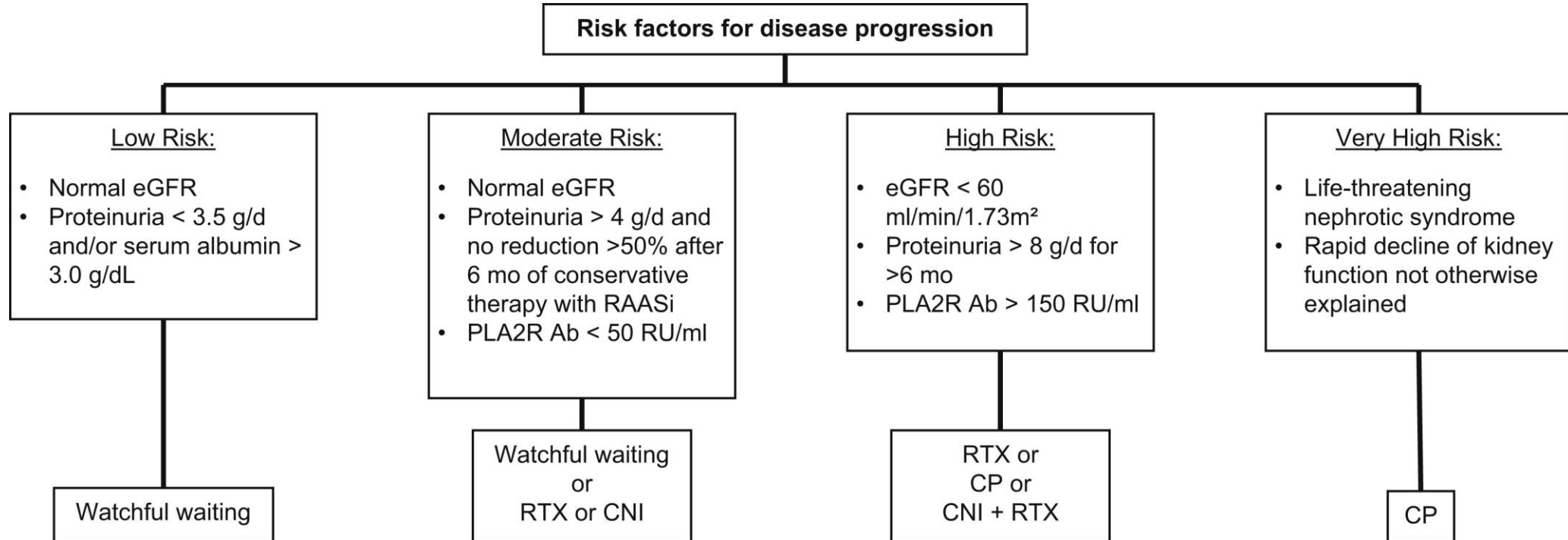


Membranous Nephropathy Presentation

- The majority of patients (~2/3) present with nephrotic syndrome
 - Proteinuria > 3.5 d/day and hypoalbuminemia
- Complications of nephrotic syndrome
 - Edema (can be debilitating)
 - Hyperlipidemia
 - Thrombosis (~ 10%)
 - Infection

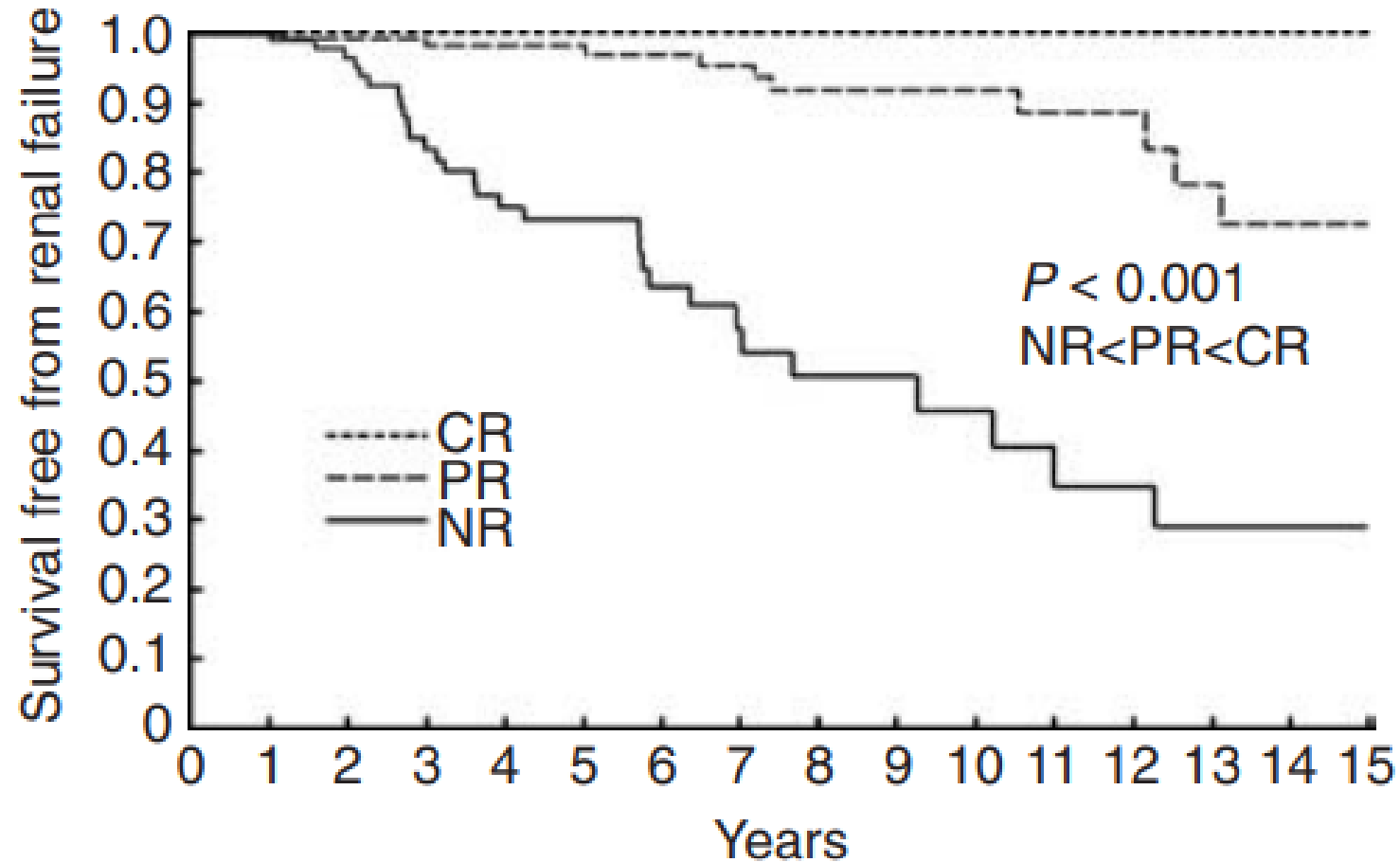


Risk Stratification



Importance of Remission

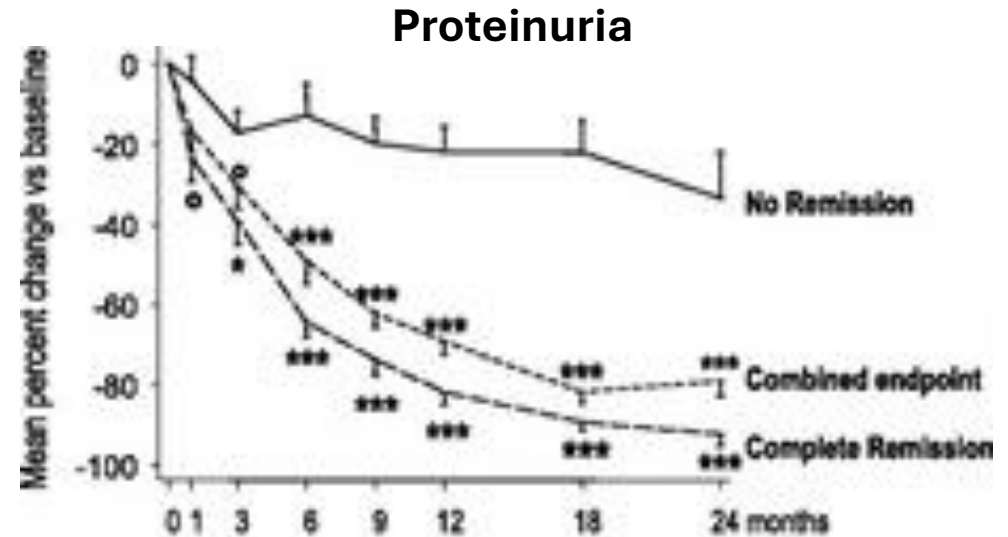
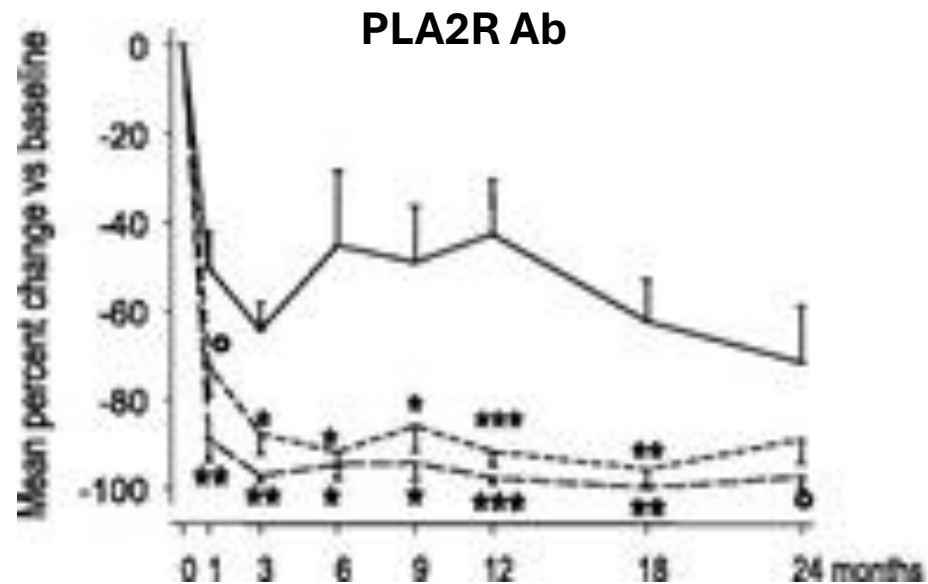
348 nephrotic patients with primary MN



Kidney Int. 2004 Sep;66(3):1199-205.

