JUNE 6, 2024

Annual Meeting of Shareholders

Gregory A. Demopulos, MD
Chairman & CEO

Safe Harbor Statement

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Corporate Priorities Represent Catalysts of Near-/Mid-Term Value and Growth

Substantial Cash Runway without Shareholder Dilution

- $230 million of cash and equivalents available for operations as of March 31, 2024
- Runway expected to fund operations into 2026
- <58 million shares outstanding as of March 31, 2024
- As of June 3, 2024, further strengthened balance sheet through financing transactions that extinguished or extended maturity on a majority of outstanding debt into 2028

Existing Opportunities for Future Cash Infusions

- Up to $55 million in milestones based on US OMIDRIA sales*
- Royalties on ex-US OMIDRIA sales; and all global sales after January 1, 2032
  - Royalties extend until patent expiry in US and other relevant jurisdictions – at least 2035
- OMIDRIA separate payment legislatively secured until at least January 2028 (ASCs already underway; HOPDs start January 2025)

Driving Significant Progress across Assets

- Multiple de-risked clinical programs for zaltenibart (OMS906) slated to advance into Phase 3 late 2024 early 2025
- FDA interactions ongoing regarding BLA resubmission for narsoplimab (OMS721) in TA-TMA
- OMS1029 expected to advance into Phase 2 by 2025 in one of several indications being evaluated
- Data from immuno-oncology platforms expected in 3Q 2024

* Milestones are payable under royalty monetization arrangement with DRI Capital, the purchaser of Omeros' royalty rights on U.S. OMIDRIA sales through 2031. Since its market launch in 2015, OMIDRIA has fueled the Company's innovation pipeline with ~$1 billion in non-dilutive capital.

Debt Exchange Transactions: Substantial Step in Strengthening our Capital Structure

Secured Term Loan Overview

- $67.1 million initial principal amount
- 4-year term with maturity in June 2028
- Quarterly interest at adjusted term SOFR + 8.75%
- Partial prepayment of $20M principal required by November 1, 2025 unless 2026 note balance < $38.5M
- $25 million delayed draw term loan for launch of narsoplimab

Key Highlights

- No equity component included; preventing shareholder dilution
- Pricing at 74.75% represents no premium to market range
- Highly competitive interest rate
- Lower net principal and interest payments over the term of 2026 Notes
- Omeros retains all potential future value of the capped call purchased in connection with the 2026 notes

Debt Exchange Transactions: Substantial Step in Strengthening our Capital Structure

De-Risked Balance Sheet

- $118.1 million (55%) of outstanding 2026 convertible notes repurchased at ~75% of notional value
- Extinguished $51 million of total debt

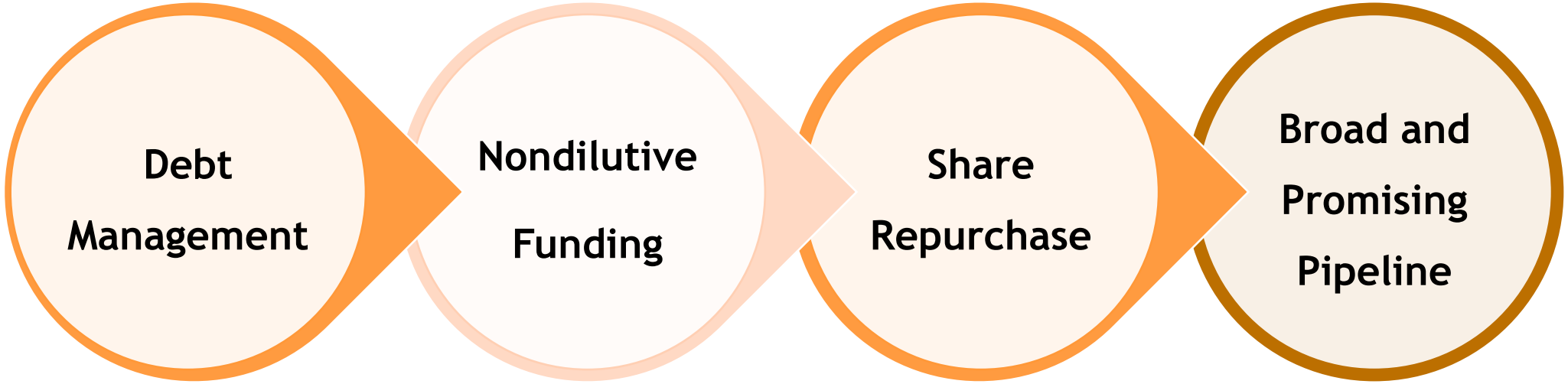
Additional Debt Reduction Enabled

- Can make open market and private repurchases of outstanding 2026 Notes, subject to certain limitations
- Can exchange additional $16.9 million notional in 2026 notes for cash and/or more term loan

Expected Key Value-Driving Milestones through June 2028 Maturity Date Extension

- Narsoplimab established as first-line therapeutic
- Zaltenibart commercialized
- OMS1029 advancing through Phase 3
- OMS527 and immuno-oncology programs clinically advancing

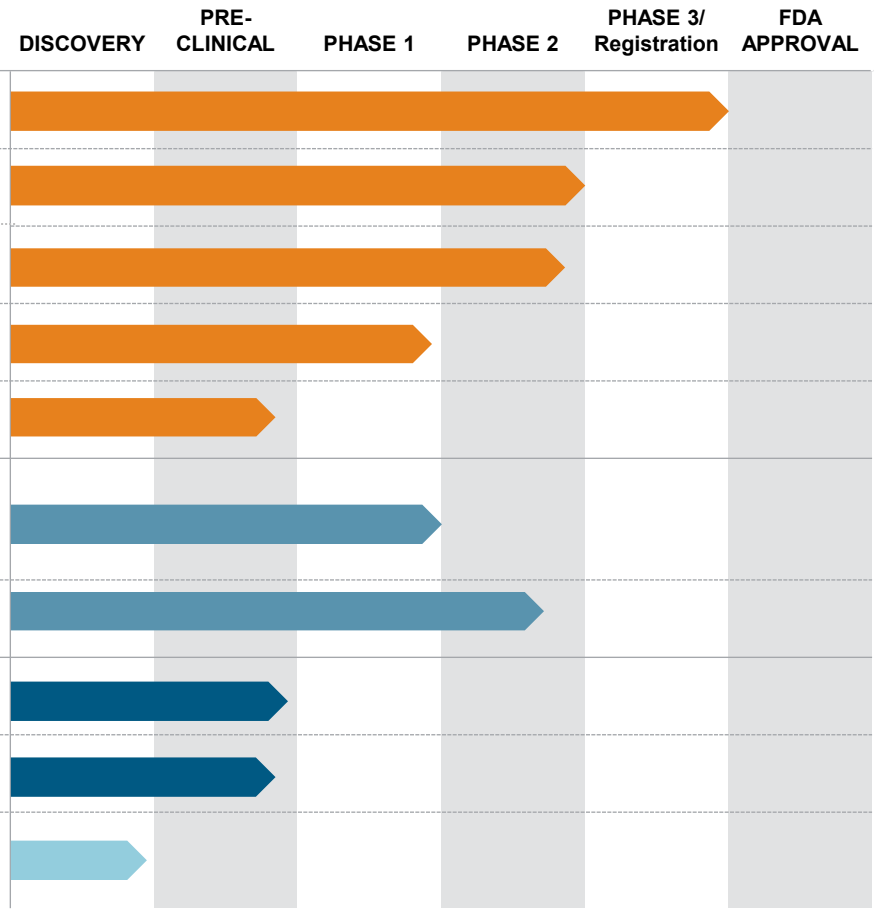
Resource Allocation that Drives Shareholder Value



- Since its market launch in 2015, OMIDRIA has fueled the company's innovation pipeline with ~$1 billion in non-dilutive capital — ~$700 million since its sale to Rayner in December 2021
- 8% of outstanding OMEROS shares (5 million) repurchased over 4Q 2023 and 1Q 2024

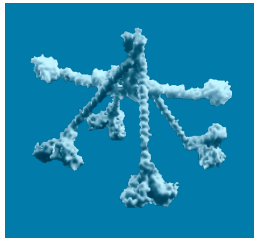
Pipeline of Novel Targets and Programs: Each Proprietary to Omeros

	PROGRAM / (CANDIDATE)	MOLECULE	TARGETED DISEASE
COMPLEMENT FRANCHISE	MASP-2, Lectin Pathway (narsoplimab [0MS721])*	Ab	Stem Cell Transplant-Associated TMA
			COVID-19 and ARDS
	MASP-3, Alternative Pathway (zaltenibart [OMS906])*	Ab	PNH, C3 Glomerulopathy, and Other Alternative Pathway Disorders
	MASP-2 (OMS1029)*	Ab	Long-Acting 2nd Generation Antibody Targeting Lectin Pathway Disorders
	MASP-2, MASP-3, MASP-2/3*	SM	Disorders of the Lectin and Alternative Pathways of Complement
SUBSTANCE ABUSE DISORDERS AND COMPULSIONS	PDE7 (OMS527)*	SM	Substance Abuse and Compulsive Disorders; Movement Disorders
	PPARγ (OMS405)	SM	Opioid and Nicotine Addiction
IMMUNO-ONCOLOGY	Cellular Therapies (adoptive T cell and CAR T)	SM/LM	Cancer
	Biologic Therapeutics (immunomodulator, oncotoxins, cancer vaccines)	SM/LM	Cancer
DISCOVERY PLATFORM TECHNOLOGIES	GPCR Platform	SM	Immunologic, CNS, Metabolic, CV, Musculoskeletal and Other Disorders



Notes: Ab, antibody; ARDS, acute respiratory distress syndrome; CNS, central nervous system; COVID-19, coronavirus disease 2019; CV, cardiovascular; FDA, Food and Drug Administration; GPCR, G protein-coupled receptors; LM, large molecule; MASP, mannan-binding lectin-associated serine protease; PDE7, phosphodiesterase 7; PNH, paroxysmal nocturnal hemoglobinuria; PPARγ, peroxisome proliferator-activated receptor gamma; SM, small molecule; TMA, thrombotic microangiopathy
*Program directed to inhibiting associated target

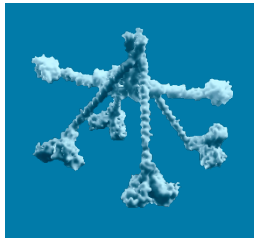
The Premier Complement Franchise With First-in-Class Therapeutics



MASP-2
Effector enzyme of the lectin pathway

Narsoplimab

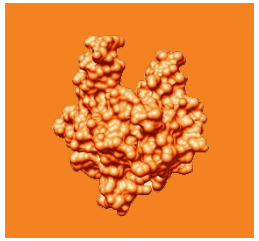
- Efficacy observed in TA-TMA, LN, aHUS and broader EIS; no safety signal of concern
- Ongoing discussions with FDA toward resubmission of BLA in TA-TMA
- Development as a therapeutic with associated diagnostic tests for acute and long COVID-19 and ARDS under discussion



MASP-2
Effector enzyme of the lectin pathway

OMS1029

- Long acting, quarterly IV or SC ideally suited for chronic administration
- Phase 1 SAD completed; Phase 1 MAD wrapping up mid-2024; no safety signals of concern
- Several large-value indications under review; indication selection for Phase 2 development planned in 3Q 2024



