FEBRUARY 2026

Rallybio

On a mission to deliver groundbreaking therapies that transform the lives of patients with devastating rare diseases

Forward-Looking Statements

This presentation contains forward-looking statements that are based on management's beliefs and assumptions and on information currently available to management. All statements other than statements of historical facts contained in this presentation are forward-looking statements. In some cases, you can identify forward-looking statements by terms such as "may," "will," "should," "expect," "plan," "anticipate," "could," "intend," "target,", "seek," "goal," "project," "contemplate," "believe," "estimate," "predict," "potential" or "continue" or the negative of these terms or other similar expressions, although not all forward-looking statements contain these words. Forward-looking statements include, but are not limited to, statements concerning: the initiation, timing, progress, results, and cost of our research and development programs, and our future clinical studies, including statements regarding our clinical development plan for RLYB116; the scientific and medical attributes of RLYB116 that we believe differentiate it from candidates developed by third parties, and the potential patient demand for such product candidates; the potential clinical effects and benefits of RLYB116; whether RLYB116 will be suitable to treat a broad range of complement mediated diseases; whether RLYB116 efficacy and safety will be similar to approved C5 inhibitors; timing of potential milestones for our programs; our ability to compete with companies currently marketing or engaged in the development of treatments for diseases that RLYB116 is designed to target; our estimates of the commercial opportunity for RLYB116; and our ability to serve those markets. The forward-looking statements in this presentation are only predictions and are based on management's current expectations and projections about future events and financial trends that we believe may affect our business, financial condition and results of operations. These forward-looking statements speak only as of the date of this presentation and are subject to a number of known and unknown risks, uncertainties and assumptions, including but not limited to, our ability to successfully initiate and conduct our planned clinical studies and complete such clinical studies and obtain results on our expected timelines, or at all, whether our cash resources will be sufficient to fund our operating expenses and capital expenditure requirements, competition from other biotechnology and pharmaceutical companies, and those risks and uncertainties described in our filings with the Securities and Exchange Commission (the "SEC"), including under the heading "Risk Factors" in our Quarterly Report on Form 10-Q for the period ending September 30, 2025, and any subsequent filings with the SEC.

Because forward-looking statements are inherently subject to risks and uncertainties, some of which cannot be predicted or quantified and some of which are beyond our control, you should not rely on these forward-looking statements as guarantees of future events. The events and circumstances reflected in our forward-looking statements may not be achieved or occur and actual future results, levels of activity, performance and events and circumstances could differ materially from those projected in the forward-looking statements. Moreover, we operate in an evolving environment. New risks and uncertainties may emerge from time to time, and it is not possible for management to predict all risks and uncertainties. Except as required by applicable law, we are not obligated to publicly update or revise any forward-looking statements contained herein, whether as a result of any new information, future events, changed circumstances or otherwise.

The team with broad experience **poised to deliver a next-generation complement therapeutic to the market**

Select Rare Disease Drugs Developed by Members of the Rallybio Team at Prior Companies:

SOLIRIS® ULTOMIRIS

Sharon Arrol, PhD
Bioanalytical

Michael Bombara
Clinical Operations

Amanda Hayward, PhD
Business Development and Commercial Strategy

Jon Lieber
CFO

Mark Ma, MD, MSc
Translational Sciences

Steve Ryder, MD
CMO

Doug Sheridan, PhD
R&D

Mike Sullivan, PMP
Portfolio & Project Management

Steve Uden, MD
CEO

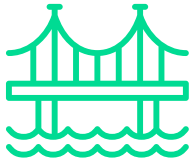
SELECT EXPERIENCE INCLUDES:

Pfizer ALEXION astellas

Driving value in Rallybio by doing what we know best

A LEADING MOLECULE

Robust clinical data validates **RLYB116** as potential best in class therapeutic to treat complement mediated diseases



CLINICALLY VALIDATED TARGETS

Initial focus on **immune PTR** (ability to develop and launch independently) and **APS** (attractive path to Phase 2 PoC) representing $5+ billion revenue opportunity; gateway to additional indications with partner

HIGHLY EFFECTIVE TEAM

Deep experience in drug development and management of emerging growth biotechnology companies; >11 drug approvals including two market leading complement inhibitors

EFFICIENT CLINICAL DEVELOPMENT

Immune PTR **PoC data anticipated within one year** from study start and APS PoC data anticipated within two years of study start; possibility of interim reads

MULTIBILLION-DOLLAR OPPORTUNITY

Pipeline in a product with multibillion-dollar revenue potential; strong IP supports durable differentiation and long-term value creation

OPPORTUNITY:
TWO PH 3 READY PROGRAMS

Immune PTR Phase 2 PoC funded with existing cash; additional funding enables PoC in APS and Phase 3 preparatory activities

Potential for two proof-of-concept readouts in multibillion $ markets

RLYB116:
a potential best in class complement therapeutic

C5 is a proven therapeutic target with the potential to address a broad range of complement-mediated diseases

RLYB116 is a small protein therapeutic that binds both C5 and albumin that gives antibody-like activity at a fraction of the size (12KDa)

CHARACTERISTICS

- Complete, rapid and sustained inhibition of terminal complement
- Patient friendly, once weekly, SQ autoinjector
- No loading dose
- No need for at-home refrigeration
- Efficient manufacturing results in lower COGS

DEVELOPMENT PARADIGM

- Focus on high unmet need
- Fast, small PoC with high probability of success
- Rapid path to potential approval
- Pipeline in a product

Phase 1 data demonstrated rapid, complete and sustained complement inhibition at the 300mg dose

Significant unmet need in immune PTR and APS with a $5B+ peak commercial opportunity (major markets)

A true pipeline in a product with potential to capture significant market share and expand the $10B+ global complement inhibitor space

PTR= platelet transfusion refractoriness; APS = Antiphospholipid Syndrome

RLYB116 is a true pipeline in a product opportunity; complement-mediated diseases are greatly underserved

1. Few complement-mediated diseases have effective, approved treatments

2. **IV infusion**[1]
~66 mL, ~42 min

Currently approved therapies do not provide convenient, patient-friendly administration

3. **~$470,000**[2]
per year in PNH

High price point of currently approved therapies has limited use in broader indications