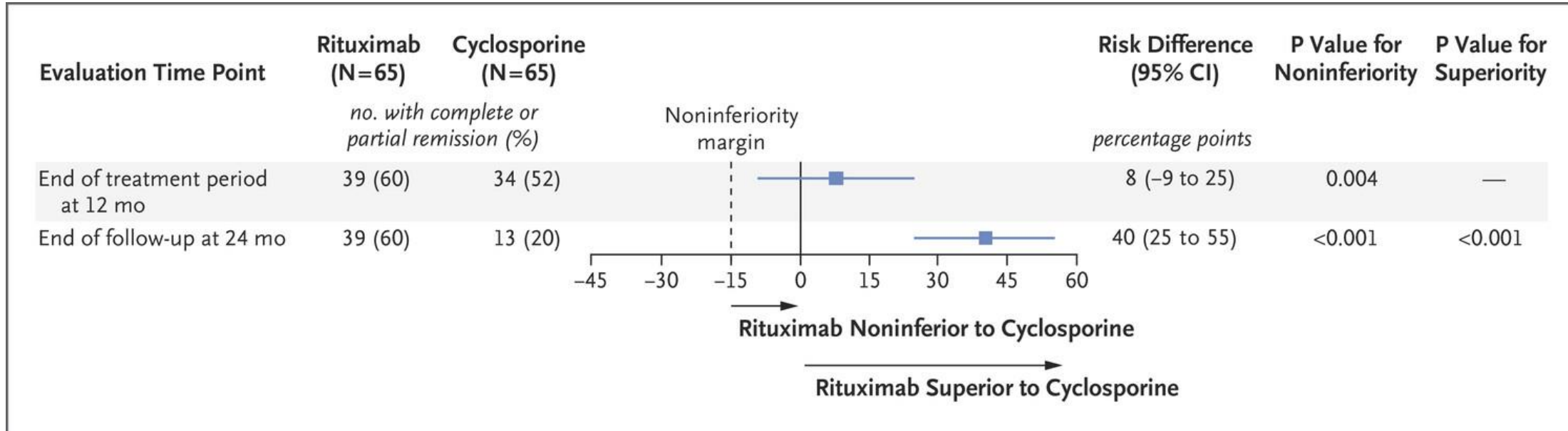
Immunologic Remission

81 nephrotic patients with PLA2R-associated MN treated with rituximab



Rituximab for MN

MENTOR TRIAL: 130 patients with primary MN and proteinuria > 5 g/d



40% of patients receiving rituximab had no remission



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Rituximab for MN

Complete Remission

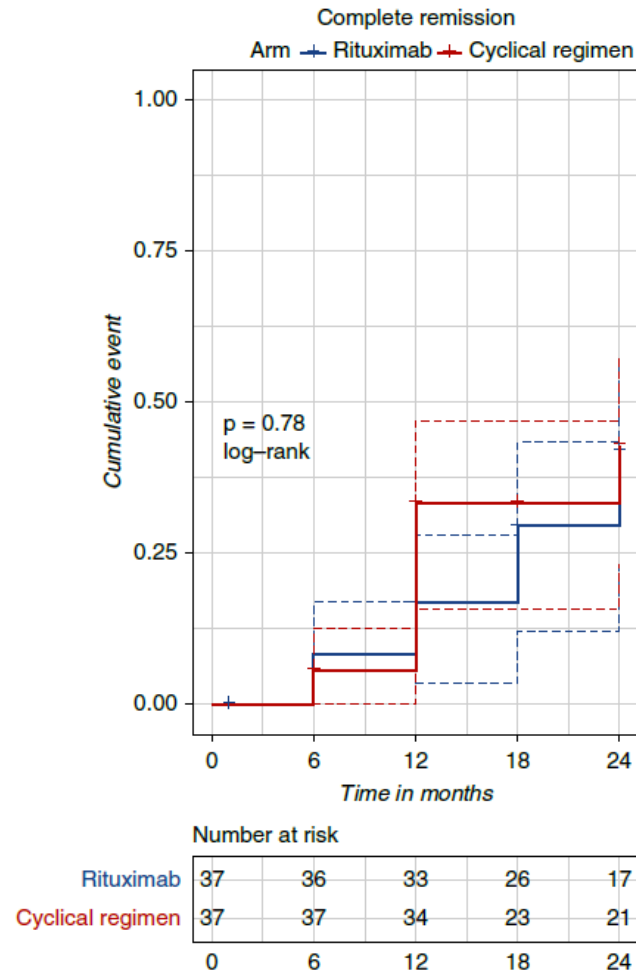
| Time from randomization | Rituximab | | Cyclosporine | | Risk difference (95% CI) |
|-------------------------|-----------|-----------|--------------|---------|--------------------------|
| | n | CR (%) | n | CR (%) | |
| ITT population | | | | | |
| 6 months | 65 | 0 (0.0) | 65 | 1 (1.5) | -1.5 (-4.5 to 1.5) |
| 12 months | 65 | 9 (13.8) | 65 | 3 (4.6) | 9.2 (-0.6 to 19.1) |
| 18 months | 65 | 18 (27.7) | 65 | 1 (1.5) | 26.2 (14.9 to 37.4) |
| 24 months | 65 | 23 (35.4) | 65 | 0 (0.0) | 35.4 (23.8 to 47.0) |

Immunological Remission (ELISA < 40 u/mL)

| Time from randomization | Rituximab | | Cyclosporine | | Risk difference (95% CI) |
|-------------------------|-----------|-----------|--------------|-----------|--------------------------|
| | n | CR/PR (%) | n | CR/PR (%) | |
| Immunological response | | | | | |
| 6 months | 50 | 26 (52.0) | 46 | 13 (28.3) | 23.7 (4.7 to 42.7) |
| 12 months | 50 | 33 (66.0) | 46 | 14 (30.4) | 35.6 (16.9 to 54.3) |
| 18 months | 50 | 32 (64.0) | 46 | 5 (10.9) | 53.1 (37.1 to 69.2) |
| 24 months | 50 | 33 (66.0) | 46 | 6 (13.0) | 53.0 (36.6 to 69.3) |

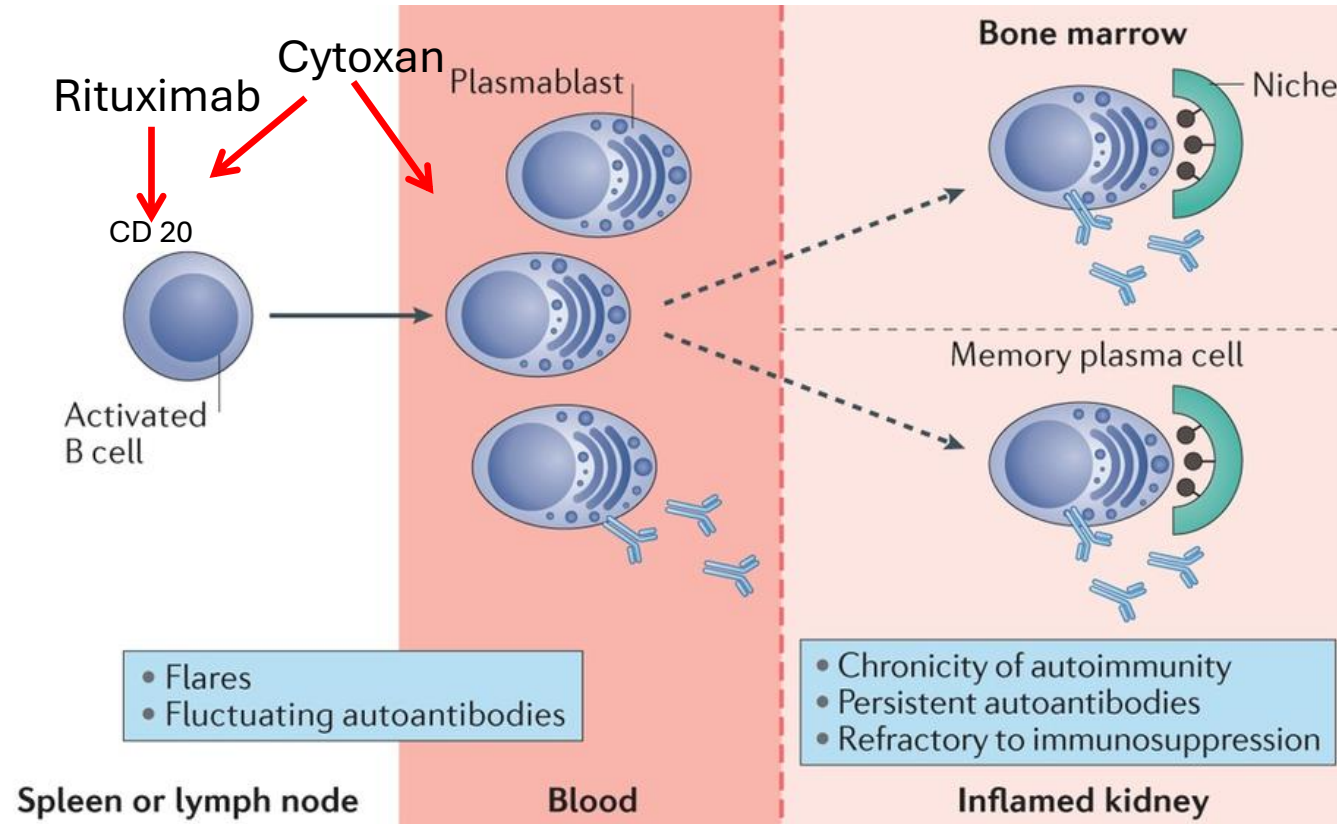


Cyclophosphamide for MN



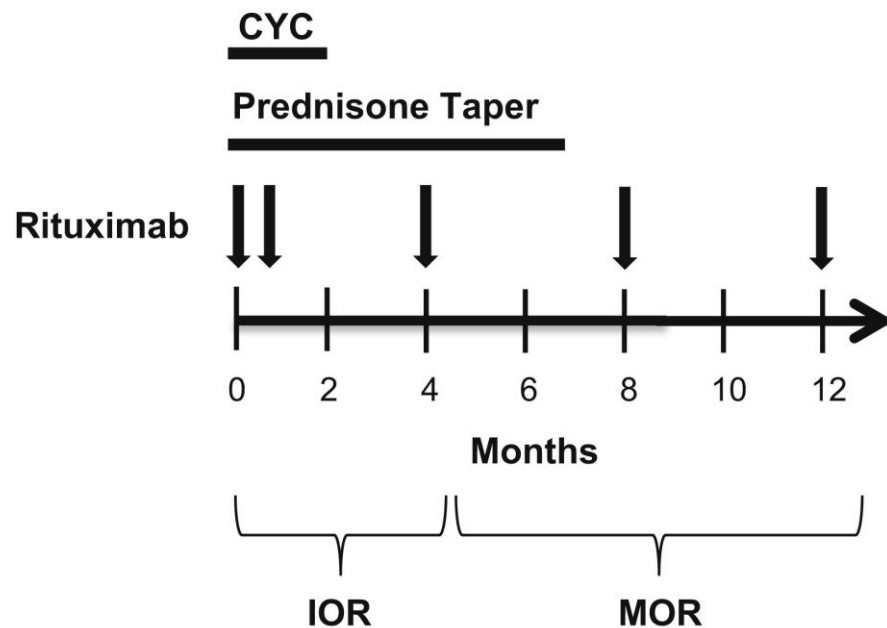
- RI-CYCLO Trial: 74 patients with primary MN and proteinuria > 3.5 g/day randomized to RTX vs CYC/Steroids
- At 12 months: CR in 32% of CYC and 16% in RTX
- At 24 months: CR ~ 40% of both groups
- **Concerns about toxicity and need for intensive monitoring limit cyclophosphamide use**

