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Omeros Corporate Presentation

August 14, 2020

Safe Harbor

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Highly Diversified Pipeline to Drive Sustainable Growth

Omeros Controls All Economic Rights Across Its Programs and Platforms

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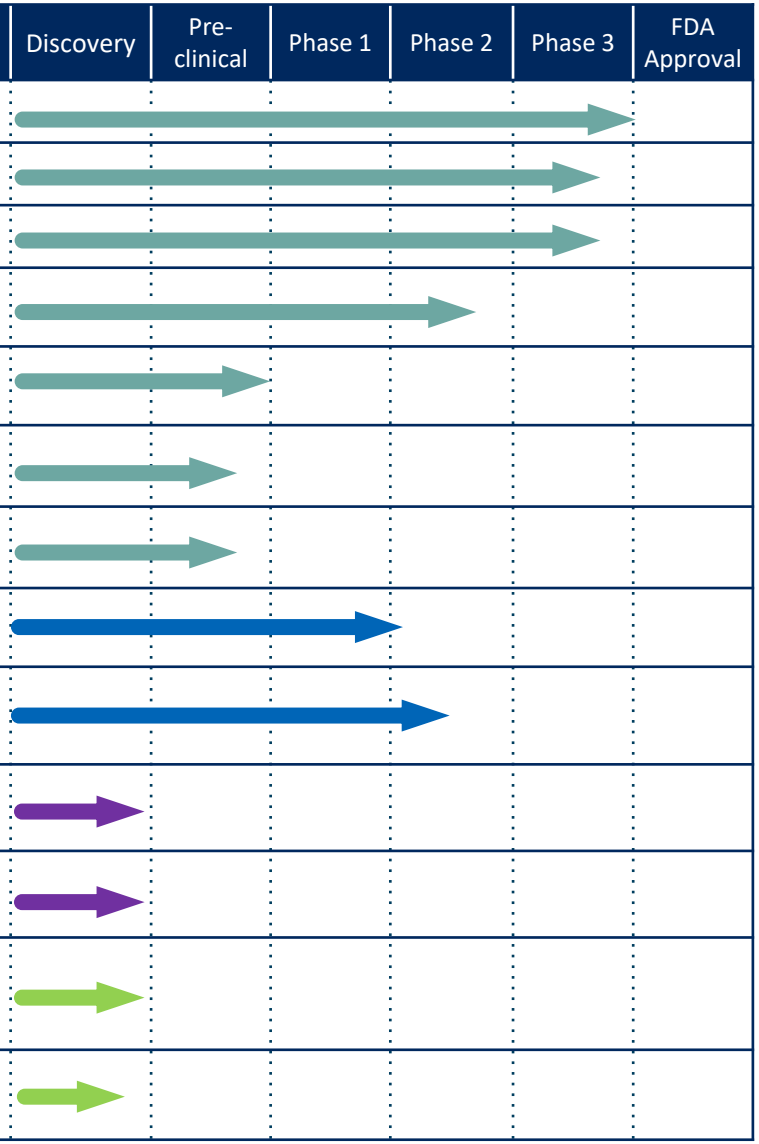
	Program / (Candidate)	Molecule	Targeted Disease
Complement Franchise (iCAB)	MASP-2, lectin pathway (narsoplimab (OMS721))	Ab	Stem Cell Transplant-Associated TMA
			IgA Nephropathy
			Atypical Hemolytic Uremic Syndrome
			Lupus Nephritis & Other Renal Diseases
	MASP-3, alternative pathway (OMS906)	Ab	PNH and a Wide Range of 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
Addiction	PDE7 (OMS527)	SM	Addictions and Compulsive Disorders; Movement Disorders
	PPARγ (OMS405)	SM	Opioid and Nicotine Addiction
Immuno-oncology	GPR174	SM	Cancer
	GPR161	SM	Cancer
Other	GPCR	SM	Immunologic, immuno-oncologic, CNS, Metabolic, CV, Musculoskeletal & Other Disorders
	Antibody	Ab	Metabolic, CV, Oncologic, Musculoskeletal & Other Disorders