MASP-3
Key activator of the alternative pathway

Zaltenibart (OMS906)

- Interim data from 2 ongoing Phase 2 PNH studies demonstrate strong efficacy and safety profile
- Designed to prevent breakthrough of underlying disease and preserve lytic function of classical pathway to fight infections
- Long acting, up to quarterly IV or SC, ideal for patient adherence; advancing 2 treatment regimens – every 8 weeks and every 12 weeks – to provide options to suit patient and physician preferences

MASP-2 and -3 Small-Molecule Programs

- Designing for life-cycle management to deliver enhanced attributes over narsoplimab and zaltenibart in specific indications
- Ideally suited for chronic administration in treatment and prevention of large-market diseases
- Together with our antibody programs, expected to provide exclusive control of MASP-2 and MASP-3 inhibitor franchises

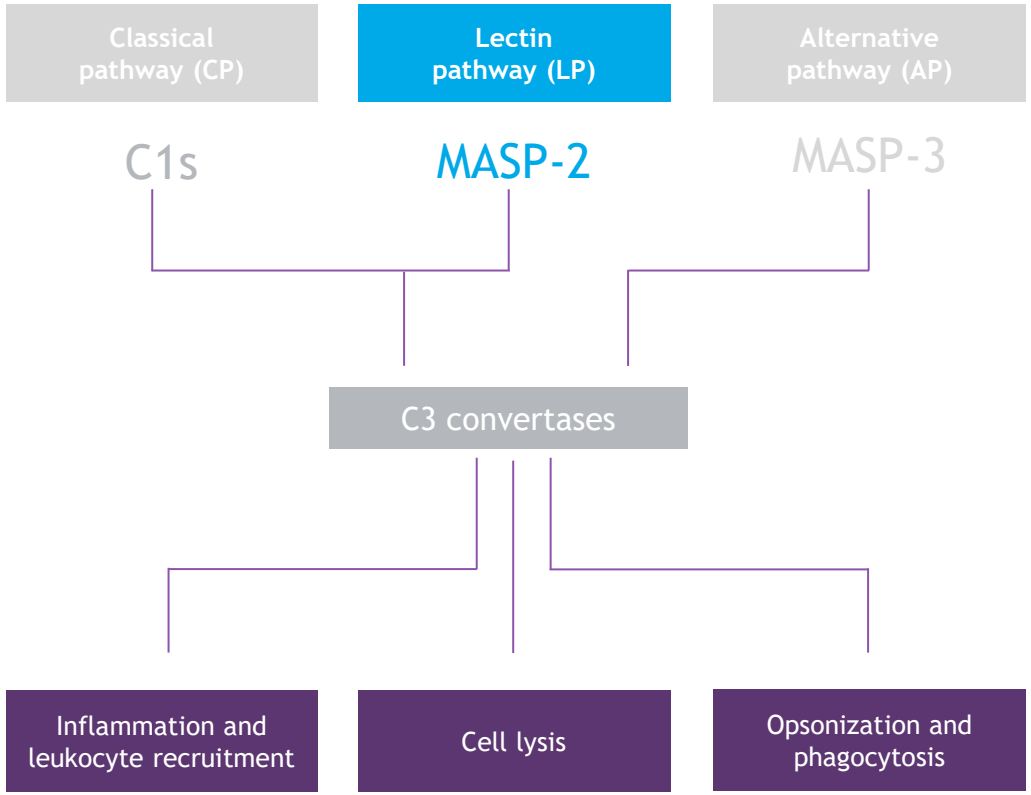
TA-TMA, hematopoietic stem cell transplant-associated thrombotic microangiopathy; LN, lupus nephritis; aHUS, atypical hemolytic uremic syndrome; EIS, endothelial injury syndromes

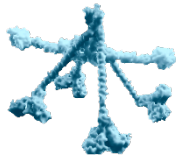
MASP-2 Inhibitor Programs

Narsoplimab (OMS721) and OMS1029

MASP-2 is the Key Activator of the Lectin Pathway of Complement

Role of MASP-2 in the complement system





MASP-2

Effector enzyme of the lectin pathway

The **lectin pathway** is a pattern-recognition system, and its dysregulation is associated with many diseases (e.g., TA-TMA, ischemia-reperfusion injury)

Narsoplimab is a first-in-class, potent and selective inhibitor of MASP-2 for acute or episodic treatment of serious/life-threatening conditions

OMS1029 is a potent and selective, long-acting inhibitor of MASP-2, better suited for chronic administration compared to narsoplimab

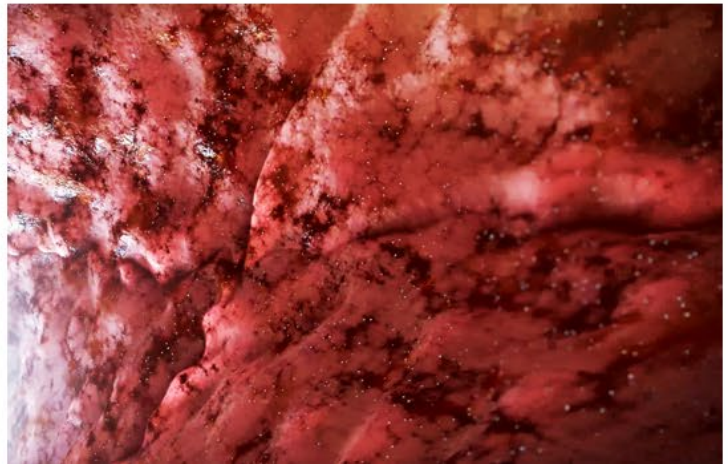
Small-molecule MASP-2 inhibitors nearing selection of drug development candidate

Notes: TA-TMA, transplant-associated thrombotic microangiopathy; aHUS, atypical hemolytic uremic syndrome; SM, small molecule

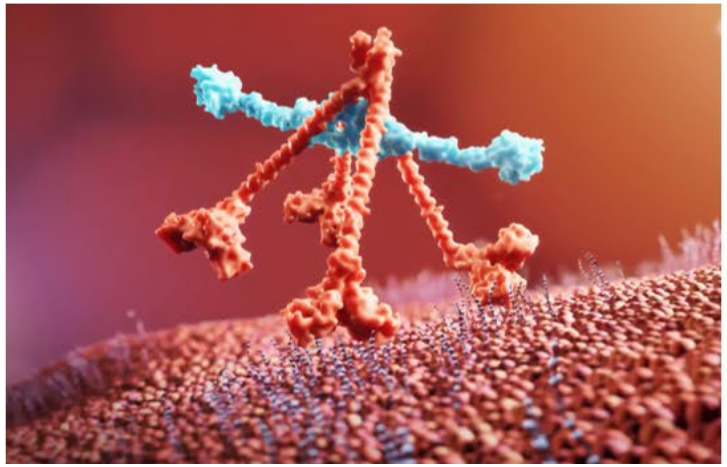
Source: Dunkelberger JR et al. *Nat. Cell Biol.* 2010

TA-TMA Disease Process

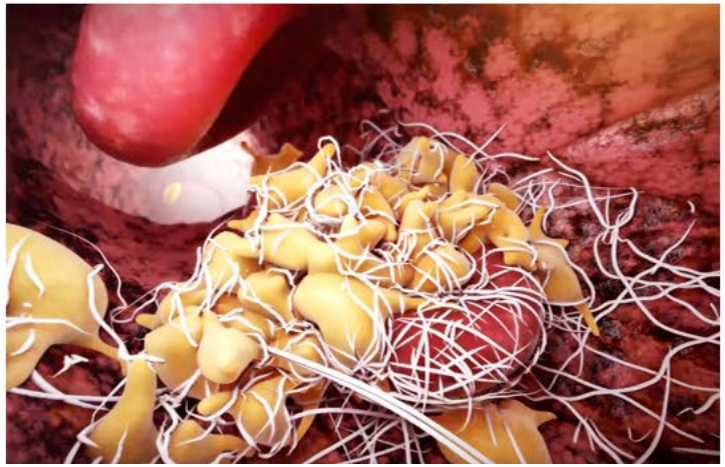
TA-TMA is initiated by endothelial injury associated with the stem cell transplantation journey



Endothelial injury



Complement activation



Thrombus formation

Endothelial injury activates the complement system's lectin pathway[1]

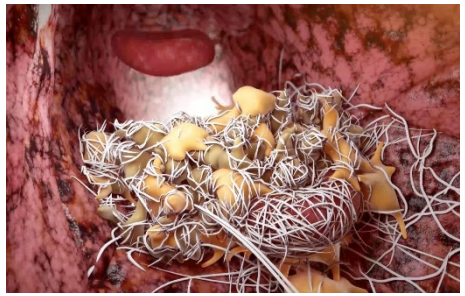
Complement activation leads to inflammation and thrombus formation that result in organ dysfunction and failure[1]

TA-TMA has been reported to occur in up to 39% of patients undergoing allogeneic HSCT but remains under-recognized[2]

HSCT, hematopoietic stem cell transplantation; TA-TMA hematopoietic stem cell transplantation-associated thrombotic microangiopathy.

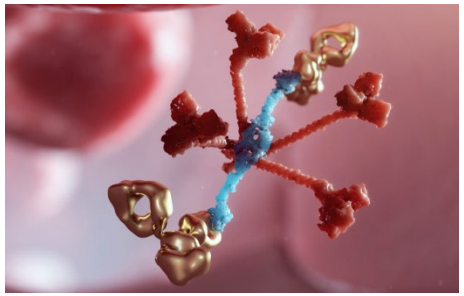
1. Carreras E et al. *Bone Marrow Transplantation*. 2011;46:1495-1502. 2. Jodele S et al. *Blood*. 2014;124:645-653.