Combination Therapy for MN



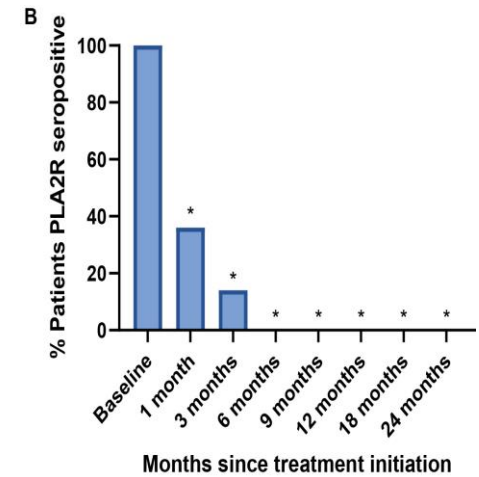
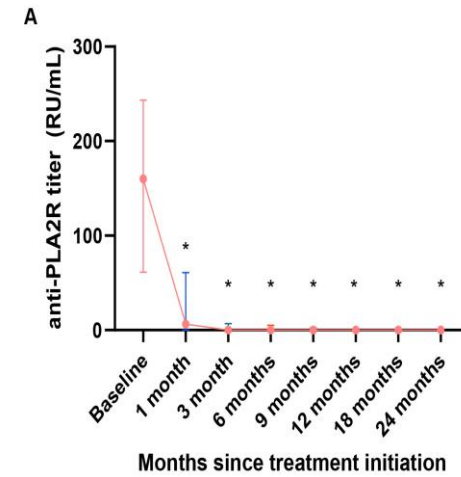
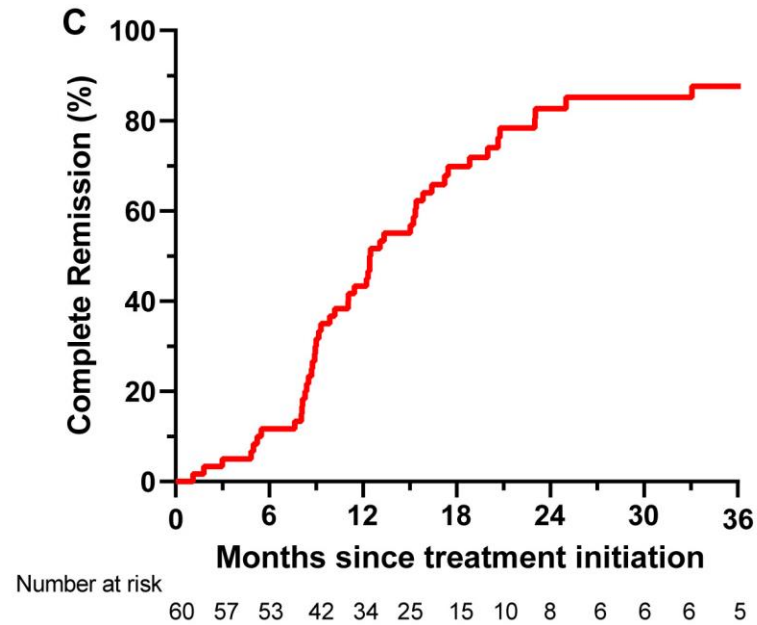
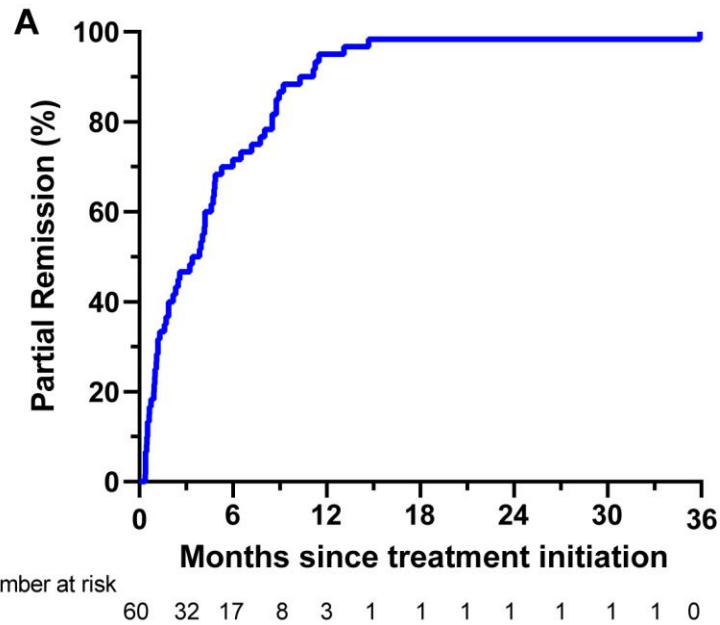
Nature Reviews | Nephrology

Combination Therapy for MN

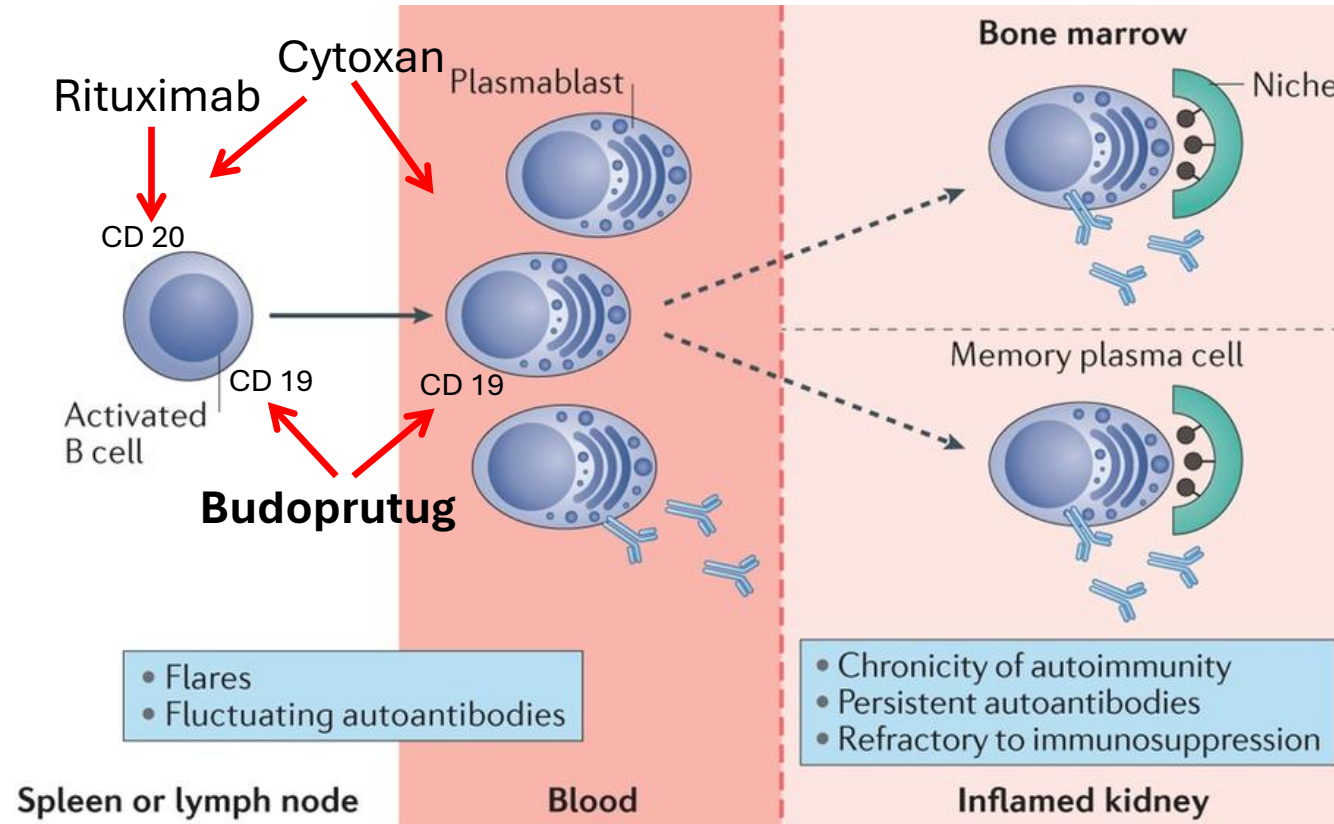


- Rituximab
 - 1 gm x 2
- Prednisone
 - Tapered to 15mg by day 30
 - Then tapered by 2.5 mg/month
- Cytoxan
 - 2.5 mg/kg for 1 week
 - 1.5 mg/kg for 7 weeks
 - **Adjusted for renal fx**