Experienced Management with Deep Industry Experience

	Position	Background
Gregory Demopulos, MD	*Chairman, President & CEO*	Stanford and Duke Departments of Orthopedic Surgery
Chris Bral, PhD, DABT	*VP, Nonclinical Development*	Arrowhead Research, Vertex, Schering-Plough Research Institute
Peter Cancelmo, JD	*VP, General Counsel and Secretary*	Garvey Schubert Barer, Choate Hall
Tim Duffy	*Head of Business Development*	MDRNA, Prometheus, Procter & Gamble
George Gaitanaris, MD, PhD	*Chief Scientific Officer*	Nura, Primal, NCI
Michael Jacobsen	*Chief Accounting Officer & Treasurer*	Sarepta, ZymoGenetics, ICOS, Onvia, Mosaix
Daniel Kirby	*Chief Commercial Officer*	Celgene, Juno, Medivation, Amgen
Bruce Meiklejohn, PhD	*VP, CMC*	Eli Lilly
Catherine Melfi, PhD	*Chief Regulatory Officer*	Eli Lilly, Indiana University
Narinder Nangia, PhD	*VP, Biostatistics, Data Management & Programming*	Alkermes, PPD, Abbvie, Pfizer, Burroughs Wellcome, Proctor and Gamble
Tina Quinton, MS, JD	*VP, Patents*	Christensen O'Connor Johnson Kindness
J. Steven Whitaker, MD, JD	*Chief Medical Officer*	Allon Therapeutics, ICOS
Pete Williams	*VP, Human Resources*	Redbox, Outerwall, Coinstar, Washington Mutual, Expedia

Narsoplimab for Treatment of Diseases Associated with Endothelial Injury

Endothelial Injury Plays a Major Role in Pathogenesis of a Wide Range of Diseases

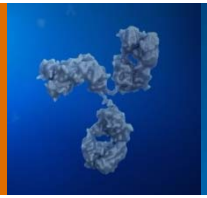
Endothelial injury plays a role in the pathogenesis of:

Stroke	Viral infections (e.g. COVID-19)	Chronic kidney disease
Peripheral vascular disease	Stem cell transplant-related complications (e.g., TMA, aGVHD, VOD, DAH, IPS, CLS)	Venous thrombosis
Cancer	Heart disease	Diabetes

Injury to endothelial cells occurs in many ways: physically, chemically, and immunologically

Damage to the vascular endothelium results in a procoagulant state and complement pathway activation

- Lectin pathway is a pattern-recognition system
- Lectin pathway is activated by carbohydrate patterns on microbes and surfaces of damaged cells



Endothelial Injury with Complement Activation is Central to the Pathophysiology of COVID-19

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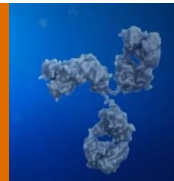
- Severe acute respiratory syndrome-associated coronavirus-2 (SARS-CoV-2) directly infects endothelial cells, leading to diffuse endothelial damage
- The resulting injury/inflammation specifically activates lectin pathway of complement on the endothelial cell surface
- Complement activation amplifies underlying cellular injury and induces cytokine response (e.g., IL-6)
- Complement activation has been demonstrated to cause lung injury; and complement blockade reduces that injury in models of MERS-CoV, SARS-CoV and SARS-CoV-2
- Complement activation in COVID-19 appears to be through the lectin pathway
- MASP-2, the lectin pathway's effector enzyme, binds the nucleocapsid protein of SARS-CoV-2, resulting in complement activation and lung injury

Role of Endothelial Injury in COVID-19 Published Across Numerous Peer-reviewed Journals



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Article Contents

Funding

References

COVID-19 as a cardiovascular disease: the potential role of chronic endothelial dysfunction

Register

THE LANCET Haematology

ARTICLES | ONLINE FIRST

Endotheliopathy in COVID-19-associated coagulopathy: evidence from a single-centre, cross-sectional study

PDF [498 KB] Figures

Matthew L Meizlish, MPhil

Show footnotes

Clinical & Experimental Immunology

The Journal of Translational Immunology

British Society for immunology

Original Article | Free Access

MASP2 levels are elevated in thrombotic micro association with microvascular endothelial cell suppression by anti-MASP2 antibody narsoplir