Complement is implicated in numerous diseases across many therapeutic areas

NEUROLOGY / NEUROMUSCULAR
Generalized Myasthenia Gravis (gMG)
Neuromyelitis Optica Spectrum Disorder (NMOSD)
Multifocal Motor Neuropathy (MMN)
Guillain–Barré Syndrome (GBS)
Chronic Inflammatory Demyelinating Polyneuropathy (CIPD)
Immune-Mediated Necrotizing Myopathy (IMNM)
Idiopathic Inflammatory Myopathies
Dermatomyositis
Amyotrophic Lateral Sclerosis (ALS)
Complex Regional Pain Syndrome (CRPS)
Traumatic Brain Injury (TBI)
Sub-arachnoid hemorrhage

NEPHROLOGY
C3 Glomerulopathy (C3G)
IgA Nephropathy
Membranoproliferative glomerulonephritis (MPGN)
Lupus Nephritis
Dense Deposit Disease (DDD)
Focal Segmental Glomerulosclerosis (FSGS)
Delayed Graft Function (DGF)
Antibody-mediated rejection (post kidney transplant)

INFLAMMATORY
ANCA-Associated Vasculitis
Antiphospholipid Syndrome (APS)
Systemic Lupus Erythematosus (selected subsets)

HEMATOLOGY
Paroxysmal Nocturnal Hemoglobinuria (PNH)
Cold Agglutinin Disease (CAD)
Atypical Hemolytic Uremic Syndrome (aHUS)
Immune Thrombocytopenia (ITP)
Transplant-Associated Thrombotic Microangiopathy (TA-TMA)
Sickle Cell Disease
CHAPLE disease (CD55 deficiency)
Pre-eclampsia

OPHTHALMOLOGY
Geographic Atrophy (Dry AMD)

RHEUMATOLOGY
Wegener's Granulomatosis (GPA)
Behçet's Disease
Sjögren's Syndrome
Systemic Sclerosis

DERMATOLOGY
Bullous Pemphigoid
Pemphigus Vulgaris
Epidermolysis Bullosa (EB)
Hidradenitis Suppurativa (HS)
Chronic Spontaneous Urticaria (CSU)
Palmoplantar Pustulosis
Pyoderma Gangrenosum
Lichen Sclerosus
Psoriasis

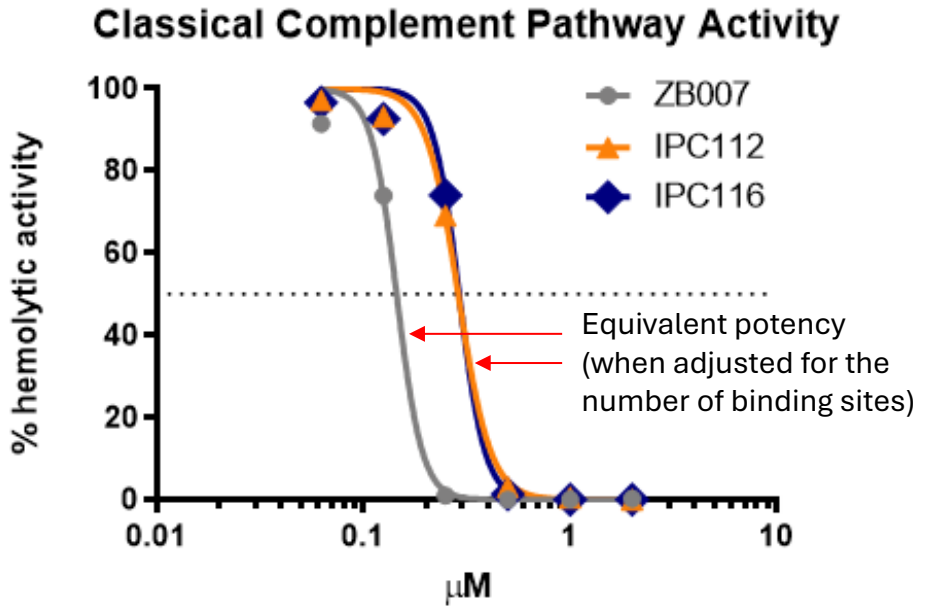
Sources: clintrials.gov; academic literature

[1]Ultomiris Package Insert; [2]U.S. annual price of Soliris or Empaveli

Confirmatory Clinical PK/PD Study

RLYB116 demonstrates potency equivalent to Ultomiris

- Both RLYB116 and Ultomiris bind to C5 and prevent its activation through competitive blockade of convertase
- RLYB116 affinity for C5 = ~ 400 pM kD
- Ultomiris affinity for C5 = ~ 500 pM kD
- The target concentration for Ultomiris is ≥ 175 µg/mL, which equates to a 2:1 molar ratio of binding sites to C5
- Therefore, a dose regimen which maintains RLYB116 levels at a molar ratio ≥ 2:1 over C5, should achieve complete inhibition- comparable to Ultomiris



	ZB007	IPC112	IPC116
IC50	0.1448	0.2879	0.2904

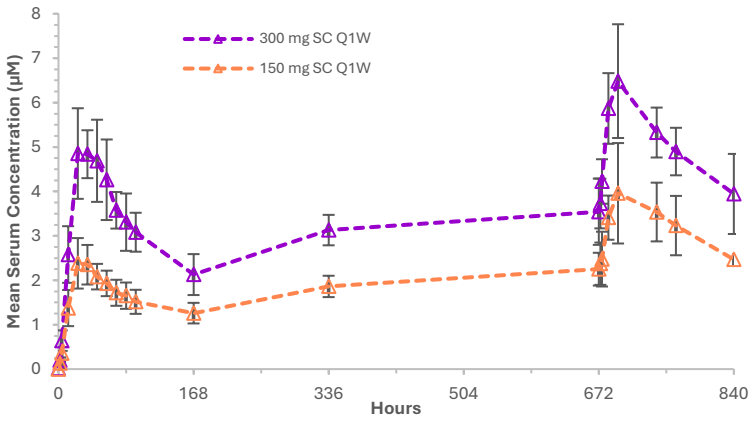
IPC116 = RLYB116 ZB007 = Ultomiris

RLYB116 therapeutic target concentration is guided by the pharmacodynamic parameters empirically established for efficacy with Ultomiris