Combination Therapy for MN



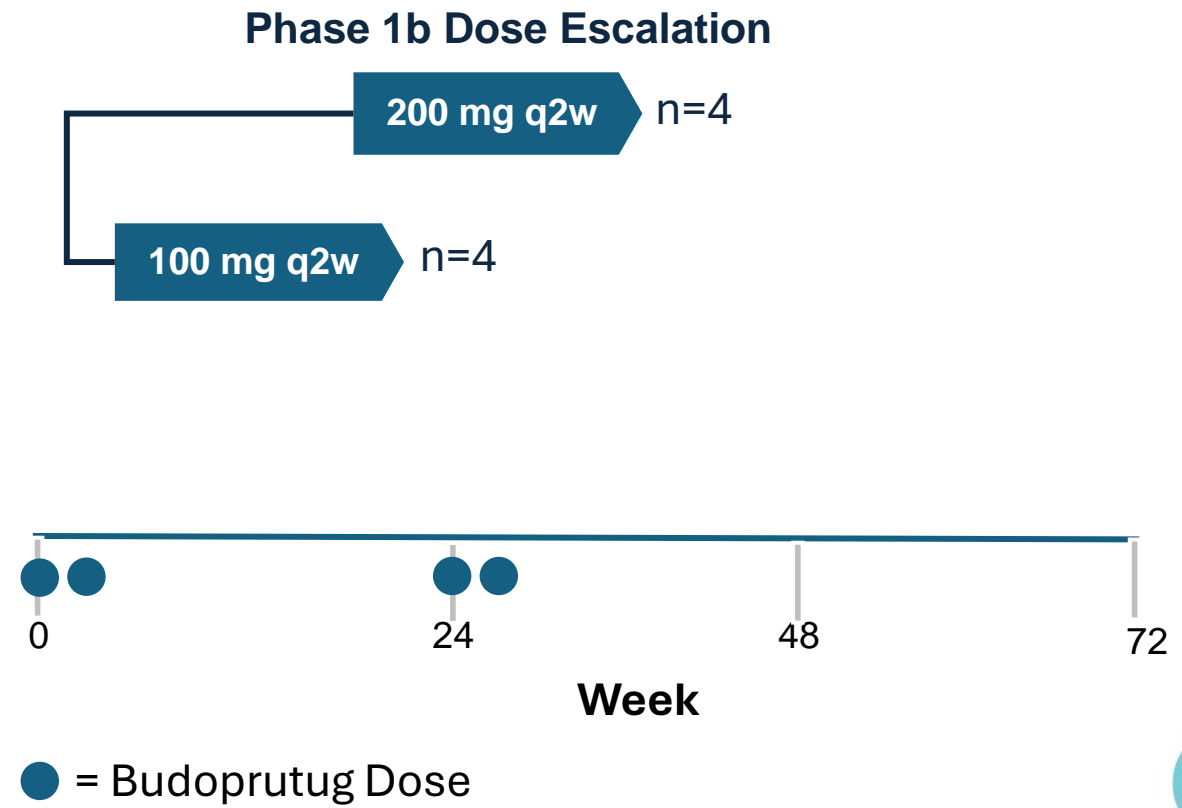
Combination Therapy for MN



Nature Reviews | Nephrology

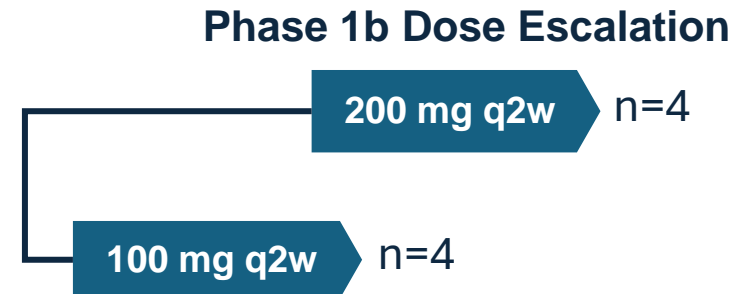
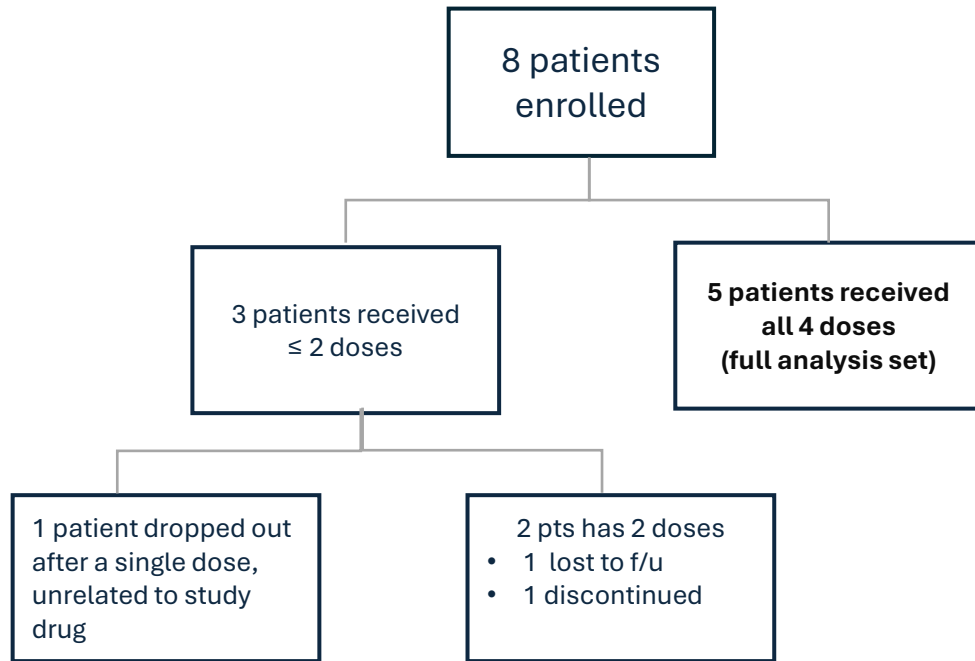
Budoprutug MN Phase 1b Study Design

| | |
|--------------------|--|
| ELIGIBILITY | <ul style="list-style-type: none"> UPCR ≥ 2.0 g/g B-cell count $>LLN$ (80 cells/μL) |
| DESIGN | <ul style="list-style-type: none"> Dose escalation & expansion 18-month follow-up |
| ELIGIBILITY | <ul style="list-style-type: none"> UPCR ≥ 2.0 g/g B-cell count $>LLN$ (80 cells/μL) |
| DESIGN | <ul style="list-style-type: none"> Dose escalation & expansion 18-month follow-up |
| DOSING | 2 doses 14 days apart† <ul style="list-style-type: none"> 100 mg 200 mg |
| ENDPOINTS | <ul style="list-style-type: none"> Safety, Tolerability & PK PD markers (B-cells, PLA2R) Proteinuria response |



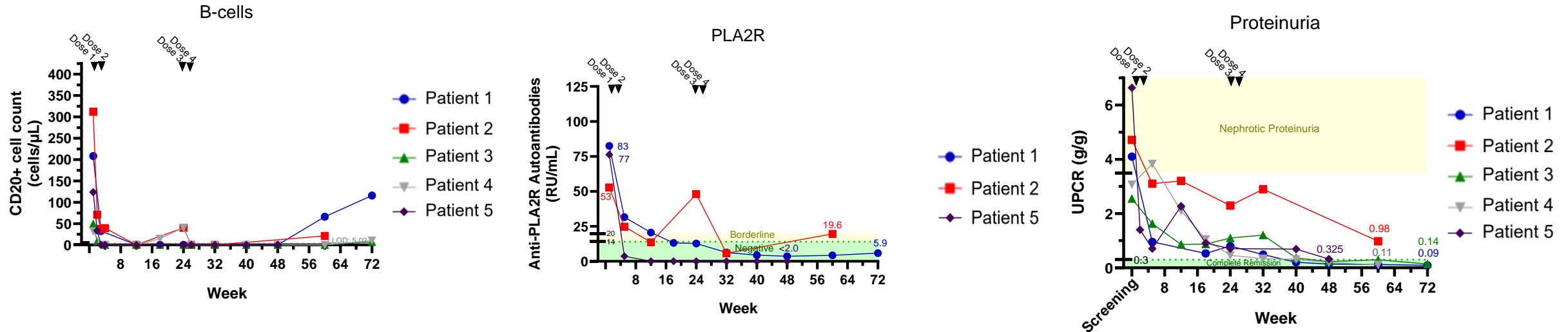
Budoprutug Phase 1b Study

| PARAMETER | BASELINE (MEAN) |
|-----------|--------------------|
| B-cells | 145 cells/ μ L |
| PLA2R | 71 RU/mL |
| UPCR | 4.03 g/g |



Budoprutug Phase 1b Study

Data for MN subjects (n = 5) who have completed ≥ 48 -weeks



Complete remission achieved in 60% (3/5) of patients at Week 48

- Partial remission ($>50\%$ reduction in UPCR + UPCR <3.5 g/g) achieved in all (5/5) subjects
- Complete B-cell depletion (CD20⁺ count <5 cells/ μ L) achieved in all (5/5) subjects
- Anti-PLA2R Ab negativity (<14 RU/mL) achieved in all (3/3) evaluable subjects
- 2 subjects on study that have not entered complete remission have achieved PLA2R negativity (serological remission)
- *Patient 1 remains in CR 2 yrs after last infusion*



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Budoprutug Safety

Budoprutug was generally well tolerated at doses of up to 200mg

- 8 Patients received at least one injection of budoprutug and are included in the safety analysis population
 - There were no deaths on study
 - There were 3 SAEs, none of which were 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, 1 was bacterial pneumonia