FDA Approval Would Make Narsoplimab the First and Only Approved Treatment for TA-TMA



Unmet need

TA-TMA is deadly and underdiagnosed

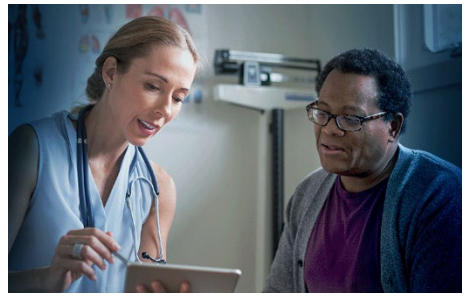


First and only

Currently no approved treatment for TA-TMA

Efficacy

Studied in TA-TMA patients with numerous risk factors associated with poor outcomes



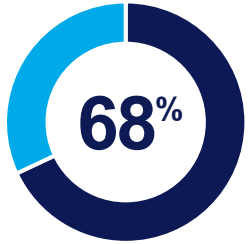
Safety

Well tolerated with no apparent safety concerns; no vaccinations required

Narsoplimab led to impressive survival in clinical trials and in the real world

NARSOPLIMAB PIVOTAL TRIAL (FAS) [1]

Median overall survival

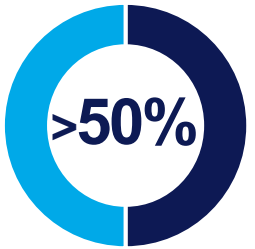


100-day survival post-TA-TMA diagnosis

NARSOPLIMAB TREATMENT IN EXPANDED ACCESS PROGRAM

Adult & pediatric TA-TMA patients

Of the ~40% who previously failed or stopped other unapproved agents*



Achieved 1-year survival from date of TMA diagnosis

FAS, full analysis set; OS, overall survival; TMA, thrombotic Microangiopathy;
* eculizumab, ravulizumab, defibrotide and/or pegcetacoplan

1. Khaled SK et al. *J Clin Oncol*. 2022;40(22):2447-2457; n=28

Safety and Tolerability

- Narsoplimab was well tolerated
  - No safety signals of concern observed
- Reported adverse events were consistent with those typically seen in immuno-suppressed post-transplant population
- 6 patients died during the core study period due to causes common in HSCT

Most Common TEAE Occurring in > 20% of Patients[a] (N=28)	
Event	**Number (%)**
Pyrexia	10 (36)
Diarrhea	9 (32)
Vomiting	9 (32)
Nausea	7 (25)
Neutropenia	7 (25)
Fatigue	6 (21)
Hypokalemia	6 (21)
Deaths[b]	
Septic shock	1
Progressive AML	2
Neutropenic sepsis	2
GVHD and TMA	1

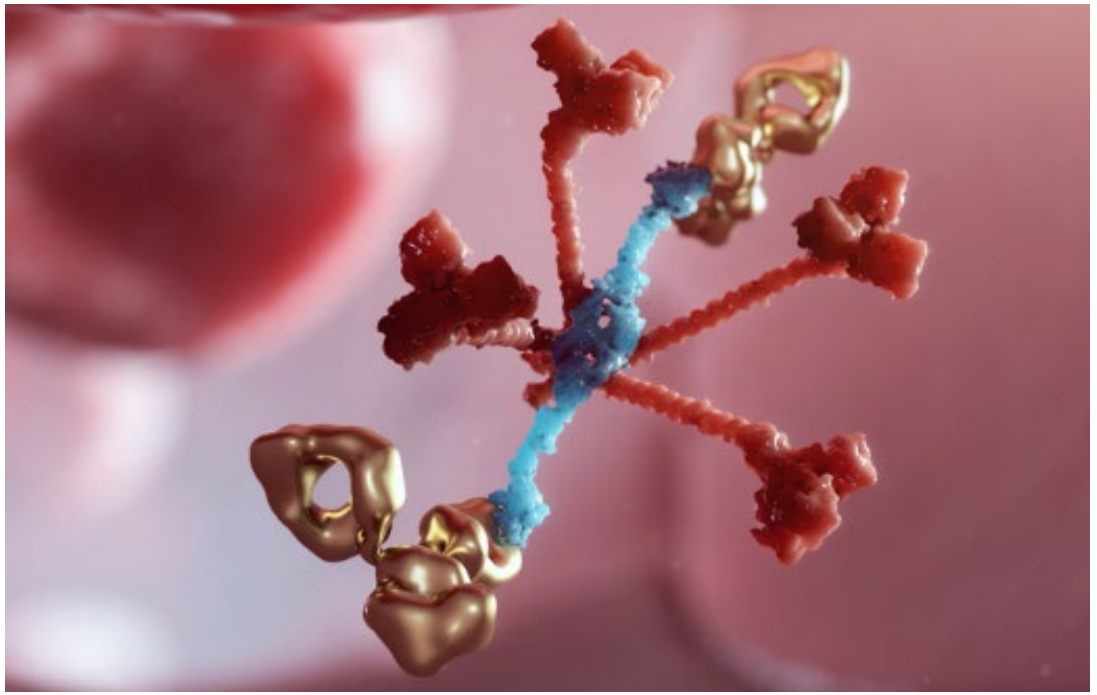
[a]Treatment-emergent adverse events during the safety evaluation period (defined as duration from informed consent to 37 days after last narsoplimab dose);
[b]Two of the 6 deaths occurred after the safety evaluation period but within the core study period (defined as from study screening visit to last scheduled follow-up visit)

1. Khaled SK et al. *J Clin Oncol*. 2022;40(22):2447-2457. 2. Omeros data on file

Narsoplimab in TA-TMA: Regulatory Milestones and Status

BLA pending with FDA

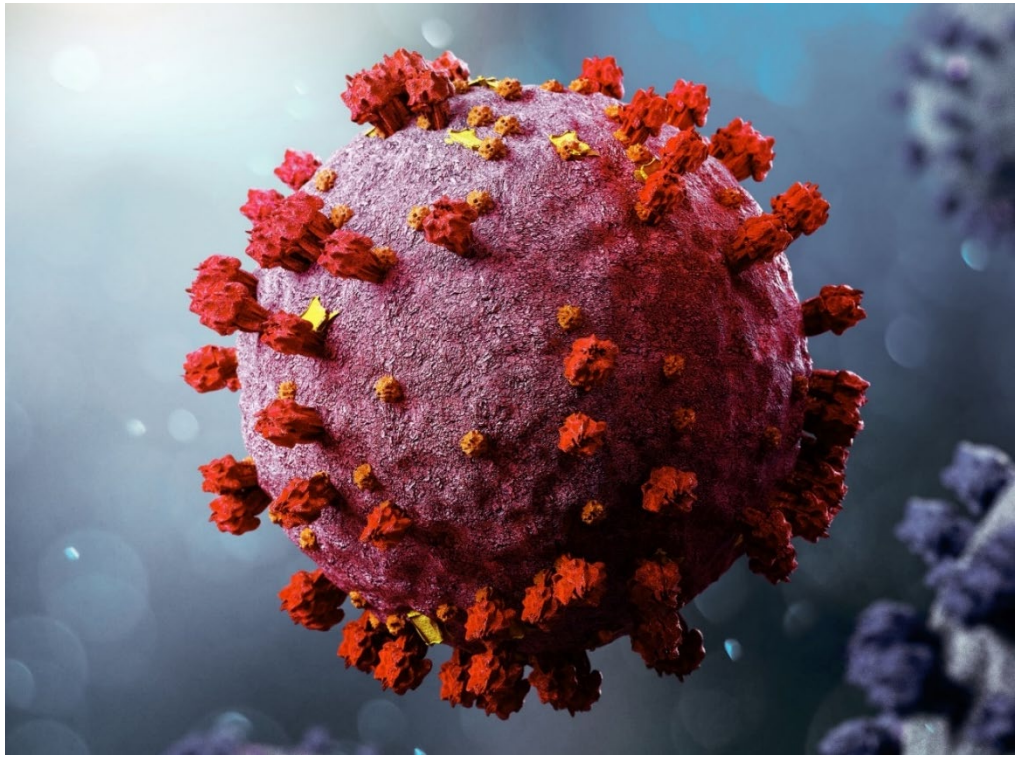
- November 2023 – Consistent with FDA interactions, submitted analysis plan to assess data from: existing clinical trial, external historical control population, narsoplimab expanded access program and narsoplimab MOA
- Ongoing FDA interactions regarding analysis plan
- Timeline to resubmission pending those discussions



Narsoplimab for COVID-19 and ARDS

Discussions regarding continued development of narsoplimab for COVID-19 and ARDS have progressed at the request of U.S. government agencies

- We continue to advance narsoplimab as a therapeutic with associated diagnostic tests for acute and long COVID-19 as well as ARDS
- Recent publications on lectin pathway inhibition in COVID-19 and ARDS listed on our website[1-5]
- Developed proprietary assay identifying levels of MASP-2/C1Inhibitor complexes to identity “high risk” COVID-19 and, more broadly, ARDS patients; appears to also be applicable to long COVID

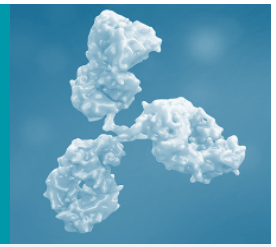


ARDS, acute respiratory distress syndrome

1. Ali YM et al. *Front Immunol*. 2022;13:841759. 2. Lynch NJ et al. *Clin Transl Med*. 2022;12(7):e980. 3. Rambaldi A et al. *Immunobiology*. 2020;225(6):152001.
4. Ali YM et al. *J Infect Dis* (2024) 229:680-690. 5. Ali YM et al. *Front Immunol* (2023) 14:1192767. 6. Ali YM et al. *Front Immunol* (2021) 12:714511.

OMS1029 Progress, Timeline and Future Plans

OMS1029



- Next-generation antibody
- Expected similar efficacy, safety to narsoplimab
- Long-acting, quarterly SC or IV for chronic treatment

- ✓ Phase 1 SAD study completed
- ✓ Dosing completed in MAD study
  - ➢ Data expected next month

- Targeting indication selection Q3 2024

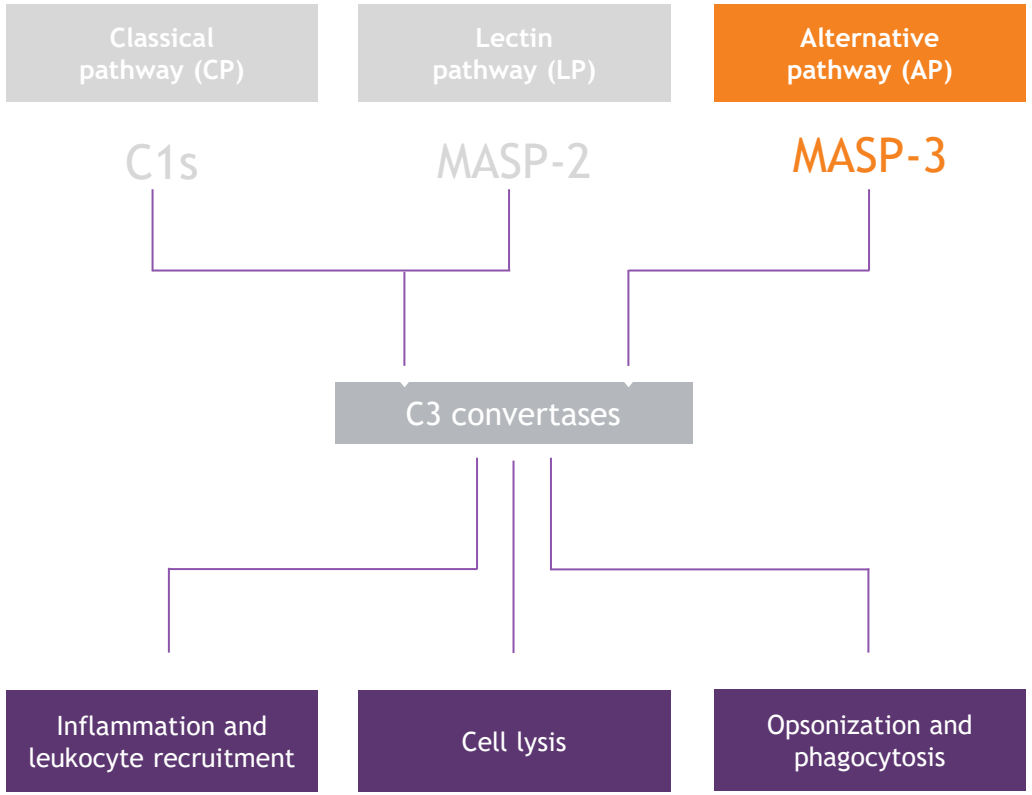
SC, subcutaneous; IV, intravenous; SAD, single-ascending dose; MAD, multiple-ascending dose