Source : RLYB116 IPC2001

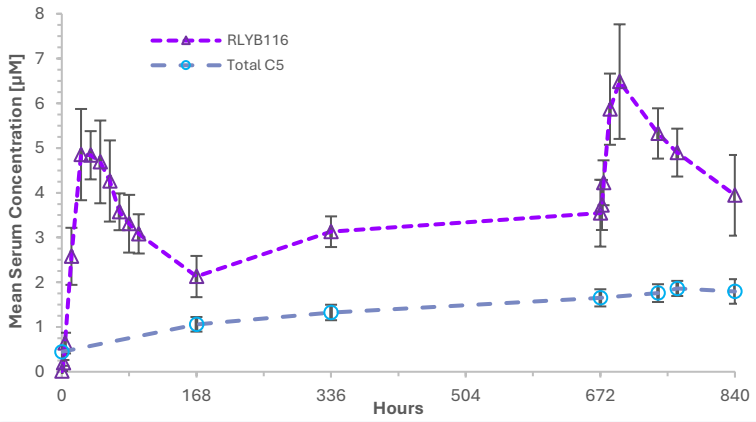
RLYB116 Phase 1 PK/PD demonstrated rapid, complete and sustained complement inhibition at the 300 mg dose

Serum RLYB116 Concentration



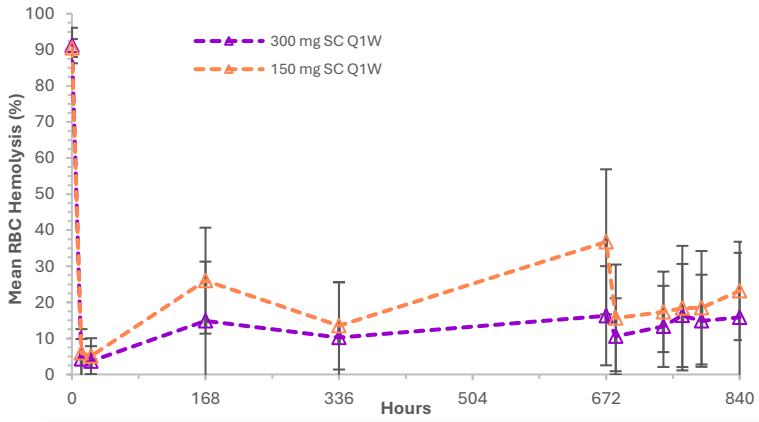
RLYB116 PK is highly predictable* with rapid absorption and long terminal elimination rates

RLYB116 to C5 Ratio



RLYB116 levels far exceed C5 within 12 hrs of first dose and are maintained at ratios > 2:1 with 300 mg SC Q1W dose regimen

Hemolytic Activity



Complete inhibition of hemolysis observed with 300 mg SC Q1W

Favorable tolerability observed at both dose levels

1: High frequency PK sampling was only conducted after first and last doses.
2: Samples at 336 hrs were collected at PK trough, immediately prior to 3rd dose

Improved gastrointestinal tolerability following manufacturing optimization

GI AEs Eliminated in Confirmatory Study vs. Initial Study

MANUFACTURING / STUDY	COHORT	GI-RELATED AES
Optimized Manufacturing *Confirmatory Phase 1*	**IPC2401 (150 mg Q1W)[1,3]**	**0 reports**
	IPC2401 (300 mg Q1W)[1,3]	**0 reports**
Original Manufacturing *Initial Phase 1*	IPC2001 (100 mg SAD)[1]	2 participants, 3 events
	IPC2001 (300 mg SAD)[1]	3 participants, 8 events
	IPC2001 (150 mg – 125 mg Q1W cohort 3 MAD)[2]	5 participants, 8 events

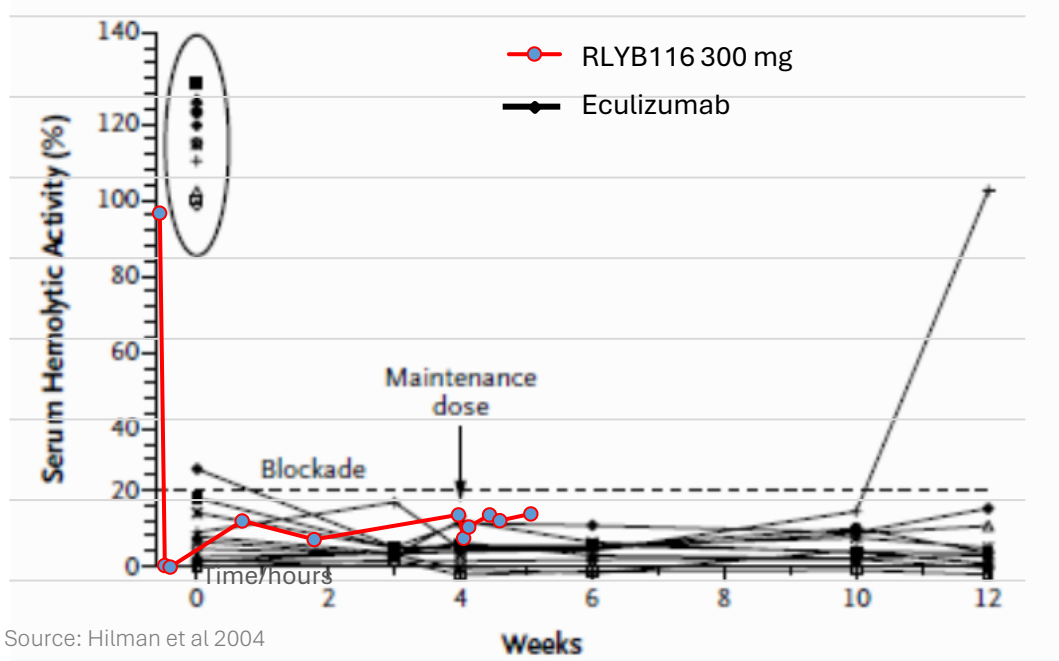
[1] n=6, [2] n=10, [3] reported from open data base

Dose limiting tolerability observed in the IPC2001 study resolved through the introduction of a **state-of-the-art** manufacturing process and forensic analytical techniques

Source : IPC2001 CSR

RLYB116 demonstrates complete and sustained terminal complement inhibition, comparable to eculizumab