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Budoprutug in MN

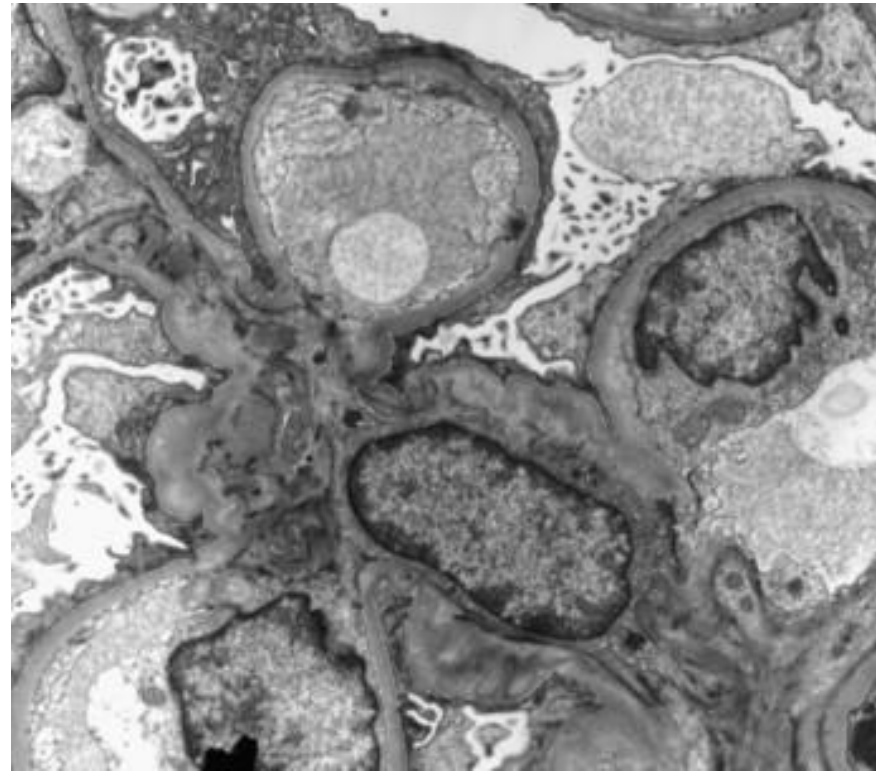
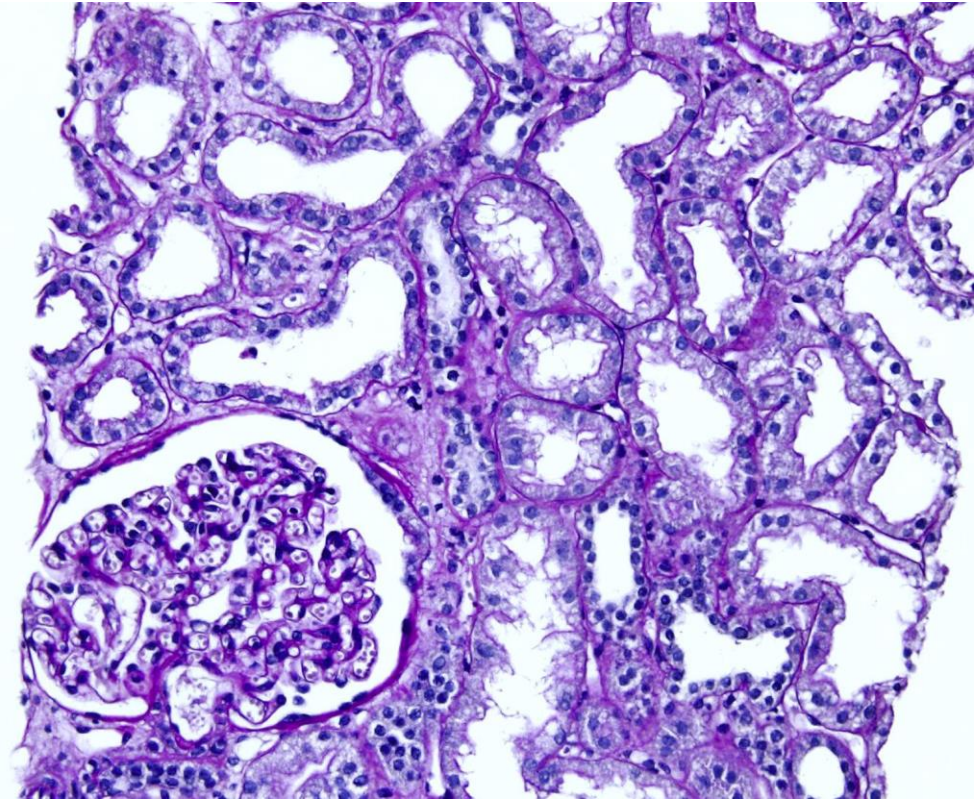
- Well tolerated
- Demonstrated clinical and immunological remission rates superior to rituximab based on historical data



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Minimal Change Disease



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MCD Overview

| | |
|---------------------------------|---|
| Pathophysiology | <ul style="list-style-type: none">• Unknown etiology, though evidence suggests a circulating autoantibody (AAb)• Diffuse foot effacement on biopsy |
| Symptoms & Diagnosis | <ul style="list-style-type: none">• Sudden onset of nephrotic syndrome• Edema, often profound |
| Epidemiology | <ul style="list-style-type: none">• Incidence of 1/150k• 20k addressable patients in US |
| Natural History | <ul style="list-style-type: none">• Uncontrolled disease results in complications of nephrotic syndrome & eventual kidney failure• Patients that respond to therapy can achieve long-term remissions |
| Standard of Care | <ul style="list-style-type: none">• Steroids used to induce, maintain remission• No labeled, FDA-approved therapy |

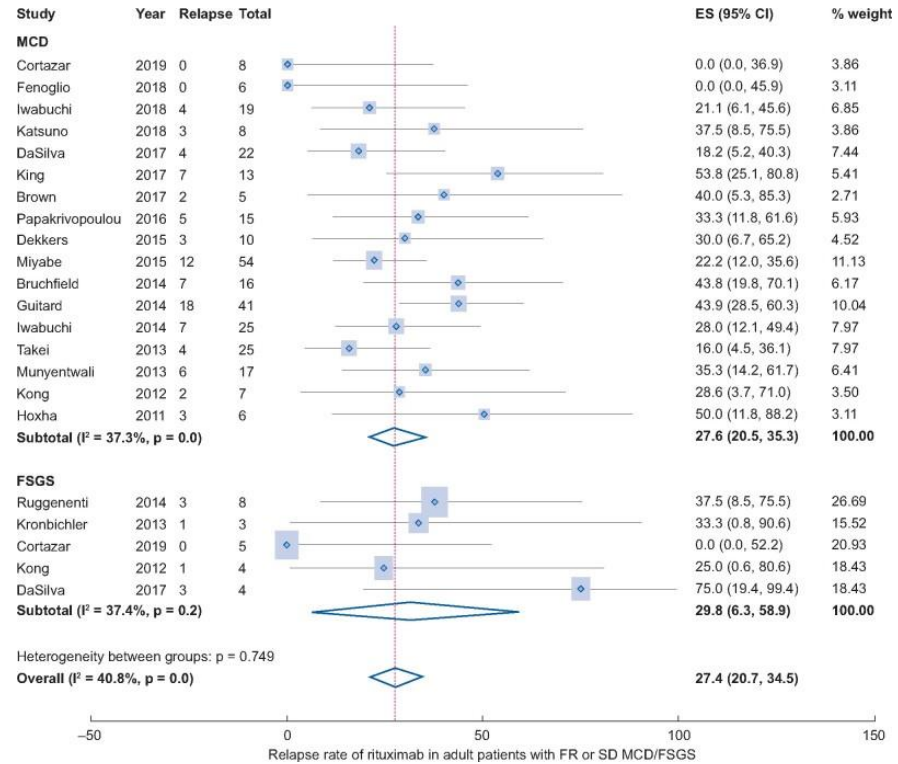
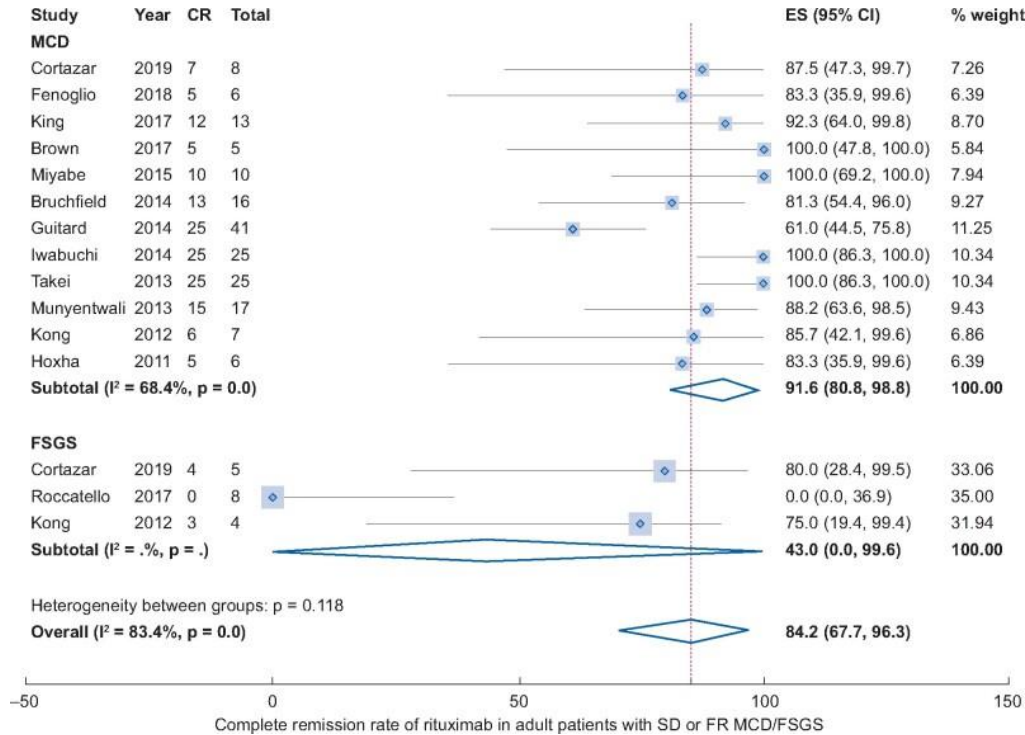


Treatment Challenges in MCD

- A significant number of patients have:
 - Steroid dependent (unable to taper steroids without a relapse)
 - Frequently relapsing disease (4 relapses/yr)
- Treatment options include
 - CNIs (nephrotoxicity/high relapse rate)
 - Cyclophosphamide (High toxicity)
 - **Rituximab**