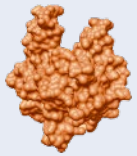
MASP-3 Inhibitor Program

Zaltenibart (OMS906)

MASP-3 is the Key Activator of the Alternative Pathway of Complement

Role of MASP-3 in the complement system





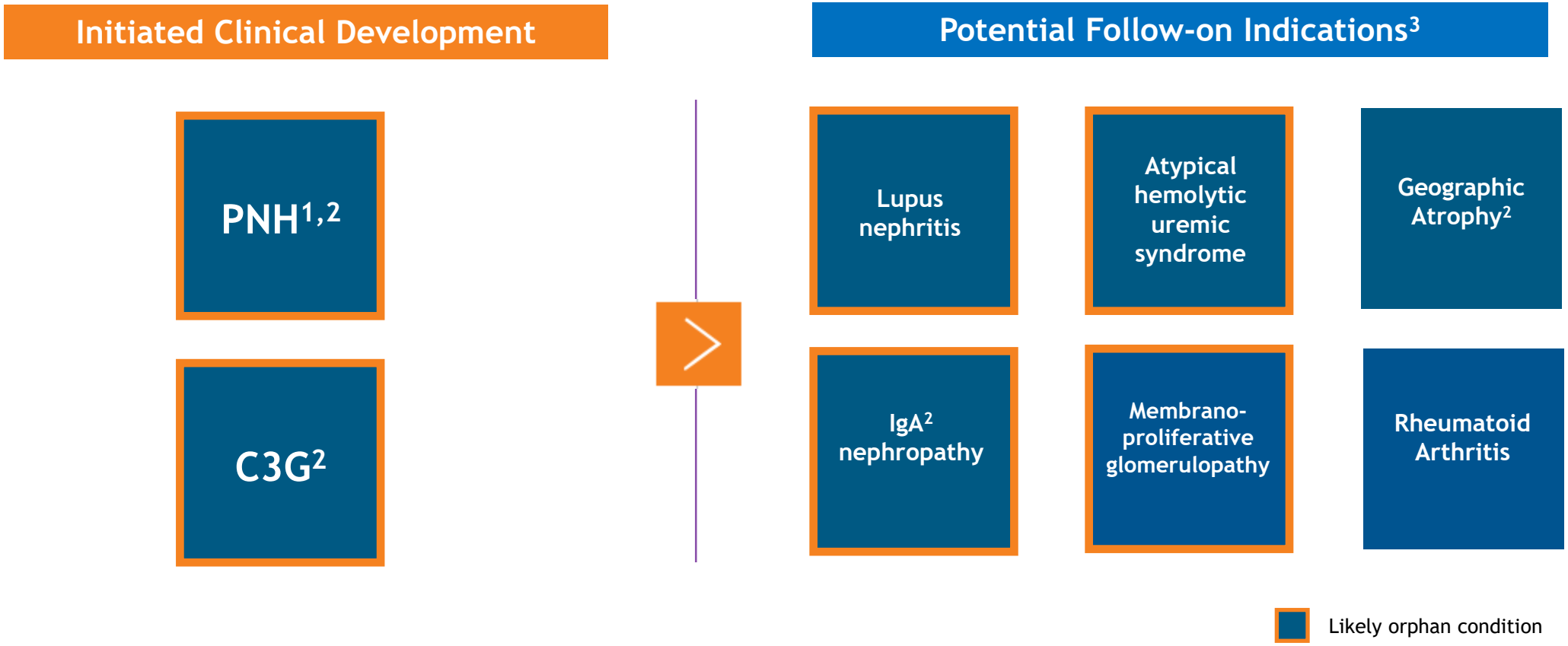
MASP-3
Key AP activator

- The AP amplifies the lectin and/or classical pathway's signaling cascade and its dysregulation is associated with a wide range of diseases (e.g., PNH, C3G, IgAN)
- The AP is also constitutively activated through C3 tick-over, causing disorders independent of lectin and classical pathways
- MASP-3 is the most upstream activator of the AP

Notes: PNH, paroxysmal nocturnal hemoglobinuria; C3G, complement 3 glomerulopathy; IgAN, IgA nephropathy

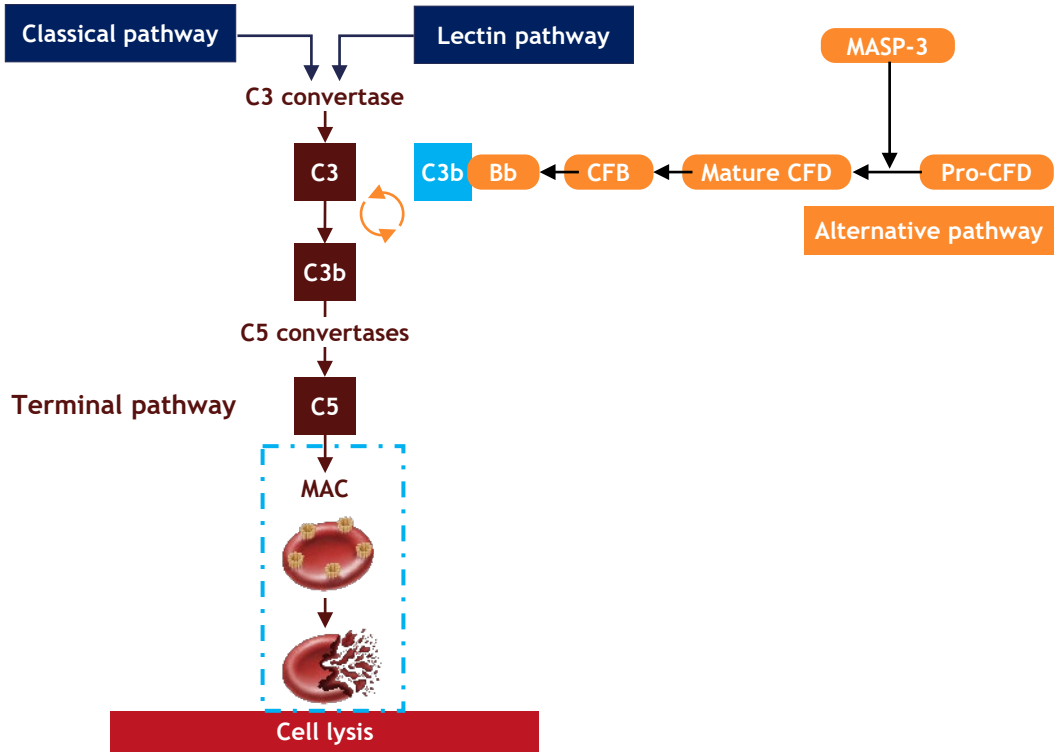
Source: Dunkelberger JR et al. *Nat. Cell Biol.* 2010

Zaltenibart (OMS906) Has a Validated and Efficient Development Pathway in PNH, C3G, and Beyond



Notes: [1]Zaltenibart has received orphan drug designation for paroxysmal nocturnal hemoglobinuria (PNH) from FDA; [2]Clinically validated in Phase 3 trials [3]Not exhaustive

The Alternative Pathway (AP) in the Complement System



- Complement injury to cells is mediated by C3b and the MAC
- The AP is comprised of a series of sequentially activated proteins
- Activation of these proteins then activates the production of C3b and the MAC
- MASP-3 is the most proximal activator of the AP, and responsible for activation of downstream proteins
- Inhibiting MASP-3 blocks formation of C3b and the MAC

Figure adapted from Belcher *et al* (2022)[6]

CFB, complement Factor B; CFD, complement Factor D; MAC, membrane attack complex; MASP-3, mannan-binding lectin-associated serine protease-3; PNH, paroxysmal nocturnal hemoglobinuria; RBC, red blood cell.

1. Risitano AM et al. *Front Immunol* 2019;10:1157. **2.** Notaro R et al. *N Engl J Med* 2022;387:160-6. **3.** Risitano AM et al. *Immunol Rev* 2023;313:262-78. **4.** Loschi M et al. *Am J Hematol* 2016;91:366-70. **5.** Fattizzo B et al. *J Blood Med* 2022;13:327-35. **6.** Belcher JD et al. *Transl Res* 2022;249:1-12.

Targeting MASP-3 has Advantages Over Other Clinically Validated Complement Targets

By targeting MASP-3, zaltenibart is designed to provide…

	Rationale	Alternative Pathway: MASP-3	Alternative Pathway: Factor D	Alternative Pathway: Factor B	Terminal Pathway: C3	Terminal Pathway: C5a	Terminal Pathway: C5
BETTER disease control	**Favorable resting target properties** MASP-3 has lower plasma concentrations and slower turnover, allowing a longer/more consistent dosing profile — resulting in lower risk of PK/PD breakthrough	✓					
	Favorable target dynamics Based on available data, MASP-3 is not an acute phase reactant, which is important for chronic disease treatment in the setting of inflammation (e.g., infection)	✓	✓				
LOWER RISK of adverse events	**Preserves complement's lytic function** Classical, lectin, and terminal pathway functions retained to mount appropriate response to infection	✓	✓	✓		✓	
BETTER adherence	**Enhanced convenience** The dosing frequency should improve patient adherence	✓					✓*

Note: *Ravulizumab (Q8W regimen), but not eculizumab (Q2W regimen), has convenient dosing

Source: Dobo, J. et. al. *Front. Immunol.* 2018; Pascual, M. et al. *KI.* 1988; Bereman, M. et al. *Anal. Bioanal. Chem.* 2012; Ekdahl, K. et al. *Mol. Immunol.* 2019; Sissons JG, Liebowitch J, Amos N, Peters DK. Metabolism of the fifth component of complement, and its relation to metabolism of the third component, in patients with complement activation. *J Clin Invest.* 1977; Bokisch, V. et al. *Proc. Natl. Acad. Sci. USA.* 1975; Skjoedt, M. et al. *Immunobiology* 2010; Barratt, J. et al. *Front. Immunol.* 2021; Schnabolk, G. et al. *Investig. Ophthalmol.* Vis. Sci. 2015; Andoh, A. et al. *J. Clin. Immunol.* 1997; Data on file.