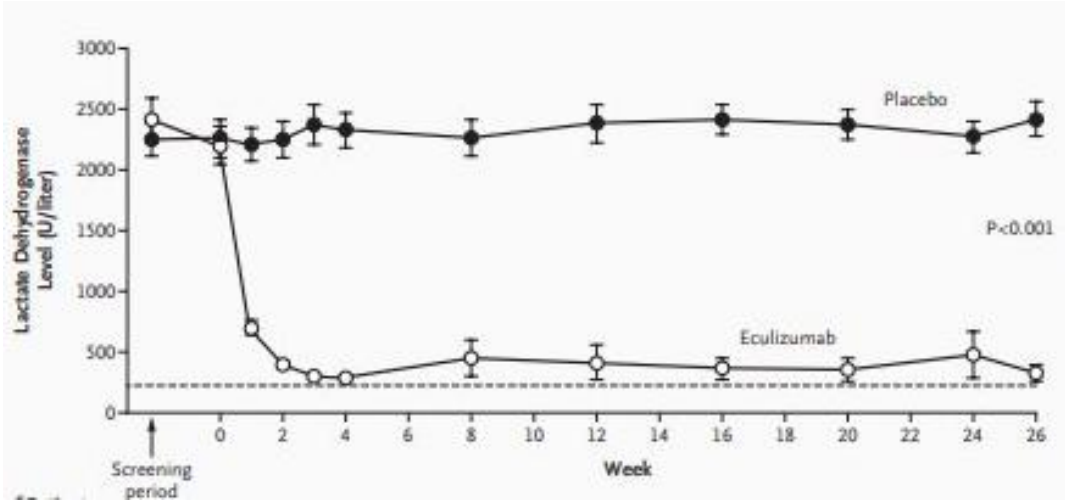
Ex Vivo Hemolytic Activity (RLYB116 vs Eculizumab)



Source: Hilman et al 2004

RLYB116 Phase 1 data demonstrates complete inhibition of complement through week 5, comparable to eculizumab[1]

In Vivo LDH Levels (Eculizumab vs. Placebo)



Source: Hilman et al 2006

In PNH patients, eculizumab demonstrated sustained reductions in LDH, consistent with effective inhibition of terminal complement activity

1: No head-to-head clinical trials have been conducted

Rapid, complete and sustained complement inhibition at the 300mg dose – game, set, match

- RLYB116 Phase 1 PK/PD demonstrated rapid, complete and sustained complement inhibition at the 300mg dose
- Favorable tolerability observed at both the 150mg and 300mg dose
- GI AEs eliminated following manufacturing optimization
- RLYB116 exhibits complete and sustained suppression in the same functional assay as eculizumab, supporting its translational potential
- Clinical experience with eculizumab demonstrates sustained functional complement inhibition translates to complete control of LDH in PNH patients
- Strong clinical data support progression into Phase 2 studies with initial focus on immune PTR and APS (with additional funding)

Immune PTR

Immune Platelet
Transfusion Refractoriness

Immune Platelet Transfusion Refractoriness

OPPORTUNITY

Patient Population:
>25,000 annually (major markets)

Unmet Need:
Many patients refractory to standard of care and experience relapses

Current Standard of Care:
Platelet matching

Commercial Potential:
$1.1B Peak Market Opportunity

STRONG BIOLOGICAL RATIONALE

Existing POC
Published data from IIT with eculizumab Tx demonstrate profound improvement in 40% of patients with immune PTR[1]

Differentiation
Ease of administration (autoinjector with no IV loading dose) in a complex patient care setting and lower COGS

PROPOSED TRIAL DESIGN

- **Phase:** Phase 2 (Proof of Concept)
- **Design:** Single-arm adaptive* trial with patients receiving a weekly single dose SC administration of RLYB116
- **Patients:** 10 patients*
- **Primary Endpoint:** Sustained Platelet Transfusion Responsiveness
- **Secondary Endpoint:** Safety, tolerability, PK/PD

ANTICIPATED TIMELINE & ESTIMATED COST

- **Study Start:** 2H 2026
- **POC Data:** Within 12 months of study start
- **Phase 2 total:** ~$4.8M
- **Key Drivers:** Sites, drug supply
- **Efficiency Levers:** Small N, clear endpoints

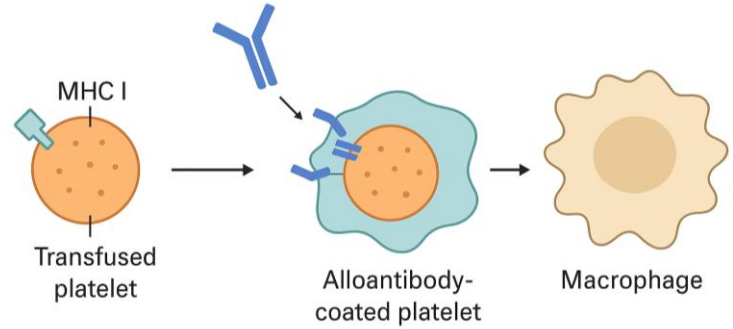
*10 patients estimated to confirm 40% response reported by Vo et al. Adaptive design allows empiric-based adjustment
1: Vo et al, 2020

Immune PTR is persistent low platelet counts, caused by immune-mediated platelet depletion pathways

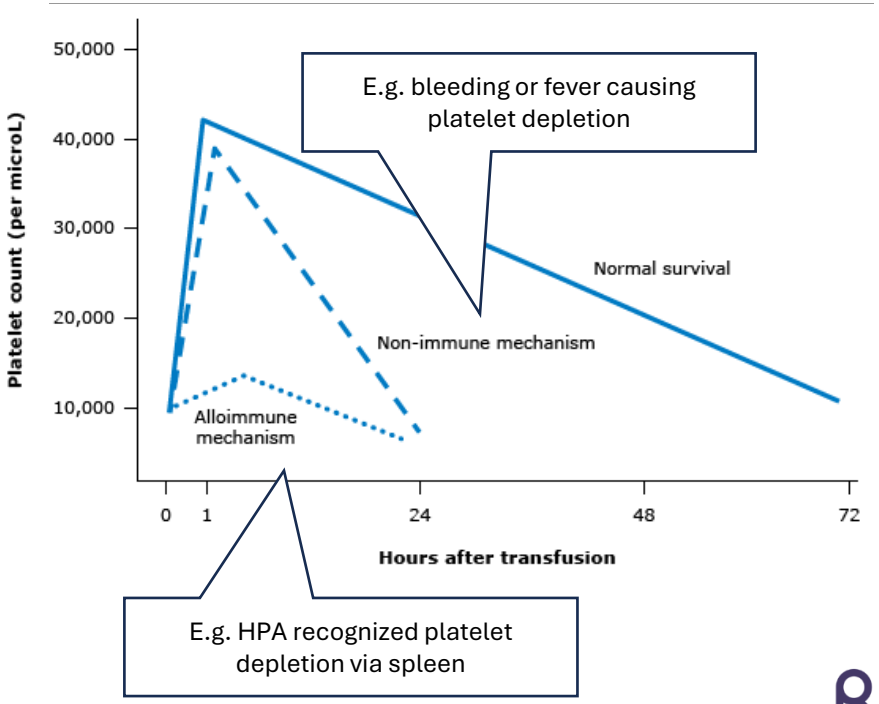
Immune caused platelet transfusion refractoriness (IPTR), is a **persistently low platelet count** after transfusion caused by immune recognition