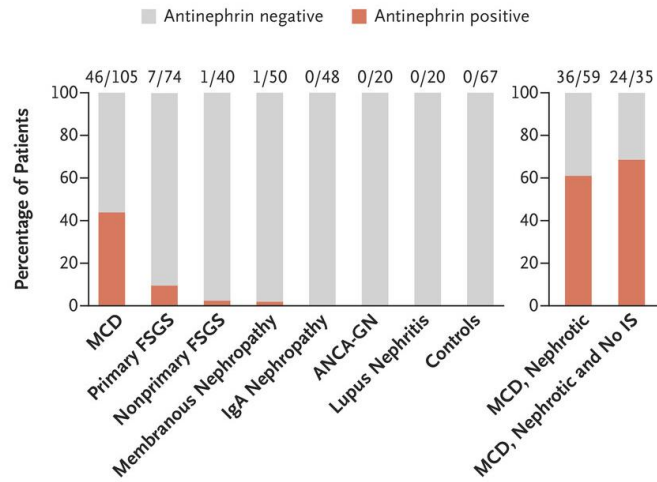


Rituximab for complicated MCD

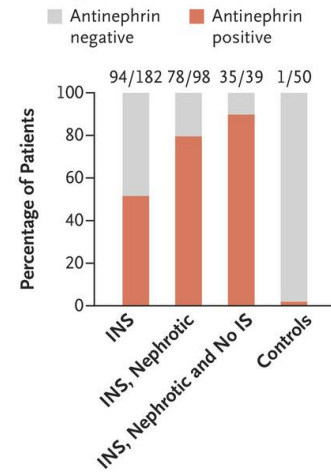


Antinephrin antibodies in MCD

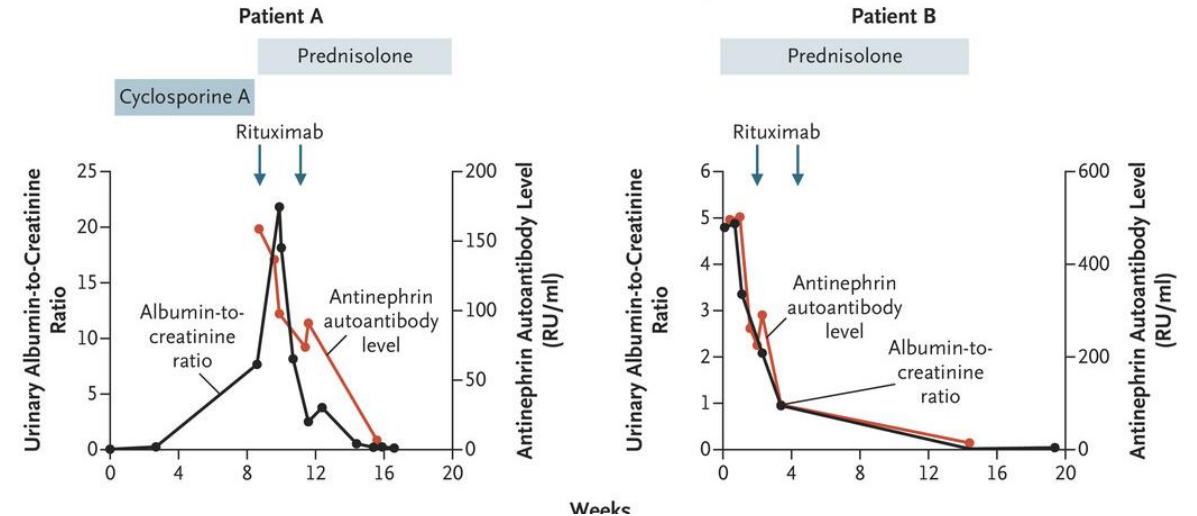
B Antinephrin Autoantibody Prevalence among Adults



C Antinephrin Autoantibody Prevalence among Children



D Rituximab Treatment in Patients with Antinephrin-Associated Podocytopathies



Budoprutug for complicated MCD

- Phase 1b Study
- Enrolled a 57-year-old with frequently relapsing MCD
 - Relapsed on tacrolimus
 - Remission after steroid course; relapsed within 2 months and steroids reinitiated
- Received budoprutug: 2 induction doses and 2 maintenance doses
 - B cells effectively depleted
 - Steroids discontinued; remains in remission 18 months after last infusion
 - No safety signals

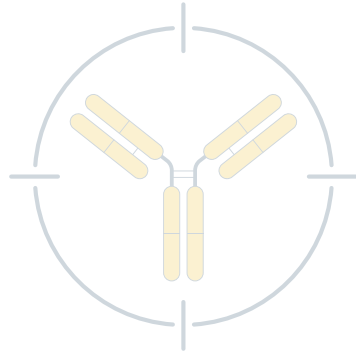


Complex Systemic Disease Opportunity

Jan Hillson, MD | Senior Clinical Advisor

Corporate Strategy & Vision

Climb is well-positioned to advance budoprutug across three distinct opportunity sets



Primarily IgG4-Mediated

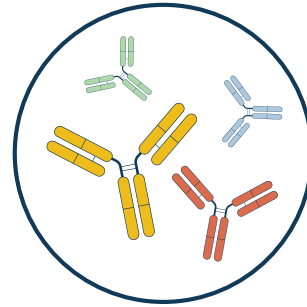
Clear pathophysiology supporting targeting of CD19-expressing B-cells

Lead indication

Membranous Nephropathy

Opportunity to Differentiate

Potential for “immune reset”, improved efficacy



Complex Systemic

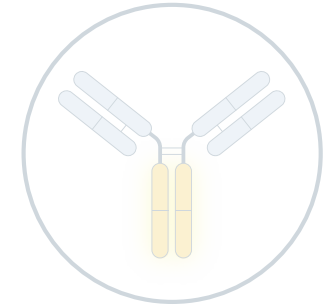
Multi-organ, systemic diseases with heterogenous patient populations

Lead indication

Systemic Lupus Erythematosus

Opportunity to Differentiate

Improve efficacy while balancing safety, tolerability, and convenience



Primarily single organ IgG1 - 3

Orphan diseases with compelling clinical proof-of-concept using B-cell depletion

Lead indication

Immune Thrombocytopenia

Opportunity to Differentiate

Demonstrate efficacy in relapsing and/or refractory patients

Systemic Lupus Erythematosus (SLE): Indication Overview

| | |
|---------------------------------|--|
| Pathophysiology | <ul style="list-style-type: none">• SLE comprises a group of disorders characterized by the generation and persistence of autoreactive lymphocytes and autoantibodies directly interfere with critical functions, target cells for destruction, and damage tissues through immune complex depositions |
| Symptoms & Diagnosis | <ul style="list-style-type: none">• Diagnosis is clinical, based on serology and organ system involvement without other cause• Symptoms and severity vary widely across patients. Nephritis is the most common organ system threat; fatigue and cognitive dysfunction are the most common disabling manifestations |
| Epidemiology | <ul style="list-style-type: none">• US burden is ~240,000 active patients; ~80,000 with lupus nephritis• Global prevalence is ~1-2 per 100,000 adults, with 9:1 female predominance |
| Standard of Care | <ul style="list-style-type: none">• Corticosteroids to rapidly control inflammation• Antimalarials for rash and to reduce flares• Small molecule immune suppressants to reduce corticosteroid use• Belimumab (B cell activating factor blockade), Anifrolumab (type 1 interferon receptor blockade) for refractory disease• Rituximab used off label |
| Unmet Need | <ul style="list-style-type: none">• 10% – 20% are refractory to current therapies; much larger numbers are dependent on corticosteroids• Relapses, especially of nephritis, vasculitis, thrombosis, lead to cumulative damage and organ failure• Fatigue and cognitive dysfunction respond poorly, impairing participation and quality of life• Treatment-associated burden of cardiovascular mortality, infection, and neoplasm risk |

Targeting B-Cells Has Shown Promise in SLE

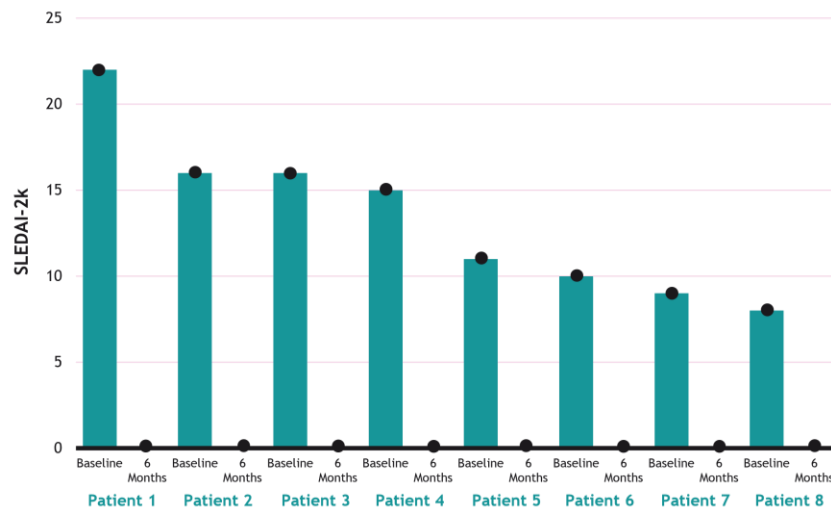
| DRUG | CO. | TARGET | ROUTE | STAGE | SLE CLINICAL DATA |
|--------------|-------|--------|--------|---|---|
| Rituximab | Roche | CD20 | IV, SC | Phase 3 SLE and LN (failed) | Phase 3 (vs. placebo, n=257) <u>Week 52 SRI-4</u> 27.2% (vs. 22.7%) |
| Obinutuzumab | Roche | CD20 | IV | Phase 3 LN (completed; results pending) | Phase 2 (vs placebo, n=125) <u>Week 52 CRR</u> 35% (vs 23%) <u>Week 104 CRR</u> 41% (vs 23%) |
| Belimumab | GSK | BAFF | SC | SLE and LN (marketed) | Phase 3 (vs. placebo, n=836) <u>Week 52 SRI-4</u> 61% (vs. 48%) |

Patients with poor response to depletion of CD20+ B cells are characterized by any among:

- Inadequate CD20+ B cell depletion
- Persistence of CD19+ self-reactive B cell subsets
- Continued production of pathogenic autoantibodies by plasma cells
- Rapid recovery of pathogenic B cell subsets