S. Elhadad, J. Chapin, D. Copertino, K. Van Besien, J. Ahamed, J. Laurence

First published: 18 July 2020 | https://doi.org/10.1111/cei.13497 | Citatic

nature reviews immunology

Comment | Published: 21 May 2020

COVID-19: the vasculature unleashed

Laure-Anne Teuwen, Vincent Geldhof, Alessandra Pasut & Peter Carmeliet

Nature Reviews Immunology 20, 389–391(2020) | Cite this article

39k Accesses | 8 Citations | 693 Altmetric | Metrics

An Author Correction to this article was published on 04 June 2020

This article has been updated

On the basis of -

Is COVID-19 an Endothelial Disease? Clinical and Basic Evidence

Celestino Sardu, Jessica Gambardella, Marco Bruno Morelli, Xujun Wang, Raffaele Marfella, Gaeta Santulli*

Version 1 : Received: 9 April 2020 / Approved: 13 April 2020 / Online: 13 April 2020 (02:23:33 CEST)

...VID-19, we ...to the ...here, we discuss ...between endothelial cells, viral ...se novel therapeutic

The NEW ENGLAND JOURNAL of MEDICINE

ORIGINAL ARTICLE

Ticagrelor and Aspirin or Aspirin Alone in Acute Ischemic Stroke or TIA

NEJM Catalyst eBook: The Clinician Role in Health Care Delivery and Innovation

ORIGINAL ARTICLE

Timing of Initiation of Ren... Replacement Therapy in Acu... Kidney Injury

Editor's Note: This article was published on May 21, 2020, at NEJM.org.

Mount Sinai Study Indicates COVID-19 May be Driven by Pulmonary Thrombi & Pulmonary Endothelial Dysfunction