- **Etiology**: Platelet refractoriness can be caused by non-immune causes or immune causes: *non-immune factors are more common*
- **Pathophysiology**: Physiological process of recognized platelets being depleted via spleen through an e.g. complement mediated process
- **Diagnosis:** Platelet count increment is less than expected, after 2+ platelet transfusions, immune subtype diagnosed depending on timed platelet depletion and panel reactive antibodies (PRA)
- **Treatment: Currently matched platelet transfusion, no approved Tx**

PROGNOSIS: Can persist for weeks, months despite matched donors, increased mortality in non-transfused patients, with transfusion slightly improving survival in specific patient groups, PTR was associated with adverse events (longer hospital stays, severe hemorrhages, may have a negative impact on the success of HSCT)



Immune mechanism of platelet transfusion refractoriness in HLA alloimmunized patients



Source: Scitaris IPTR Deep-dive 2025

IIT with eculizumab demonstrates complement blockade is beneficial in immune PTR

40% responded to C5 blockade with eculizumab

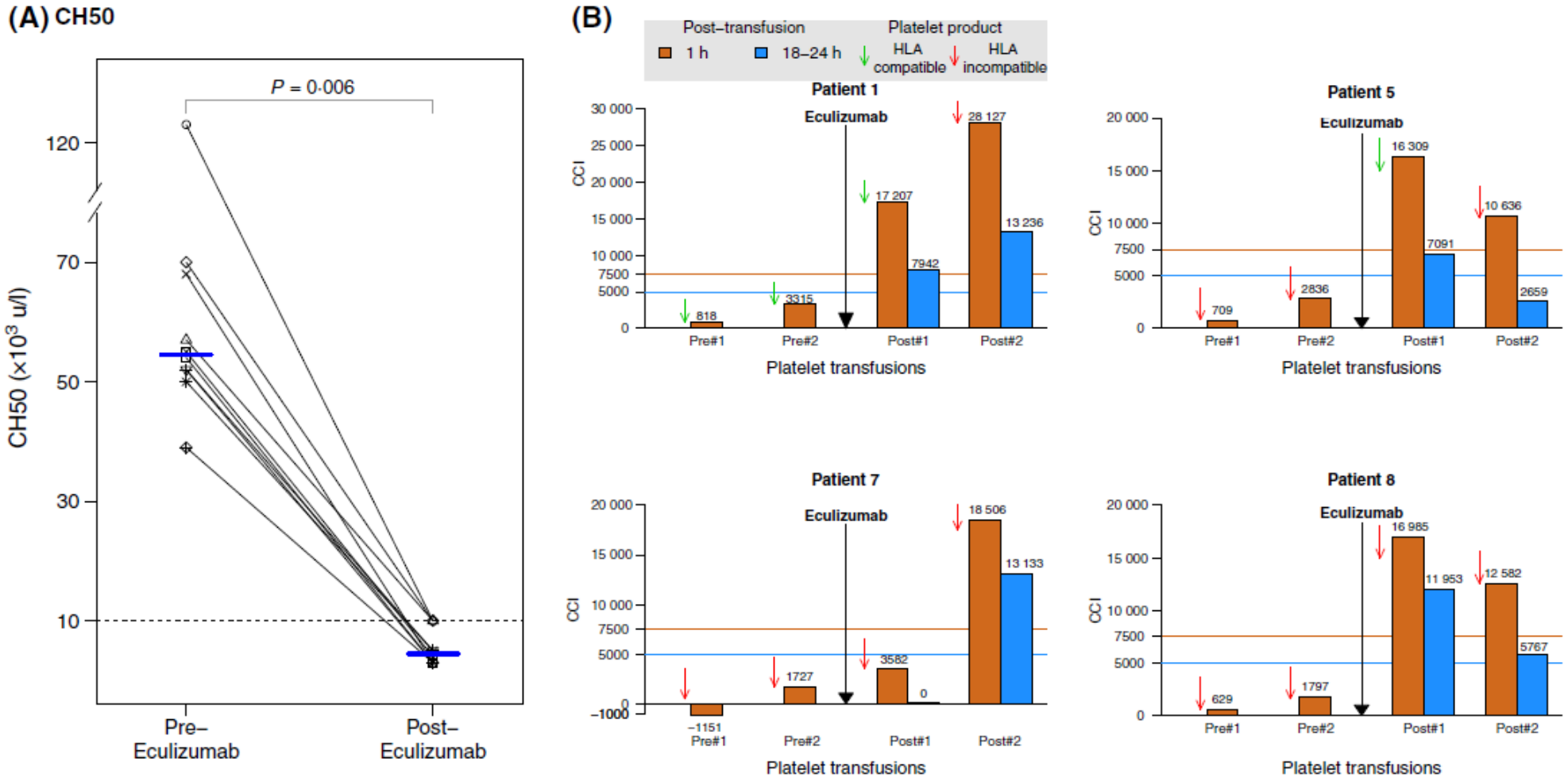


Fig 1. (A) Total complement (CH50) levels pre and post eculizumab treatment. Median (horizontal lines) are shown at the two time points. (B) Response to platelet transfusion refractoriness post eculizumab in the four responding patients. One-hour (10-60-min, orange bars) and 18-24 h (blue bars) post platelet transfusion CCIs are shown in patients before and after receiving eculizumab. Responses were defined by 1-h CCI > 7500 together with the 18-24-h CCI > 5000 following a platelet transfusion. Green and red arrows indicate HLA-compatible and HLA-incompatible products, respectively. [Colour figure can be viewed at wileyonlinelibrary.com]

Source: Vo et al, 2020

Immune PTR offers a unique fast POC opportunity enabling advancement in an untreated condition AND unlocks all other complement diseases

TRIAL DESIGN

Adaptive single-arm trial in 10 patients receiving a single dose SC administration of RLYB116

Subject Screening

RLYB116 Once-Weekly Dosing

INCLUSION CRITERIA

Detectable anti-HLA A and/or B antibodies and ≥2 consecutive occurrences of less than adequate CCI: CCI < 7500 at 10-60 min and CCI < 5000 at 18–24 h after each platelet transfusion

ASSESSMENTS

Response to platelet transfusion following RLYB116 administration, PK/PD and safety