Targeting B-Cells Has Shown Promise in SLE

CD19 CAR-T potentially 'curative' at 6 months

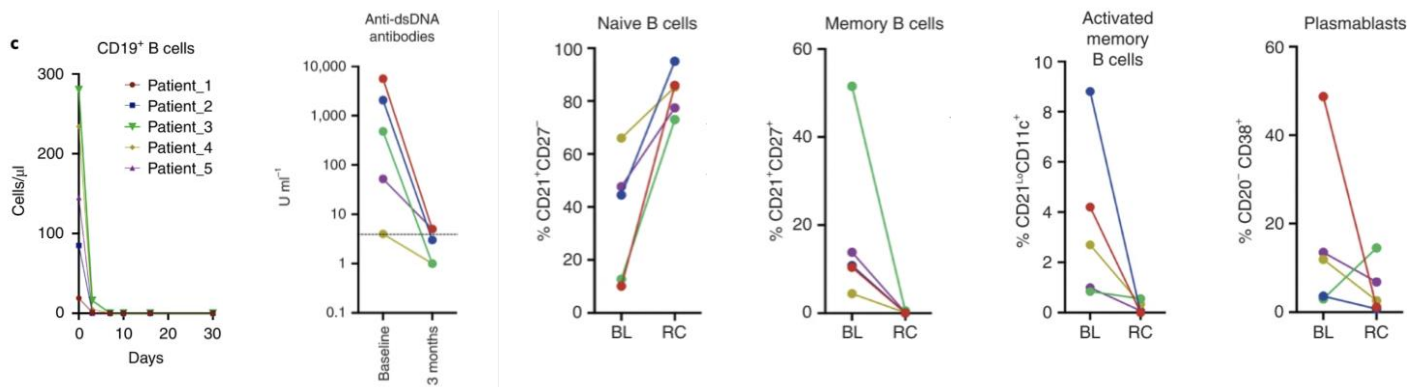


SLE patient data from Müller 2024, Mackensen 2022, SLEDAI-2K = Systemic Lupus Erythematosus Disease Activity Index 2000

Depletion of CD19+ B cells with CD19+ CAR-T cells addresses some of the shortcomings of existing therapy. Limitations include:

- Challenging to make autologous cells from highly pre-treated patients
- Toxicity associated with required conditioning therapy
- Risk of cytokine release syndrome and immune effector cell associated neurotoxicity syndrome
- Access limited by complexity and cost

Response is associated with rapid depletion of circulating B cell and autoantibodies, followed by recovery of relatively benign B cell subsets



SLE Trial Design & Objectives*

Planned open-label, dose escalation with augmented B cell and antibody analyses

POPULATION

- Diagnosis of SLE, with active disease based on SLEDAI
- Seropositive, with elevated ANA, anti-dsDNA, ENA or APL
- <20 mg prednisone by Day -28; stable limited background

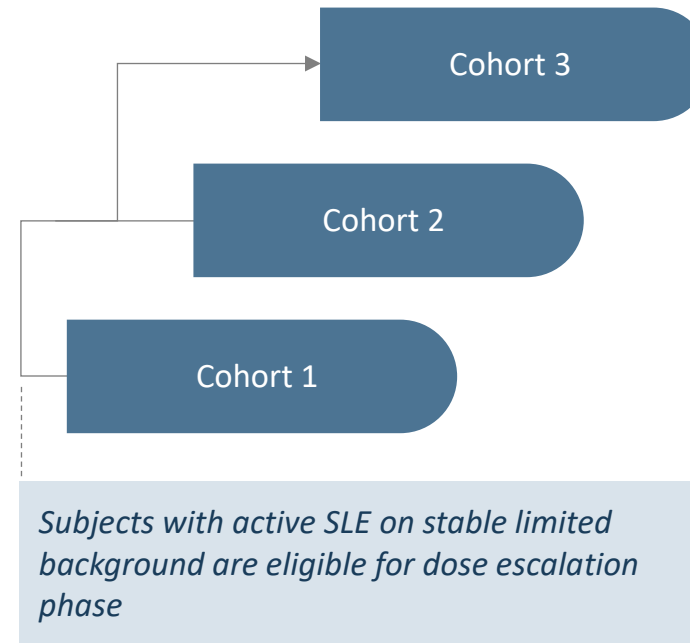
PRIMARY OBJECTIVE

- To evaluate the safety and tolerability of budoprutug in subjects with SLE

KEY SECONDARY/EXPLORATORY OBJECTIVES

- To evaluate the effects of budoprutug on B-cell depletion (prioritized pharmacodynamic [PD] response), autoantibody levels, and protective antibody levels
- To evaluate the PK and PK/PD (dose relationship) profile in patients with SLE
- To evaluate preliminary signs of clinical activity in patients with SLE
- To determine the kinetics of re-population of B-cell subsets and antibodies after depletion

Dose Escalation



Potential Dose Expansion

Dose regimen selected based on Dose Escalation

Subjects with active SLE despite adequate trial of two prior therapies
Follow until B cells return toward baseline

Corporate Outlook

Significant unmet needs across lead indications, >2.5M US patients with immune-mediated disease

Primarily IgG4-Mediated

MG (MuSK)
~7k

MN
~70k

CIDP
~20k

IgG4-RD
~20k

PV
~15k

Complex Systemic

RA
~1300k

Sjogren's
~340k

SLE
~240k

SSc
~85k

Primarily single organ IgG1 - 3

MS
~730k

ANCA-AAV
~140k

MG (AChR)
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ITP
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MG MuSK= Myasthenia Gravis muscle-specific tyrosine kinase; SLE = Systemic Lupus Erythematosus, MN = Membranous Nephropathy, ITP = Immune Thrombocytopenia NMOSD = Neuromyelitis optica spectrum disorder, BP = Bullous pemphigoid, ANCA-AAV = antineutrophil cytoplasmic antibody-associated vasculitis, SSc = Systemic sclerosis; CIDP = Chronic inflammatory demyelinating polyradiculoneuropathy, IgG4-RD = IgG4 related disease, RA = Rheumatoid arthritis, MS = Multiple sclerosis

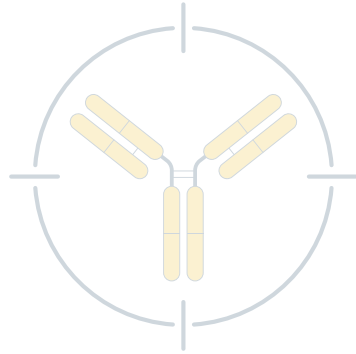
Prevalence references: SLE (Izmirly 2021), ITP (internal research), MN (internal research), MG (Ye 2024), SSc (Fan 2020), ANCA-AAV (Berti 2017), CIDP (Laughlin 2009), BP (Wertenteil 2018), NMOSD (Briggs 2024), IgG4-RD (Wallace 2023), RA (Hunter 2017), Sjogren's (Maciel 2017), MS (Wallin 2019)

Primarily Single Organ IgG1 – 3 Disease Opportunity

Nishi Rampal, MD | SVP Clinical Development

Corporate Strategy & Vision

Climb is well-positioned to advance budoprutug across three distinct opportunity sets



Primarily IgG4-Mediated

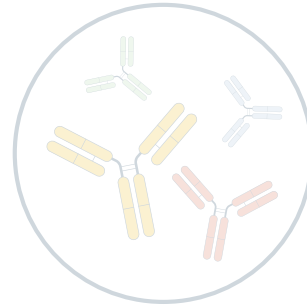
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Opportunity to Differentiate

Potential for “immune reset”, improved efficacy



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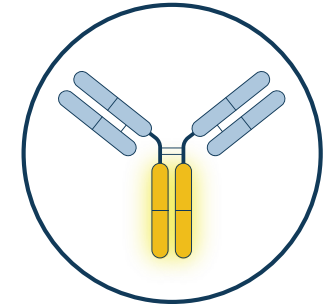
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Systemic Lupus Erythematosus