Zaltenibart (OMS906)

Best-in-Class, Differentiated Potential

Zaltenibart has a Well-Differentiated Profile for Multiple Alternative Pathway-Associated Indications

Premier AP Target - MASP-3

Novel target with favorable properties — the key activator of the AP
Preserves complement's lytic function
- Classical, lectin, and terminal pathway functions retained to mount appropriate response to infection

Compelling Clinical Profile

Compelling efficacy and safety profile demonstrated to date in ongoing Phase 2 trials

Low Treatment Burden

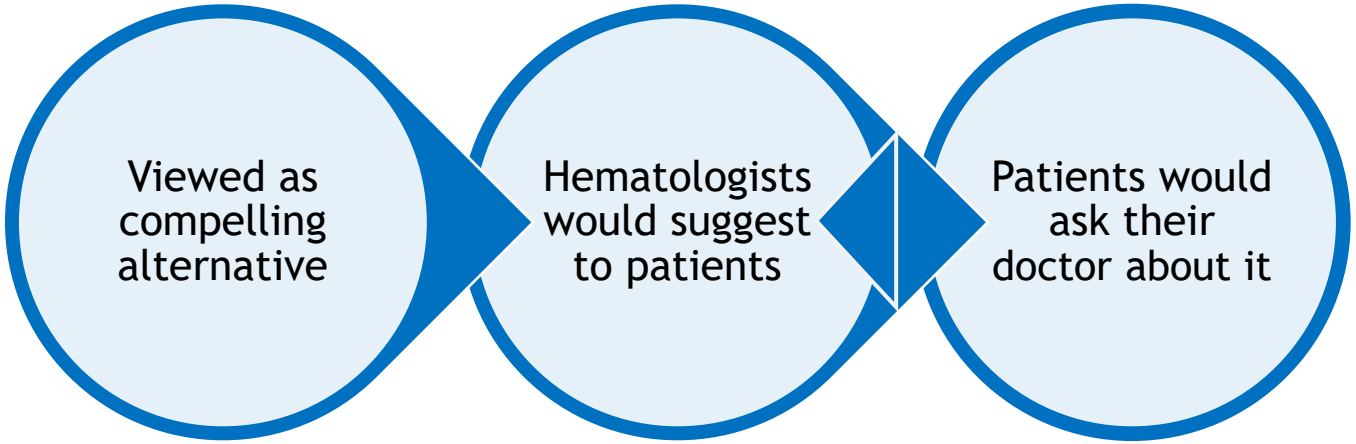
Convenient, long acting IV or SC up to once quarterly for sustained efficacy*
- ideal for patient adherence without multiple pills per day

Positive clinical data from other AP inhibitors validates and de-risks multiple potential clinical programs

Notes: AP, alternative pathway; SC, subcutaneous; *Short half-life agents could result in meaningful breakthrough disease

Hematologist and PNH Patient Preference

Recent market research confirms that zaltenibart target profile is differentiated from evolving PNH landscape



Preference Drivers

- Compelling efficacy and safety profile with low treatment burden
- Dosing 4 - 6 times per year minimizes how often patients have to think about their diagnosis
  - Dosing is more in line with usual frequency of HCP visits
- Infrequent IV minimizes risk of non-compliance and subsequent risk of breakthrough disease

> *"[Efficacy] for a monotherapy is great, plus no tablets to take 2-3 times per day"- Community* Hematologist

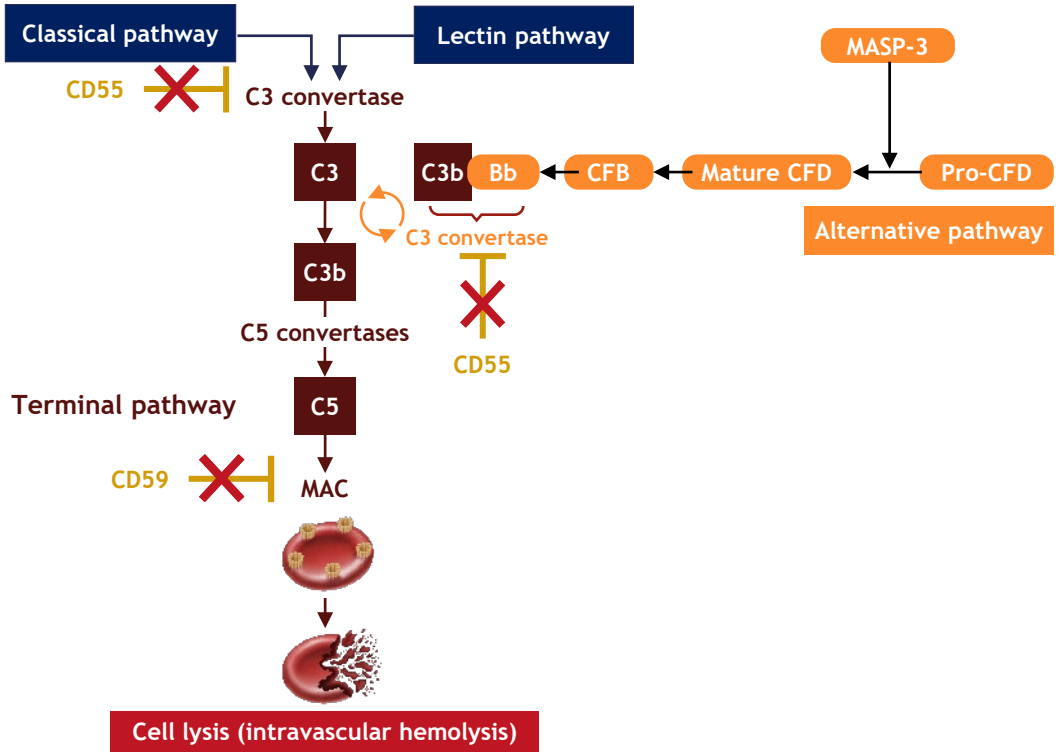
> *"It's promising. I think it will directly compete with IPTA especially if it's easy to administer"*
> *- Academic* Hematologist

Data on File

Zaltenibart (OMS906)

Clinical Validation in PNH

Clinical Presentation of PNH is Driven by Intravascular Hemolysis Due to Dysregulation of the Complement System



- PNH is a rare and life-threatening disorder[1-3]
- It is characterized by hemolytic anemia resulting from the absence of surface proteins CD55 and CD59, which regulate complement-mediated RBC lysis[1-3]
- Left untreated, PNH results in debilitating anemia, thrombosis, fatigue, and increased mortality[3-5]

Figure adapted from Belcher *et al* (2022)[6]

CFB, complement Factor B; CFD, complement Factor D; MAC, membrane attack complex; MASP-3, mannan-binding lectin-associated serine protease-3; PNH, paroxysmal nocturnal hemoglobinuria; RBC, red blood cell.

1. Risitano AM et al. *Front Immunol* 2019;10:1157. **2.** Notaro R et al. *N Engl J Med* 2022;387:160-6. **3.** Risitano AM et al. *Immunol Rev* 2023;313:262-78. **4.** Loschi M et al. *Am J Hematol* 2016;91:366-70. **5.** Fattizzo B et al. *J Blood Med* 2022;13:327-35. **6.** Belcher JD et al. *Transl Res* 2022;249:1-12.

Terminal Complement Blockade in PNH Inhibits Intravascular Hemolysis but Inevitably Leads to Extravascular Hemolysis[1,2]

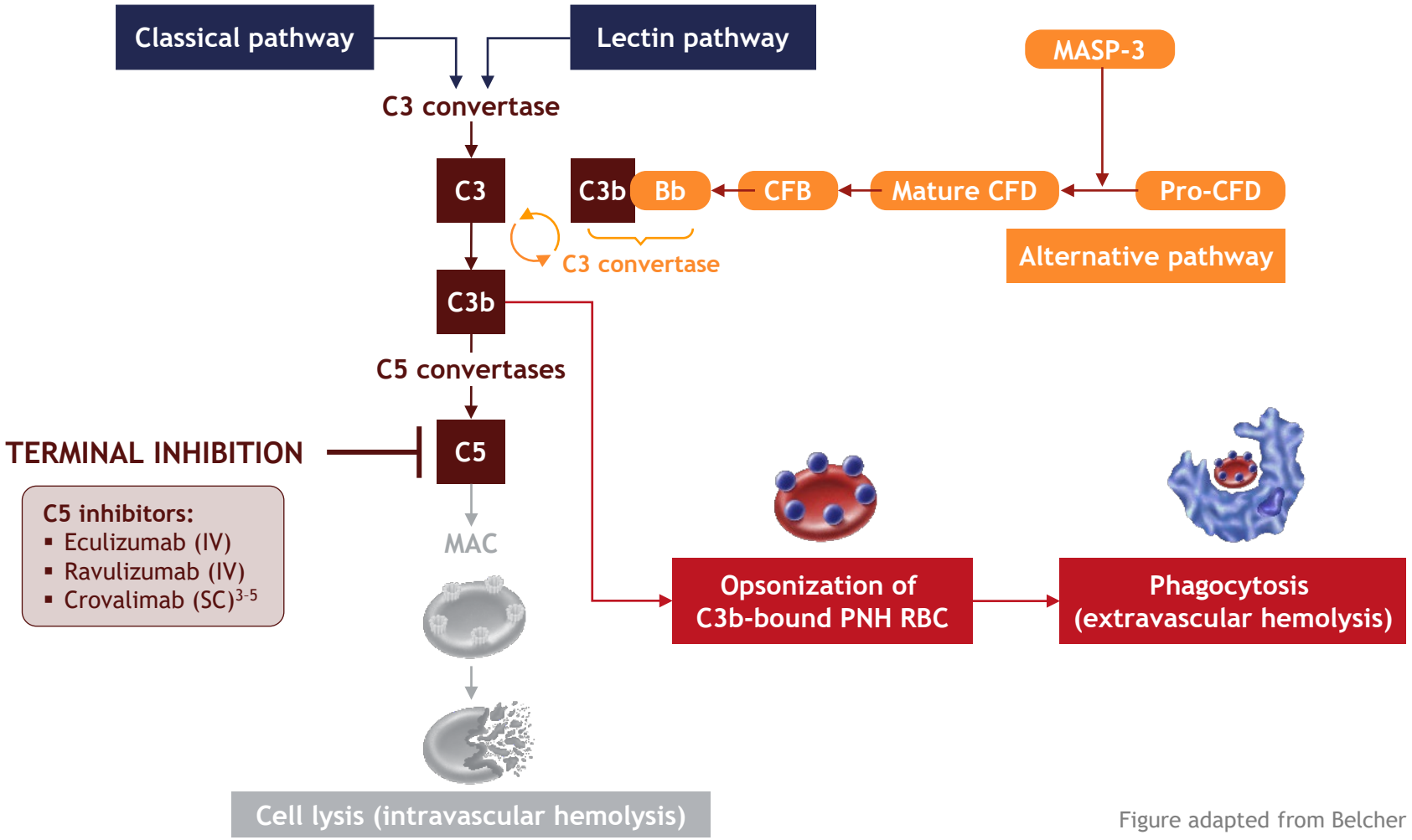


Figure adapted from Belcher *et al* (2022)[6]

CFB, complement Factor B; CFD, complement Factor D; MAC, membrane attack complex; MASP-3, mannan-binding lectin-associated serine protease-3; PNH, paroxysmal nocturnal hemoglobinuria; RBC, red blood cell.