PRIMARY OBJECTIVE

Sustained Platelet Transfusion Responsiveness

SECONDARY OBJECTIVE

Safety, tolerability, PK/PD

CCI: Corrected Count Increment; SC: Subcutaneous; HLA: human leukocyte antigen

APS

Refractory Antiphospholipid Syndrome

Refractory Antiphospholipid Syndrome

OPPORTUNITY

Patient Population:
20,000 annually (refractory)

Unmet Need:
No approved therapies addressing the underlying disease biology

Current Standard of Care:
Chronic anticoagulation (e.g., warfarin) with significant bleeding risk and incomplete protection

Commercial Potential:
$4.0B Peak Market Opportunity (major markets)

Recurrent venous and arterial thromboses despite anticoagulation therapy

STRONG BIOLOGICAL RATIONALE

Existing POC
Published data from off-label Tx with eculizumab and ravulizumab demonstrate **complete prevention** of recurrent thrombosis in patients refractory to standard of care with a history of frequent thrombotic events[1]

Differentiation
Patient centric product profile and lower COGS

PROPOSED TRIAL DESIGN

- **Phase:** Phase 2 (Proof of Concept)
- **Design:** Single-arm adaptive* trial in refractory APS patients receiving RLYB116 on top of standard of care
- **Patients:** 4-6 patients*
- **Primary Endpoint:** Composite thrombotic events
- **Secondary Endpoint:** Safety, tolerability, PK/PD

POTENTIAL TIMELINE & ESTIMATED COST

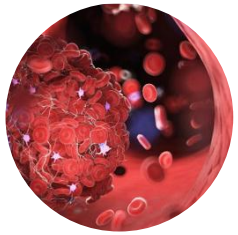
- **Study Start:** TBD with financing
- **Top-Line Data:** 26 & 52 weeks – absence of thrombosis
- **POC Data:** Within 24 months of study start
- **Phase 2 total:** Est. ~$10M
- **Key Drivers:** Treatment duration, drug supply
- **Efficiency Levers:** Small N, clear endpoints

*4-6 patients estimated to confirm response observed with off-label use reported in Ranjan et al. Adaptive design allows empiric-based adjustment

1: Ranjan et al, 2025

APS is a rare autoimmune disease that can cause potentially catastrophic thrombotic events

PATHOPHYSIOLOGY



APS is characterized by antiphospholipid antibodies (aPL), which attack phospholipids and binding proteins, causing coagulation in arteries and veins

aPLs are typically detected by three tests: lupus anticoagulant (LA), anticardiolipin antibody (aCL), and anti-Beta-2-glycoprotein-I antibody (aβ2GPI)

DISEASE OVERVIEW

Symptoms and Prognosis

- Patients typically present with blood clots in the veins of the extremities, stroke, or pregnancy problems
- Can present as a primary condition or as "secondary APS" in the context of systemic autoimmune diseases
- A rare type of APS (~1% of cases), termed Catastrophic APS (CAPS), is most severe form, defined by mortality risk from concurrent microvascular and macrovascular thrombotic complications in multiple organ systems

U.S. / EU5 Epidemiology

- Prevalence in U.S. / EU5 is ~40-50 per 100 K
- Approximately 75% have at least 1 thrombotic event
- An estimated 10% of diagnosed patients have persistent thromboses despite anticoagulation therapy

CLINICAL CONSIDERATIONS

Diagnostic Pathway and Site of Care

ER | Rheum | Heme | MFM

- Patients typically present at the ER with a thrombotic event
- With thrombotic recurrence, patients are typically referred to a hematologist for diagnosis and treatment
- Secondary APS patients are likely diagnoses by rheumatologists

Clinical Manifestation

Venous thrombosis	Arterial thrombosis	Heart valve disease	Livedo racemosa
Pregnancy Loss	Thrombo-cytopenia	aPL nephropathy	White matter lesions

+

Laboratory Tests

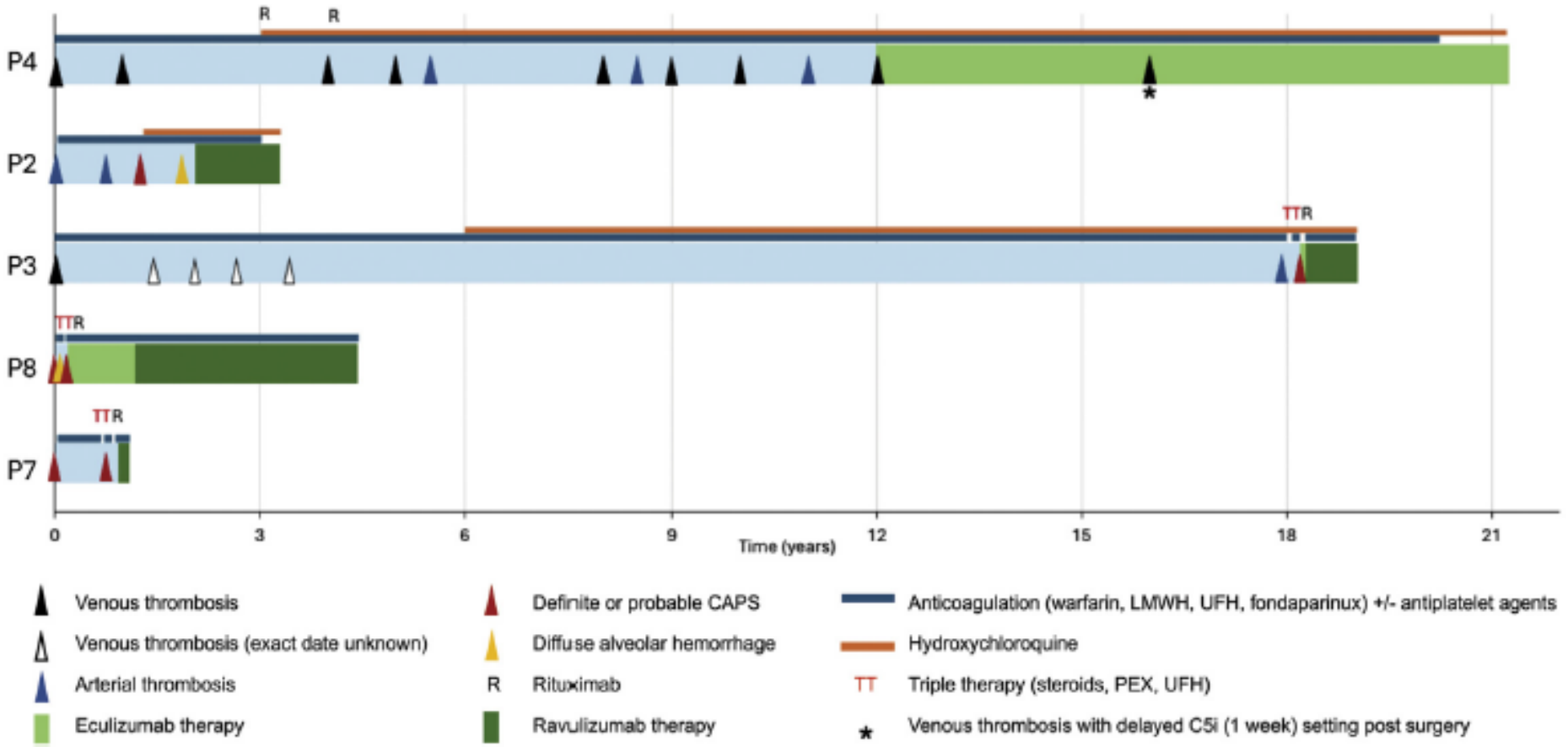
Run after acute event: LA test, aCL, aβ2GPI → At least 1 positive result → Run 12+ wks later: LA test, aCL, aβ2GPI → 2nd positive result