Opportunity to Differentiate

Improve efficacy while balancing safety, tolerability, and convenience



Primarily single organ IgG1 - 3

Orphan diseases with compelling clinical proof-of-concept using B-cell depletion

Lead indication

Immune Thrombocytopenia

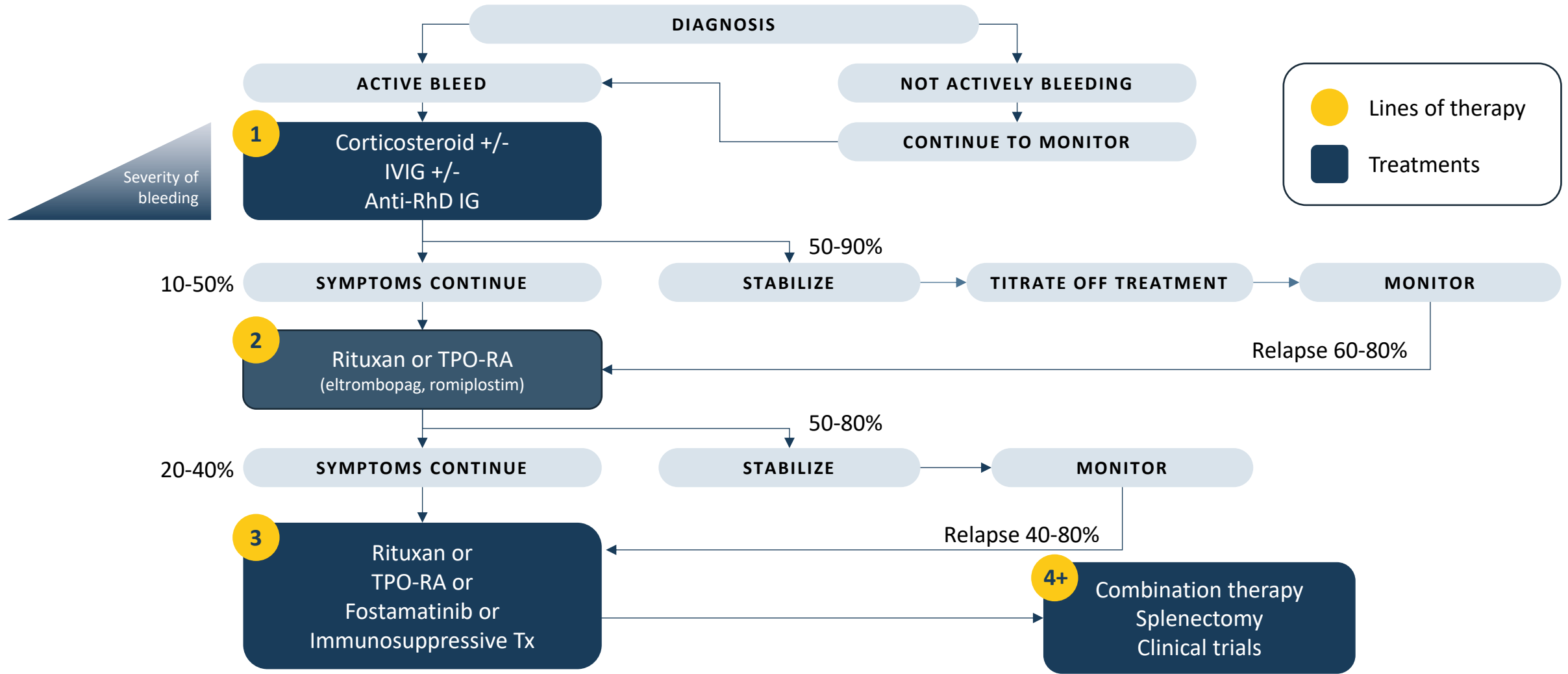
Opportunity to Differentiate

Demonstrate efficacy in relapsing and/or refractory patients

Immune thrombocytopenia (ITP): Indication Overview

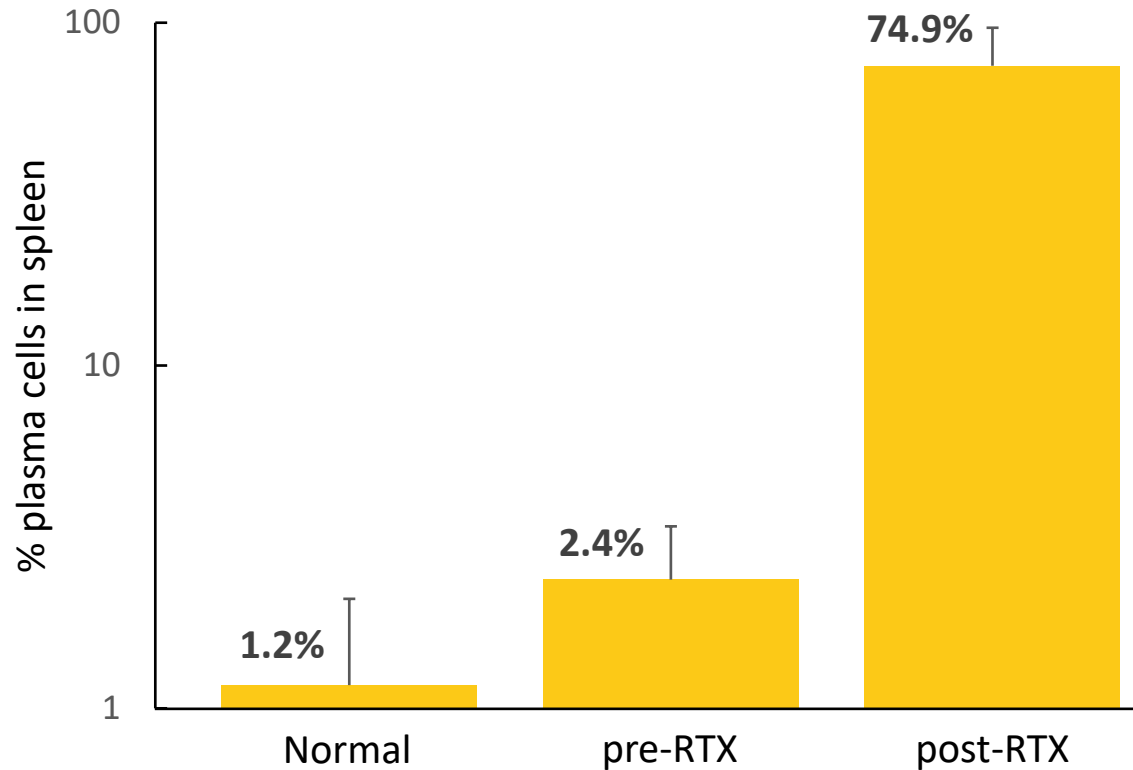
| | |
|---------------------------------|---|
| Pathophysiology | <ul style="list-style-type: none">• ITP is an autoimmune disease characterized by low platelets resulting in bruising and hemorrhage• Antiplatelet autoantibodies lead to accelerated removal of platelets by macrophages with bone marrow compensation |
| Symptoms & Diagnosis | <ul style="list-style-type: none">• Symptoms: bruising (petechiae and purpura), bleeding episodes, and fatigue• Diagnosis: Low platelet count, supported by additional blood tests i.e., CBC and blood smear, antiplatelet antibody test, bone marrow aspiration if needed |
| Epidemiology | <ul style="list-style-type: none">• The estimated global prevalence of ITP is around 200,000 patients worldwide• In the US, there are 81,000 adults with chronic ITP with >24,000 refractory to 2nd line treatment |
| Natural History | <ul style="list-style-type: none">• Most children have spontaneous remission within a few weeks or months• While adults often stabilize on 1st line therapy, the majority eventually relapse or become refractory, necessitating treatment with 2nd and at times 3rd line therapies, splenectomy in hard-to-treat situations can be considered |

Current management guidelines



ITP patients likely fail rituximab due to the presence of CD19+CD20- B-cells

CD19⁺/CD20⁻ plasma cells expand within B-cell niches post anti-CD20 treatment

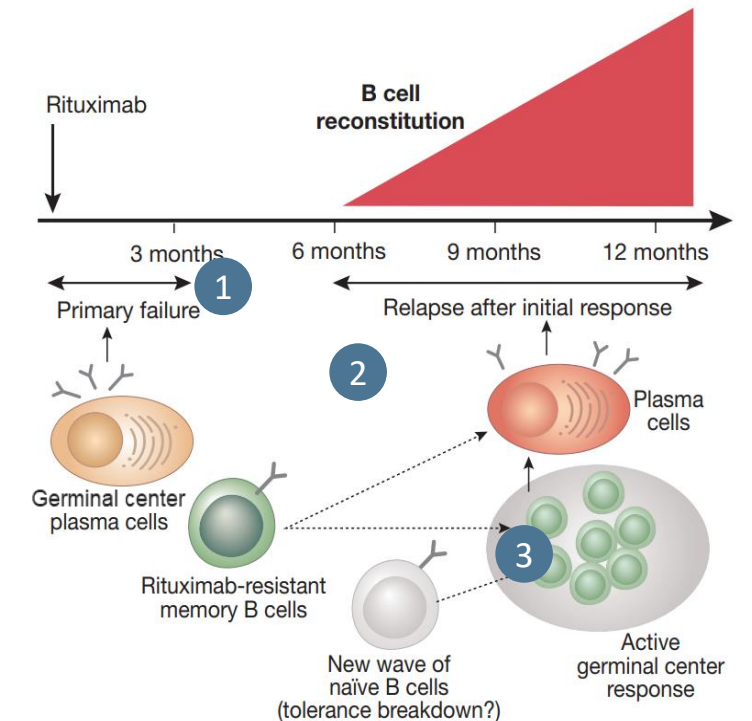


Primary failure

- 1 Pre-existing CD20⁻ PCs

Relapse after initial response

- 2 Pre-existing CD20⁻ B-cells
- 3 *De novo* CD20⁻ B-cells



ITP Phase 2 Trial Design & Objectives*

Planned single arm, open-label study focused on platelet response and B-cell depletion

POPULATION

- Insufficient response to 1 or more prior therapies
- Platelet count $<30,000/\mu\text{L}$
- B cells $> 40 /\mu\text{L}$

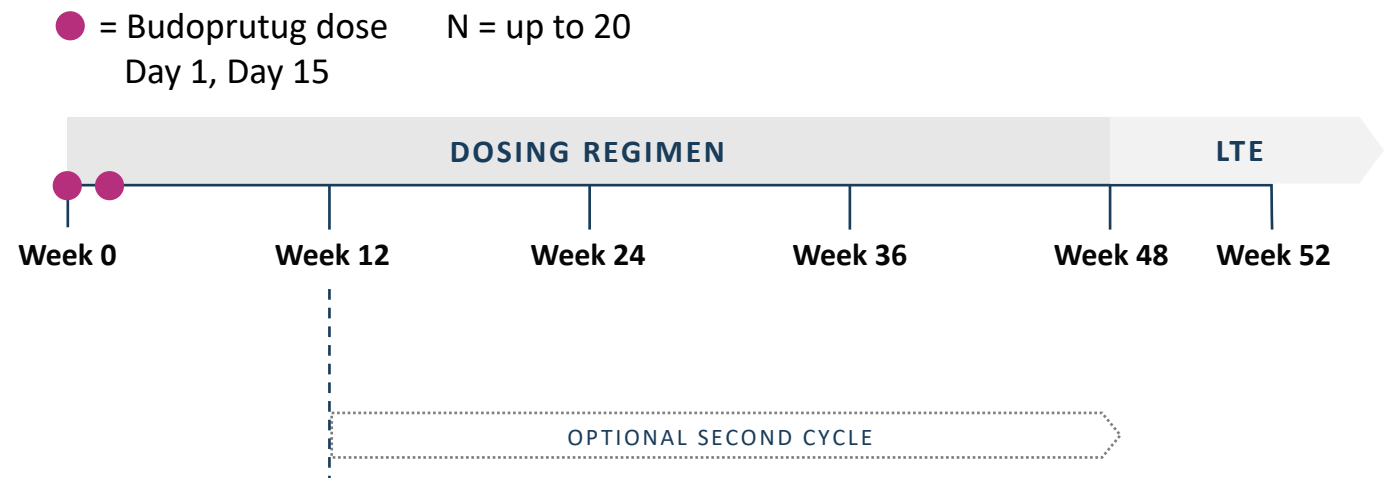
PRIMARY OBJECTIVE

- To evaluate the safety and tolerability of budoprutug in subjects with ITP
- To evaluate the efficacy of budoprutug on platelet counts

KEY SECONDARY/EXPLORATORY OBJECTIVES

- To evaluate subject reported outcomes/quality of life (QoL) measures
- To evaluate PK/PD (dose relationship) profile in subjects with ITP

Potential for treat to target approach



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Corporate Outlook

Brett Kaplan, MD | COO

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Multiple Clinical and Regulatory Milestones Over Coming 12 Months

\$218M*

67.2M*

RUNWAY THROUGH 2027

SHARES OUTSTANDING

Anticipated Milestones

Timing

Initiation of SLE global clinical trial with first patient in[†]

H1 2025

Initiation of ITP global clinical trial with first patient in[†]

H1 2025

Additional non-clinical data from subcutaneous program

H1 2025

Advance PMN program to late phase development

2025

Initiate clinical development of subcutaneous program

2025

Evaluate additional programs

2025

Building A Leading Immune-Mediated Disease Focused Company



Immune-Mediated Disease Focus

Focused solely on immune-mediated disease with budoprutug, a potentially best-in-class anti-CD19 antibody, our cornerstone asset



Broad Potential

Budoprutug in development for SLE, ITP and MN with the prospect of expanding into additional indications as well as a potential subcutaneous formulation



Well Resourced

Funded through 2027 enabling delivery of key value inflection points through the initiation of multiple clinical programs and a subcutaneous formulation clinical trial



Experienced Team

Track record of execution and operational results

Questions & Answers