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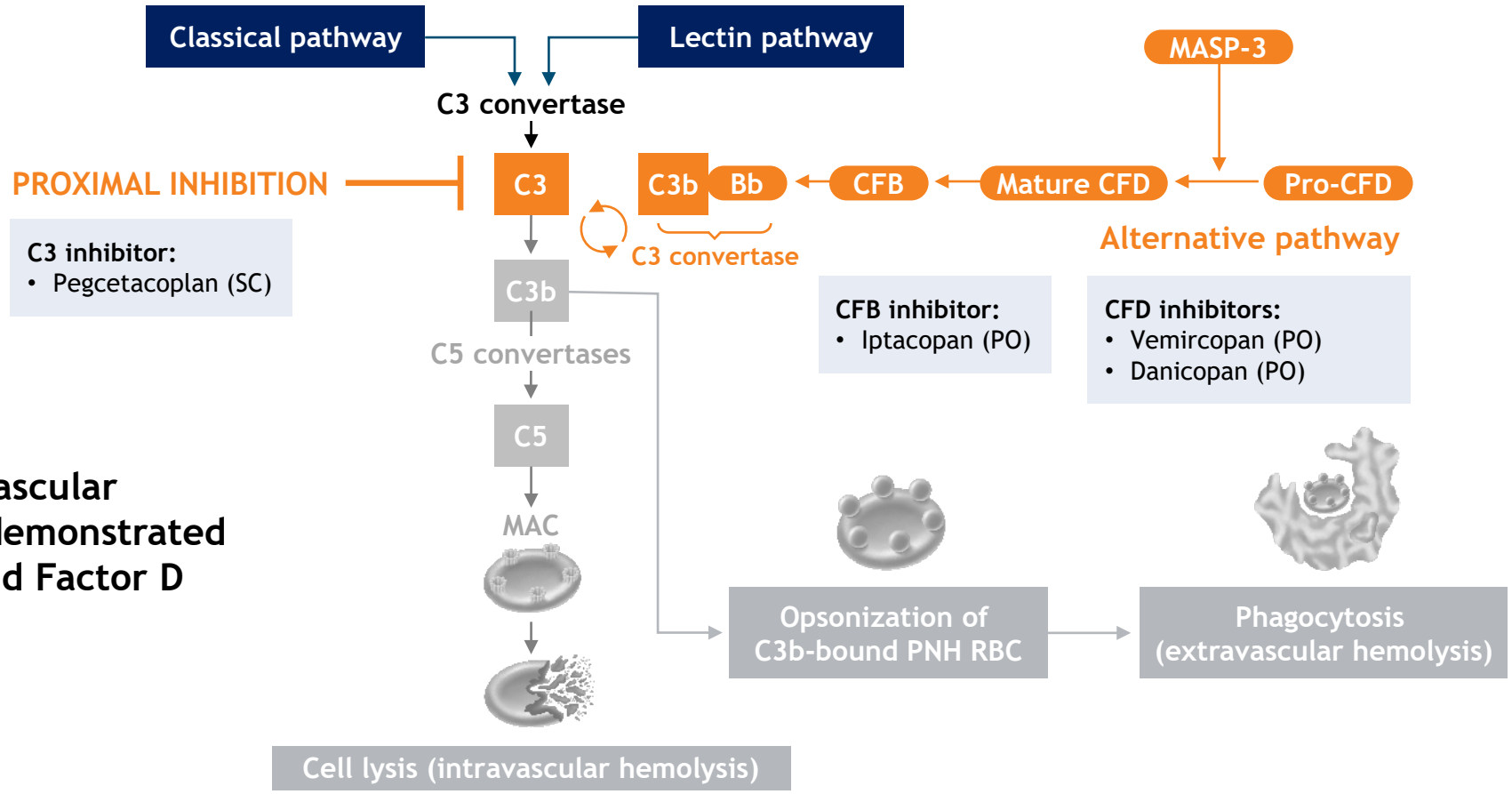
Proximal Inhibition of the Alternative Pathway Blocks Intravascular Hemolysis and Prevents Extravascular Hemolysis[1,2]



Prevention of extravascular hemolysis has been demonstrated with C3, Factor B, and Factor D inhibitors[1-6]

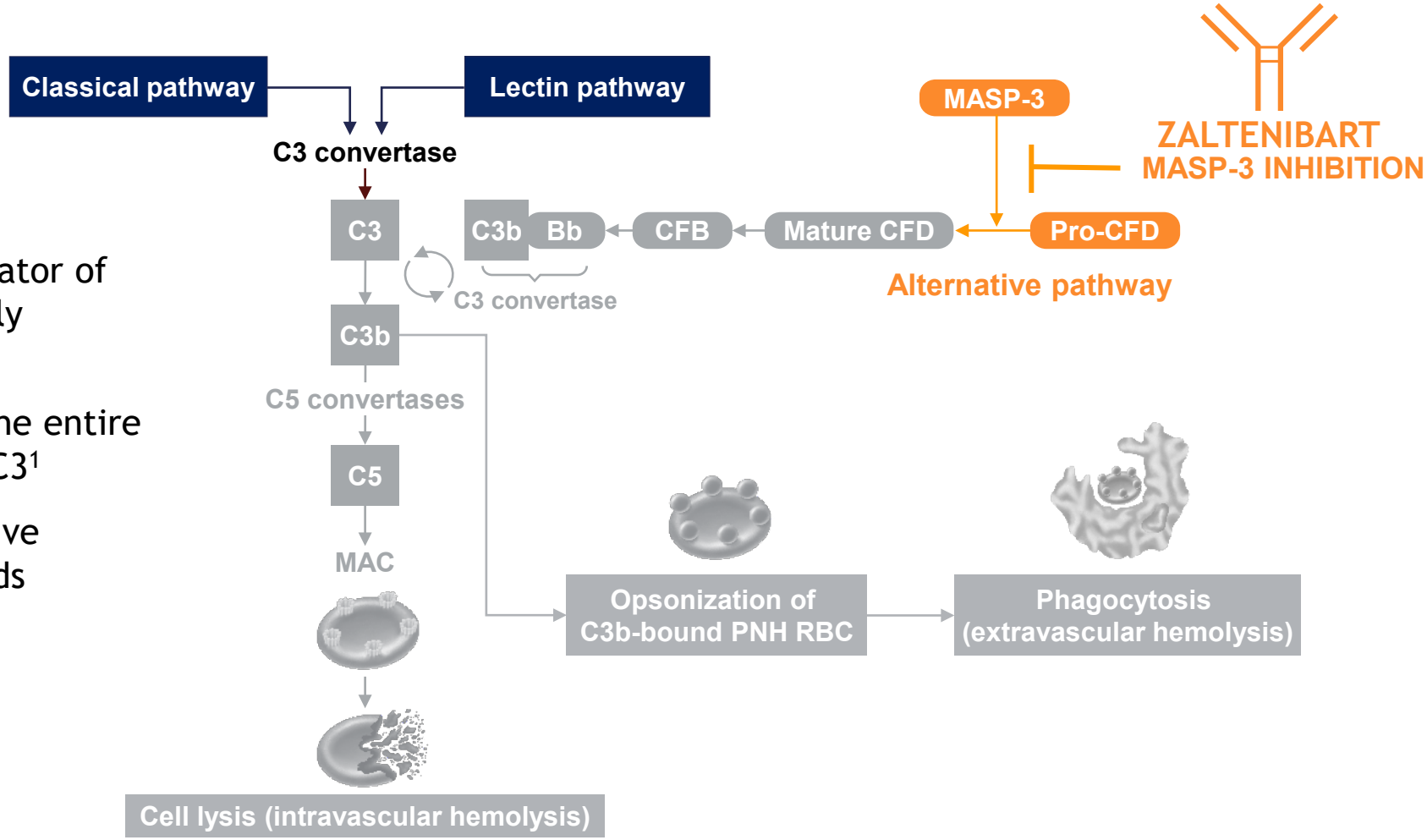
CFB, complement Factor B; CFD, complement Factor D; MAC, membrane attack complex; MASP-3, mannan-binding lectin-associated serine protease-3; PNH, paroxysmal nocturnal hemoglobinuria; PO, orally; RBC, red blood cell; SC, subcutaneous.

1. Notaro R et al. *N Engl J Med*. 2022;387:160-6. **2.** Risitano AM et al. *Immunol Rev*. 2023;313:262-78. **3.** Peffault de Latour R et al. *Blood*. 2022;140(S2):LBA-2. **4.** Risitano AM et al. 49th Annual Meeting of the EBMT 2023:OS12-06. **5.** Risitano AM et al. *HemaSphere*. 2023;7(S3):S182. **6.** Peffault de Latour R et al. *HemaSphere*. 2023;7(S3):P774. **7.** Belcher JD et al. *Transl Res*. 2022;249:1-12.

Figure adapted from Belcher et al.

MASP-3 is a Key Activator of the Alternative Pathway and a Novel Target for Treatment of PNH[1,2]

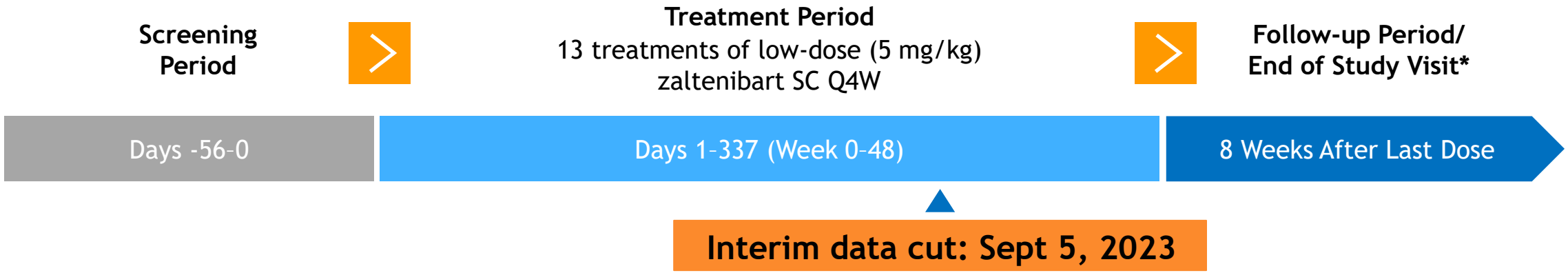
- MASP-3 is the exclusive activator of pro-CFD, thereby continuously generating mature CFD[1]
- Inhibition of MASP-3 blocks the entire AP, including CFD, CFB, and C3[1]
- Zaltenibart is a highly selective humanized IgG mAb that binds to and inhibits MASP-3[3,4]



CFB, complement Factor B; CFD, complement Factor D; IV, intravenous; MAC, membrane attack complex; MASP, mannan-binding lectin-associated serine protease; PNH, paroxysmal nocturnal hemoglobinuria; RBC, red blood cell; SC, subcutaneous.

1. Sekine H et al. *Immunol Rev*. 2023;313:15-24. **2.** Barratt J et al. *Front Immunol*. 2021;12:712572. **3.** Cummings WJ et al. *Mol Immunol* 2022;150:145. **4.** Griffin M et al. *HemaSphere* 2023;7(S3):P787. Figure adapted from Belcher et al. (2022)

OMS906-PNH-002: Phase 2 Trial in Treatment-Naïve PNH Patients – Study Design



Key Inclusion Criteria:

- Adults with confirmed PNH diagnosis by flow cytometry (clone size >10%)
- Complement inhibitor treatment-naïve†
- Hgb level of <10.5 g/dL
- LDH >1.5× ULN‡
- *Neisseria meningitidis* vaccination

Primary Endpoint:

- Safety and tolerability

Key Secondary Endpoints:

- Preliminary efficacy by effect on hemolysis and anemia:
  - Change from baseline in Hgb and LDH levels, absolute reticulocyte count, RBC transfusion burden
  - Proportion of patients achieving Hgb ≥12 g/dL or increase in Hgb ≥2 g/dL

Presented at the 65th ASH Annual Meeting and Exposition | December 9–12, 2023 | San Diego, USA

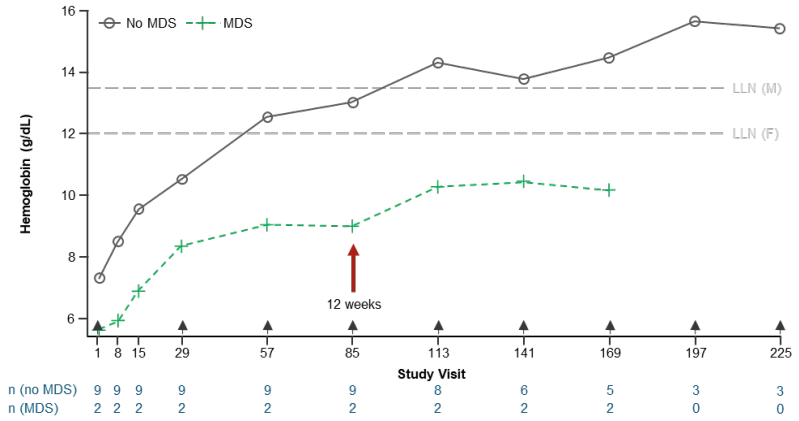
*The diagram represents the study design at the time of the interim analysis. The design has been revised to determine dose levels required to provide 8- and 12-week protection from breakthrough hemolysis

†Patients treated with any complement pathway inhibitor within 6 months prior to screening were excluded.