Criteria	Factors that increase Dx confidence			
Non-criteria	Criteria + non-criteria manifestations	Higher titer	+ IgG	+ LA

Sources: HSS.edu/healthlibrary; BlueStar BioAdvisors APS Report Nov 2024

Off–label usage with eculizumab/ravulizumab demonstrates complement blockade is effective in refractory APS

C5 blockade prevents thrombotic events in refractory APS patients



Ranjan et al Ann Rheum Dis 2025 – C5 inhibition mitigates thrombosis in CAPS

Five patients from the CAPS cohort (P2, P3, P4, P7, and P8) were treated with either eculizumab or ravulizumab at the discretion of the treating clinician (duration of therapy 2 months to 9 years).Collectively, these patients had over 40 thrombotic events despite therapeutic anticoagulation before therapeutic C5 inhibition. Since starting therapy, there has been only 1 thrombotic event when P4 received her eculizumab 1 week after it was due. Since restarting eculizumab, she has had no further thromboses despite discontinuing her anticoagulation over 1 year ago. The swimmer plot summarises the thrombotic events and duration of C5i .

RLYB116 APS proof-of-concept clinical trial design

TRIAL DESIGN

Adaptive single-arm trial in 4-6 refractory APS patients receiving SC administration of RLYB116 on top of standard of care for 52 weeks

Subject Screening

RLYB116 Once-Weekly Dosing

INCLUSION CRITERIA

Refractory APS patients with multiple recurrent thrombotic events (several times per year) despite standard of care

ASSESSMENTS

Prevention of thrombotic episodes (composite of thrombotic events – venous, arterial, possibly small vessel), PK/PD, safety

PRIMARY OBJECTIVE

Composite thrombotic events 52-week absence of Thrombosis

(interim data readout at 26 weeks)

SECONDARY OBJECTIVE

Safety, tolerability, PK/PD

Competitive Landscape

RLYB116 is positioned for success within the commercial landscape

Confident we can achieve POC in immune PTR and APS – high value indications facilitating future indication expansion

- Expert team can leverage existing POC achieved with eculizumab/ravulizumab Investigator Initiated Trials
- Limited competition and standard of care is inadequate

We believe RLYB116 profile is favorable versus anti C5 mAbs (such as crovalimab)

- Eculizumab-like potency, rapid complete and sustained complement inhibition[1]
- Q1W small volume autoinjector, with no loading dose, favored by both patients and in clinical settings
- No refrigerated storage required for end user
- Manufacturing process allows cost flexibility – can compete with biosimilars and other MOAs (e.g. FcRns)

C5 has advantages over C3 inhibition (such as pegcetacoplan)

- C3 inhibitor black-box warning broader than C5 inhibitors (not just *Neisseria meningitidis*- all encapsulated bacteria)
- Breakthrough events related to either insufficient PK or Complement-Amplifying-Conditions are much more severe in patients receiving chronic C3 inhibitors

C5 inhibition is a better approach than C1s (such as claseprubart)

- C5 is the most effective pathway to achieve complete complement inhibition, with decades of patient safety data and easy risk management strategies
  - C1s antagonism only achieves partial complement inhibition - In Ph 1, claseprubart did not demonstrate complete suppression
  - Chronic C1s deficiency is strongly associated with risk of SLE - Ph 2 gMG study patients receiving claseprubart frequently developed ANA positivity. However, no cases of SLE reported.

Sources: Ultomiris and Empaveli black-box warnings, Dianthus Corp Presentation Mar 2025, Scitaris and Bluestar reports

1: No head-to-head clinical trials have been conducted

RLYB116 is differentiated, with clinically-demonstrated best-in-class potential[1]

	Target/Modality	Stage	Once-weekly, one-shot SC "Autoinjector pen" compatible No loading dose	Well-understood safety profile Good toleration Easily-managed AE risk#	Complete and sustained Complement inhibition achieved with standard dosing
RLYB 116 Rallybio[2]	**C5 peptide**	**Entering Ph II**	✓	✓	✓
Eculizumab AstraZeneca	**C5 mAb**	**Marketed**	✗	✓	✓
Ravulizumab AstraZeneca	**C5 mAb**	**Marketed**	✗	✓	✓
Zilucoplan UCB	**C5 peptide**	**Marketed**	✓	− *	✗
Crovalimab Roche	**C5 mAb**	**Marketed**	✗	− **	✓
Cemdisiran Regeneron	**C5 oligo** (combo with pozelimab)	**Phase III**	✓ **ALONE** ✗ **IN COMBO**	− ***	✓ **IN COMBO** ✗ **ALONE**
Gefurulimab AstraZeneca	**C5 mAb**	**Phase III**	✗	✓	?
Claseprubart Dianthus	**C1s mAb**	**Phase III**	✗	− ****	−

* FDA warning for pancreatitis

** Contraindication for hypersensitivity

*** 8% SAE in combo treatment in PNH Ph III

**** Dose related increase seen in ANA (Lupus connected biomarker) in Ph III- 36% newly positive for ANA at high dose. No cases of SLE reported