Hgb, hemoglobin; LDH, lactate dehydrogenase; Q4W, every 4 weeks; RBC, red blood cell; SC, subcutaneous; ULN, upper limit of normal.

OMS906-PNH-002: Treatment with Low-Dose Zaltenibart Rapidly Improved Hemoglobin and LDH Levels

Mean Hemoglobin Levels



At 12 weeks, mean increase in Hgb was 5.3 g/dL from baseline (*P*<0.001)

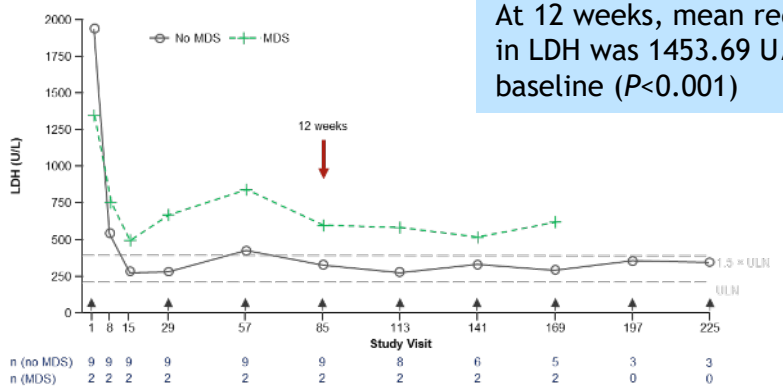
- ↑5.7 g/dL (patients without MDS)
- ↑3.4 g/dL (patients with MDS)

11/11 patients had an increase in **Hgb ≥2 g/dL**

9/9 patients without MDS achieved **Hgb ≥12 g/dL**

- Patients without MDS had more rapid improvement in hemoglobin levels after treatment with low-dose zaltenibart
- No patients required transfusions following initiation of zaltenibart

Mean LDH Levels



At 12 weeks, mean reduction in LDH was 1453.69 U/L from baseline (*P*<0.001)

- PK and PD from these patients will help inform planned dose escalation to achieve once-quarterly dosing

Presented at the 65th ASH Annual Meeting and Exposition, December 9-12, 2023, San Diego, USA. Data shown are from interim data cut as of 5 September 2023. *P* values are for testing change from baseline using t-test; *P* values may not be valid for small N. Black arrows indicate OMS906 administration following laboratory marker collection.
F, female; Hgb, hemoglobin; LLN, lower limit of normal; M, male; MDS, myelodysplastic syndrome.

OMS906-PNH-002: Zaltenibart Treatment Was Well Tolerated in Patients with PNH

Adverse Events in ≥20% of Patients	n (%)	CTCAE Grade
Itching	3 (27)	All Grade 1
Increased thrombocytopenia	4 (36)	1 patient Grade 1* 2 patients Grade 2* 1 patient Grade 3†
Transient neutropenia	3 (27)	3 patients Grade 3

- All patients with reported cytopenia had evidence of underlying bone marrow failure
- No clinical breakthrough hemolysis
- No major adverse events
- No treatment-related SAEs, discontinuations, or deaths

Presented at the 65th ASH Annual Meeting and Exposition, December 9–12, 2023, San Diego, USA
Data shown are from interim data cut as of 5 September 2023. *Received iron therapy; †Had pre-existing Grade 3 thrombocytopenia and myelodysplastic syndrome.
CTCAE, Common Terminology Criteria for Adverse Events; PNH, paroxysmal nocturnal hemoglobinuria; SAE, serious adverse event.

OMS906-PNH-001: Phase 2 “C5 Switch-over” Trial is Fully Enrolled

- Phase 2 study evaluating zaltenibart (OMS906) in patients who have had an unsatisfactory response to ravulizumab (C5 inhibitor)
  - Patients are receiving ravulizumab at time of enrollment
  - Zaltenibart is added for combination therapy with ravulizumab for 24 weeks
  - Patients demonstrating hemoglobin response continue on zaltenibart monotherapy

- At the European Hematology Association congress, a pre-specified interim analysis of the combination therapy portion is being presented on June 15th, 2024
  - Rapid and sustained response to zaltenibart
    - Significant improvements in mean hemoglobin and absolute reticulocyte counts by Week 4 of combination therapy
    - Extravascular hemolysis prevented in patients who had experienced significant extravascular hemolysis on ravulizumab alone
  - Well tolerated with a good safety profile

Zaltenibart PNH Summary

- Zaltenibart demonstrated clinically meaningful efficacy in patients with PNH
- Zaltenibart was safe and well tolerated
- PNH Phase 3 initiation is planned for this year including dosing regimens of intravenous treatment
  - Initially, dosing every 8 weeks with a plan of extending to every 12 weeks
  - Speed to market and sustained efficacy are among key decision drivers of dose

Zaltenibart (OMS906)

C3 Glomerulopathy (C3G)

C3 Glomerulopathy (C3G)

- Rare kidney disease resulting from glomerular inflammation[1]
  - Caused by dysregulation of the complement alternative pathway
  - ~30–50% of C3G patients progress to end-stage renal disease within 10 years of diagnosis
- Currently no approved therapies targeting the underlying cause of the disease
- Positive clinical data from other alternative pathway inhibitors validates and de-risks C3G clinical program
- Zaltenibart: Phase 2 study in C3G is underway and dosing initiated
- A Phase 3 program is targeted to begin in early 2025

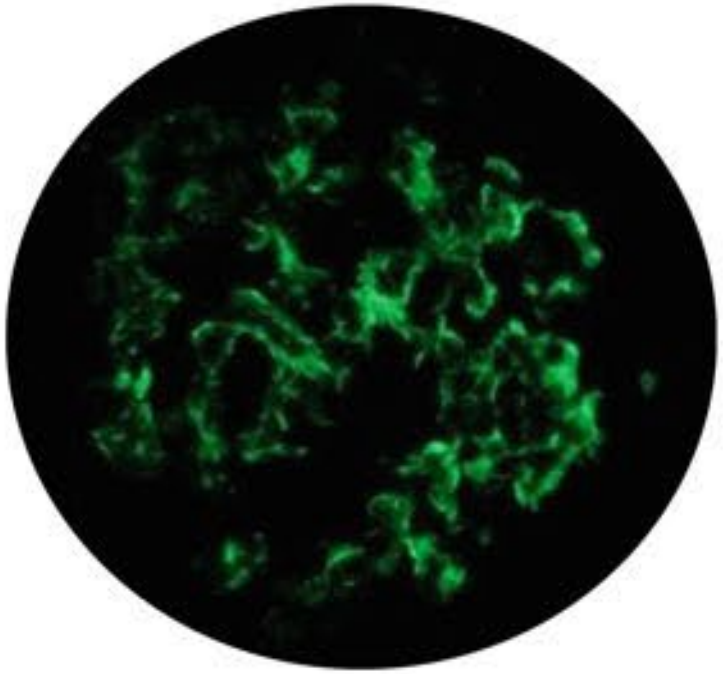


Figure adapted from Heidersheit *et al* (2022)[1]

1. Heiderscheit AK et al. *Am J Med Genet*. 2022;190C:344–357.

Zaltenibart has a Well-Differentiated Profile for Multiple Alternative Pathway-Associated Indications

Premier AP Target - MASP-3

Novel target with favorable properties — the key activator of the AP
Preserves complement's lytic function
- Classical, lectin, and terminal pathway functions retained to mount appropriate response to infection

Compelling Clinical Profile

Compelling efficacy and safety profile demonstrated to date in ongoing Phase 2 trials

Low Treatment Burden

Convenient, long acting IV or SC up to once quarterly for sustained efficacy*
- ideal for patient adherence without multiple pills per day

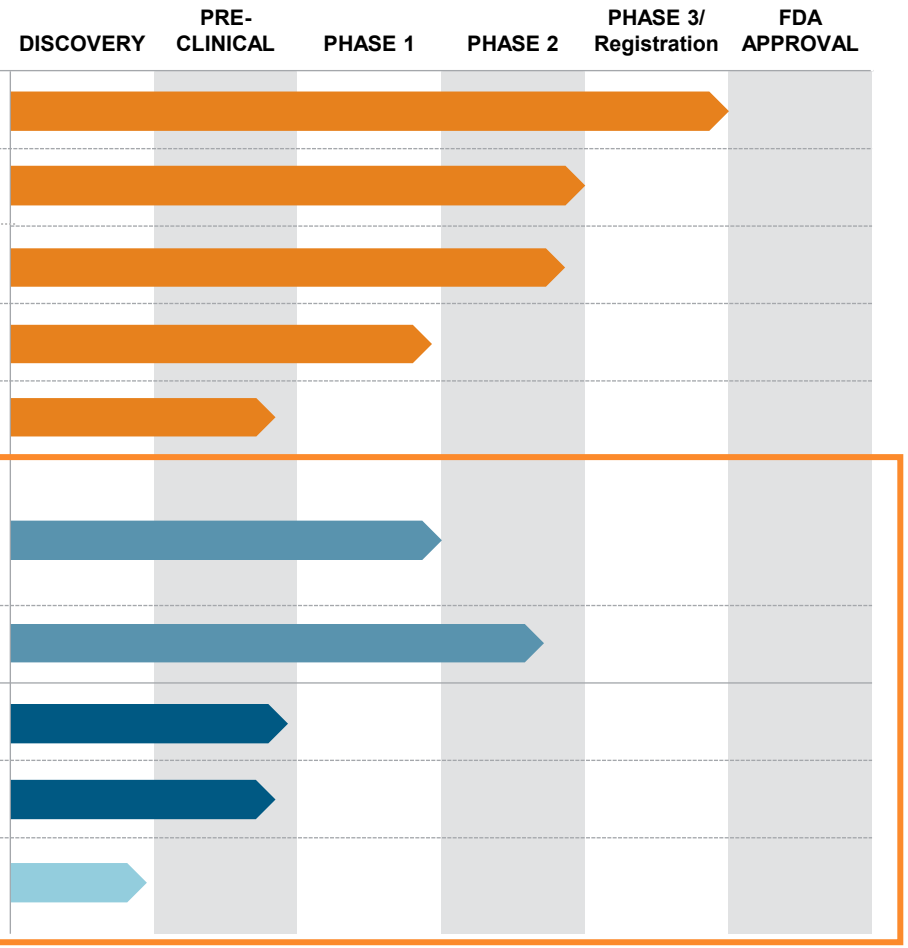
Positive clinical data from other AP inhibitors validates and de-risks multiple potential clinical programs