\# Absence of adverse events (except those commonly associated with class or route-of-administration, e.g. no contraindications besides Neisseria meningitidis infections) and absence of anti-drug antibody impact

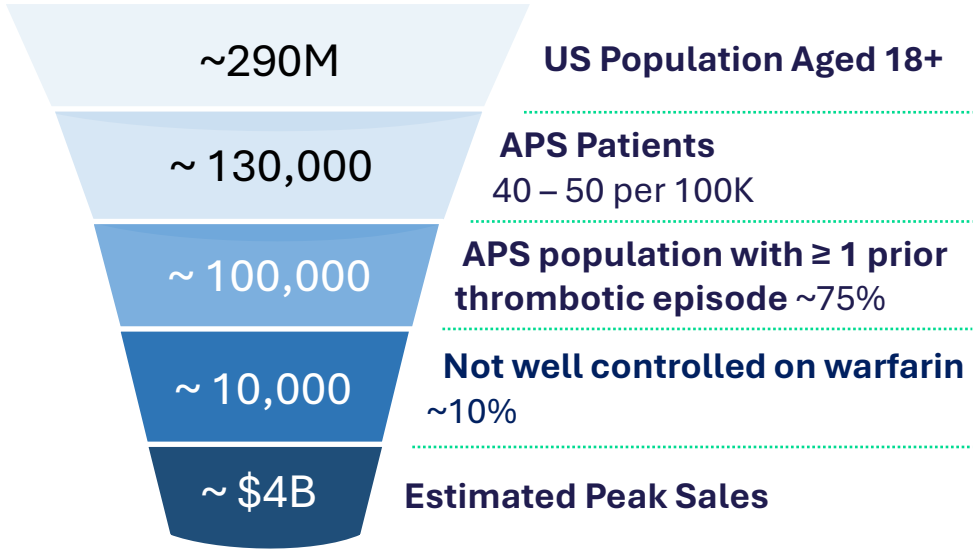
Source: Information from Scitaris competitive landscape review, Oct 2025

1: No head-to-head clinical trials have been conducted

2: Based off Phase 1 confirmatory PK/PD study

Epidemiology estimates for Immune PTR and Refractory APS target populations in the major markets support a combined peak commercial opportunity of $>5B

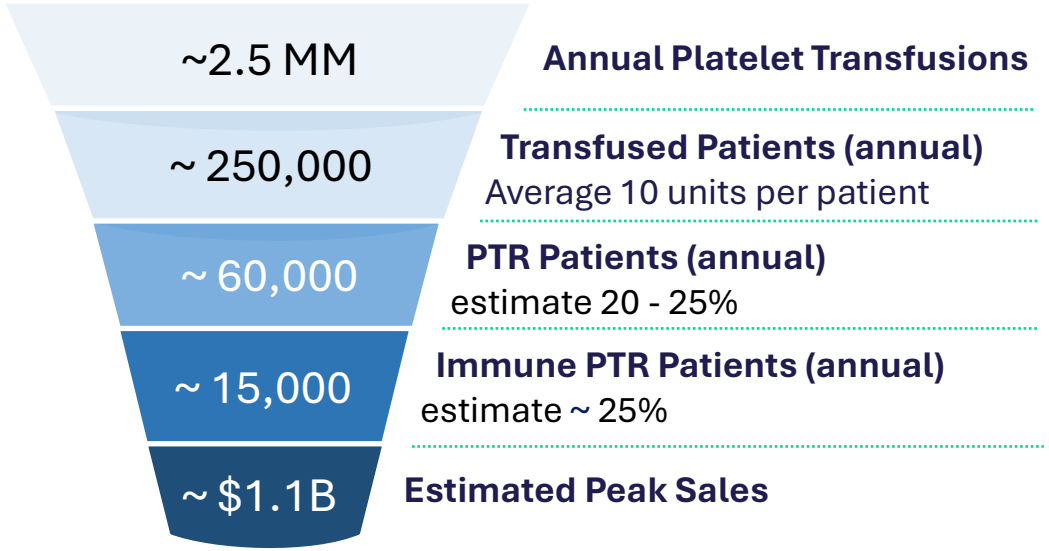
US Prevalent **APS** Population



Assumes chronic prophylactic therapy

Sources utilized to inform modeling: Bluestar APS market assessment report Nov 2023, PopulationPyramid.net, Cervara et al 2002, Cervara et al 2015

US Incident **IPTR** Population



Assumes one treatment course per patient

Sources utilized to inform modeling: Scitaris IPTR market assessment report Dec 2025, Bluestar IPTR market assessment report Nov 2025, PopulationPyramid.net

Combined estimated peak commercial opportunity is >$5B in the 7 major markets (US, EU4 + UK, Japan)

RLYB116 is a pipeline-in-a-product with potential to be a first- and best-in-class treatment for severe, refractory hematologic diseases

~$5B Market Opportunity

Immune Platelet Transfusion Refractoriness

Serious complication that increases risk of morbidity and mortality from bleeding events

>25,000 patients annually* >> $1.1B peak commercial opportunity

C5 inhibition has demonstrated ability to overcome immune PTR

No approved therapies exist; highly limited competitive landscape; ***rapid time to POC***

Phase 2 trial anticipated to initiate in 2H 2026

Refractory Antiphospholipid Syndrome

Patients at high risk of serious complications from uncontrolled thrombotic events

>20,000 patients* >> ~$4.0B peak commercial opportunity

C5 inhibition demonstrated to significantly reduce thrombotic events in refractory APS

No approved therapies exist; highly limited competitive landscape

Phase 2 trial with additional capital or a partner

Future Potential Indications

Antibody-Mediated Rejection

Generalized Myasthenia Gravis

Atypical Hemolytic Uremic Syndrome (+ related TMAs)

Paroxysmal Nocturnal Hemoglobinuria

Pipeline-in-a-product represents a multibillion market opportunity

*Across seven major markets

Milestones

Rallybio is positioned for meaningful value creation in 2026 and beyond

PILLARS OF OUR SUCCESS

- Potentially best-in-class program aimed at addressing unmet needs in devastating rare diseases
- RLYB116 offers meaningful potential for value creation
- Proven innovators in rare disease research and development

ANTICIPATED MILESTONES

PROGRAM	MILESTONE	EXPECTED TIMING
RLYB116	Initiate Phase 2 in Immune PTR	2H 2026
	Phase 2 Interim Data in Immune PTR (~5 subjects)	2Q 2027
	Phase 2 POC Data in Immune PTR	2H 2027
REV102	Receipt of Phase 1 study milestone	2026