Notes: AP, alternative pathway; SC, subcutaneous; *Short half-life agents could result in meaningful breakthrough disease

PDE7 Inhibitor and Immuno-oncology Programs

Pipeline of Novel Targets and Programs: Each Proprietary to Omeros

	PROGRAM / (CANDIDATE)	MOLECULE	TARGETED DISEASE
COMPLEMENT FRANCHISE	MASP-2, Lectin Pathway (narsoplimab [0MS721])*	Ab	Stem Cell Transplant-Associated TMA
			COVID-19 and ARDS
	MASP-3, Alternative Pathway (zaltenibart [OMS906])*	Ab	PNH, C3 Glomerulopathy, and Other Alternative Pathway Disorders
	MASP-2 (OMS1029)*	Ab	Long-Acting 2nd Generation Antibody Targeting Lectin Pathway Disorders
	MASP-2, MASP-3, MASP-2/3*	SM	Disorders of the Lectin and Alternative Pathways of Complement
SUBSTANCE ABUSE DISORDERS AND COMPULSIONS	PDE7 (OMS527)*	SM	Substance Abuse and Compulsive Disorders; Movement Disorders
	PPARγ (OMS405)	SM	Opioid and Nicotine Addiction
IMMUNO-ONCOLOGY	Cellular Therapies (adoptive T cell and CAR T)	SM/LM	Cancer
	Biologic Therapeutics (immunomodulator, oncotoxins, cancer vaccines)	SM/LM	Cancer
DISCOVERY PLATFORM TECHNOLOGIES	GPCR Platform	SM	Immunologic, CNS, Metabolic, CV, Musculoskeletal and Other Disorders



Notes: Ab, antibody; ARDS, acute respiratory distress syndrome; CNS, central nervous system; COVID-19, coronavirus disease 2019; CV, cardiovascular; FDA, Food and Drug Administration; GPCR, G protein-coupled receptors; LM, large molecule; MASP, mannan-binding lectin-associated serine protease; PDE7, phosphodiesterase 7; PNH, paroxysmal nocturnal hemoglobinuria; PPARγ, peroxisome proliferator-activated receptor gamma; SM, small molecule; TMA, thrombotic microangiopathy
*Program directed to inhibiting associated target

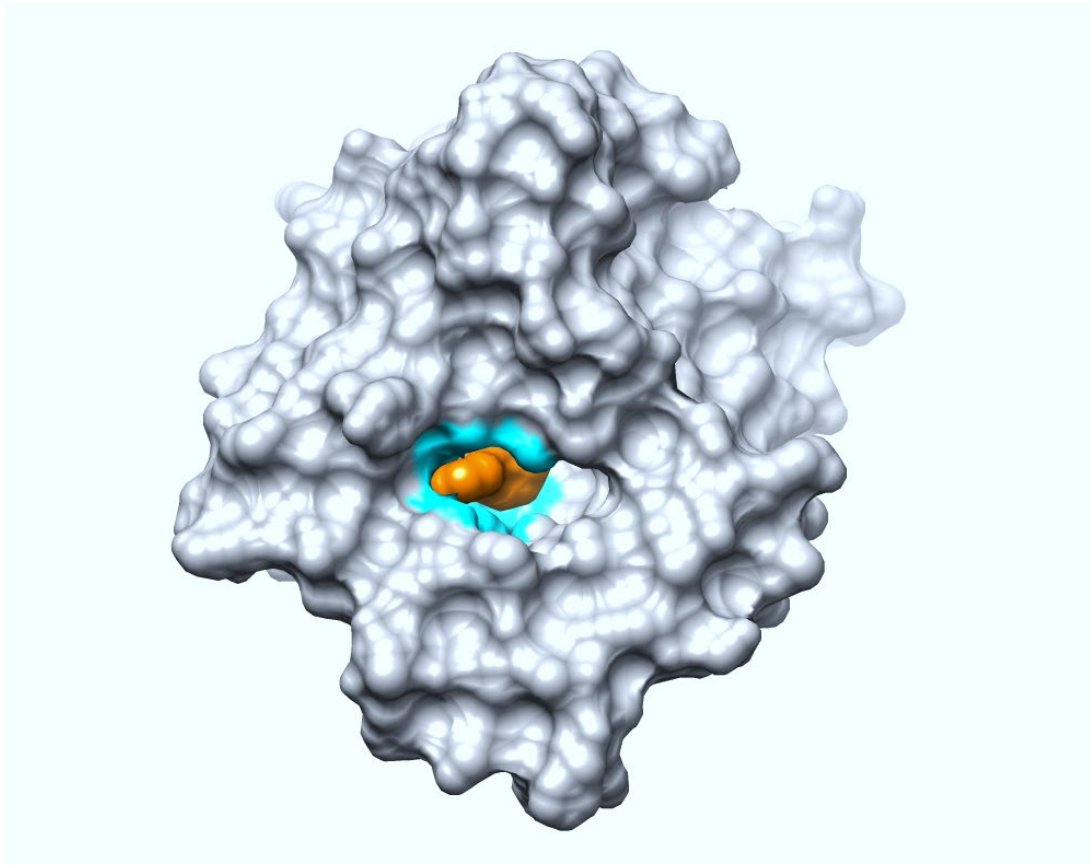
PDE7 Inhibitor – OMS527

Treatment of Substance Use Disorders, Compulsions and Movement Disorders

OMS527: PDE7 Inhibitor Program in Addictions and Compulsions

Our novel oral PDE7 inhibitor program is advancing with grant funding from the National Institute on Drug Abuse (NIDA)

- OMS527 is progressing as the first potential treatment for cocaine use disorder (CUD)
- Successfully completed Phase 1 clinical program
- $6.7 million, 3-year NIDA grant to also support*:
  - Toxicity studies in non-human primates when administered concomitantly with cocaine (results expected 2H 2024)
  - Randomized, double-blind, inpatient clinical trial comparing OMS527 safety and efficacy to placebo in CUD (study initiation planned 2025)



PDE7 Phosphodiesterase

*The described research is supported by the National Institute on Drug Abuse of the National Institutes of Health under award number U01DA058541.The content of this presentation is solely the responsibility of the authors and does not necessarily represent the official views of the National Institutes of Health.

OMS527: PDE7 Inhibitor Program in Movement Disorders

OMS527 as a potential treatment for L-Dopa-induced dyskinesia (LID)

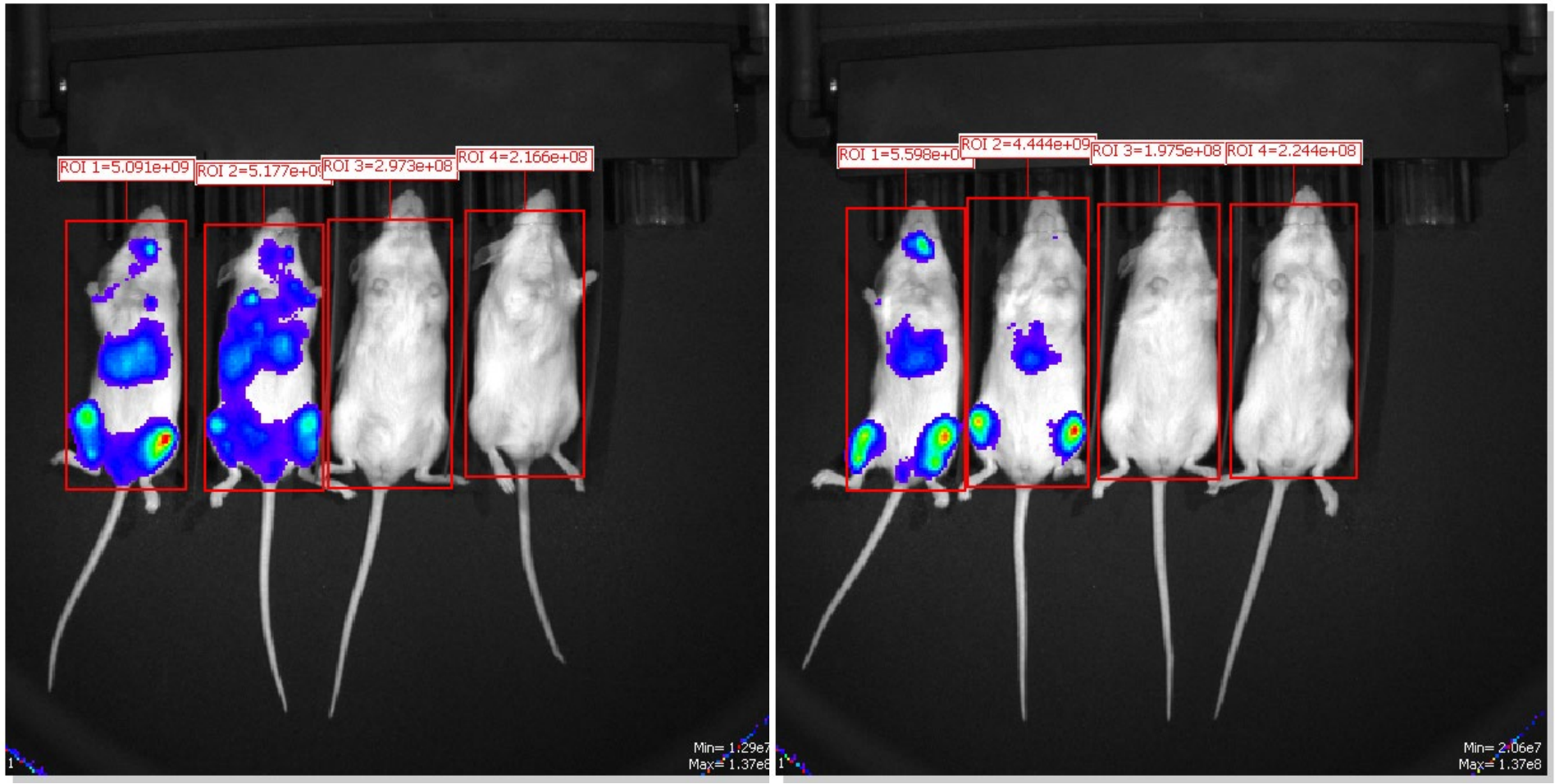
- L-Dopa is the most commonly prescribed treatment for Parkinson's disease; as PD progresses, prolonged treatment causes LID in majority of patients
- Unmet need for effective LID treatments represents substantial market opportunity in the >10 million Parkinson's patients worldwide
- Only approved non-surgical treatment for LID has limited efficacy and multiple side-effects
- OMS527 evaluated in primate model of LID at Emory University; exploring continued development with Emory collaborators

Shareholder Summary

Corporate Priorities Represent Catalysts of Near-/Mid-Term Value and Growth

One More Thing…

Immuno-oncology Franchise



Questions

*Shareholders may submit questions through the virtual meeting platform with the **16-digit control number** included on your proxy card or in the instructions that accompanied your proxy materials.*

Thank you

Annual Meeting of Shareholders

JUNE 6, 2024

OMEROS®