APR 18, 2020 | COVID-19, ENDOTHELIAL DYSFUNCTION, ICAHN SCHOOL OF MEDICINE, MOUNT SINAI, THROMBOLYSIS, THROMBOSIS

...World Health ...ization

Medscape Friday, July 17, 2020

NEWS & PERSPECTIVE DRUGS & DISEASES CME & EDUCATION ACADEMY

Perspective > Medscape Oncology > EHA 2020

Endothelial Injury May Play a Major Role in COVID-19-Associated Coagulopathy

Alan P. Lyss, MD

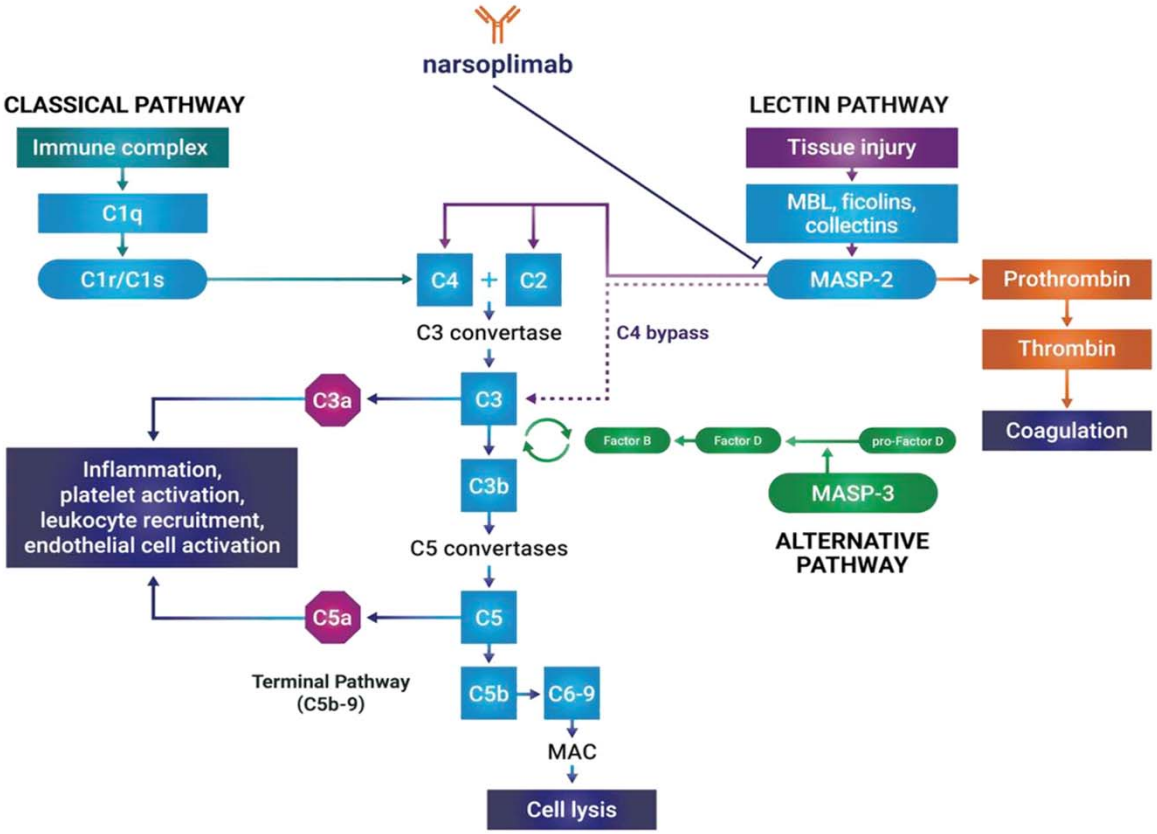
DISCLOSURES | June 29, 2020

ORIGINAL ARTICLE

Pulmonary Vascular Endothelialitis, Thrombosis, and Angiogenesis in Covid-19

Maximilian Ackermann, M.D., Stijn E. Verleden, Ph.D., Mark Kuehnel, Ph.D., Axel Haverich, M.D., Tobias Welte, M.D., Florian Laenger, M.D., Arno Vanstapel, Ph.D., Christopher Werlein, M.D., Helge Stark, Ph.D., Alexandar Tzankov, M.D., William W. Li, M.D., Vincent W. Li, M.D., et al.

Targeting the Lectin Pathway of Complement Has Potential Implications for Series of Disorders, Including COVID-19 and HSCT-TMA



Narsoplimab MoA

- Endothelial damage activates the lectin pathway of the complement system
- MASP-2 is the effector enzyme of the pathway
  - MASP-2 can activate the coagulation cascade, leading to TMAs
  - MASP-2 can activate the downstream complement cascade, resulting in potential autoimmune disorders
- Narsoplimab is designed to target and inhibit MASP-2 to prevent pathway activation

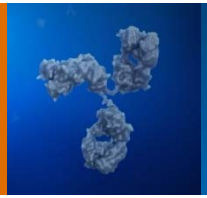
Lectin Pathway Disorders

- COVID-19
- HSCT and TMA-related EIS
  - aGVHD
  - CLS
  - DAH
  - IPS
  - SOS/VOD
  - HELLP/CAPS
- Chronic nephrology/ proteinuria diseases
  - IgAN
  - MGN
  - Lupus nephritis
- Oncology
  - Colorectal Cancer
  - Cervical Cancer
  - ESCC
- Acute transplant & surgery-related conditions
  - Delayed Graft Function-solid organ transplant

Parallels Between COVID-19 and HSCT-TMA

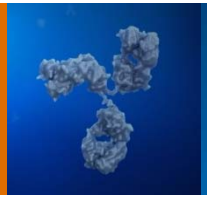
Comparator	COVID-19	HSCT-TMA
Lectin-Pathway Activation from Endothelial Damage	✓	✓
Cause of Endothelial Injury	Viral	Conditioning regimen, Immunosuppressants, GVHD, infection
MASP-2 Activation	✓	✓
Multi-Organ TMA	✓	✓

- **43 patients have been dosed with narsoplimab across the two conditions (COVID-19: 6 patients; HSCT-TMA: 37 patients) for diseases resulting from endothelial damage**
- **Marked improvement was noted in narsoplimab-treated patients in these studies**



Potential Advantages of Narsoplimab in COVID-19

- Pathophysiology of COVID-19-related lung injury is consistent with TMA – endothelial injury is central to both
- Endothelial injury, as observed in COVID-19 and HSCT-TMA, activates lectin pathway of complement
- Narsoplimab, a specific lectin pathway inhibitor, has shown positive results in HSCT-TMA
  - Has Breakthrough Therapy Designation for HSCT-TMA
  - Rolling BLA submission in progress
- Narsoplimab designed to leave classical and alternative pathways fully functional
  - Leaves the effector function of adaptive immune response intact
  - Maintains antigen-antibody complex-mediated lytic response and killing of virus-infected cells
  - Does not appear to increase infection risk
- In addition to lectin pathway inhibition, narsoplimab observed to have anticoagulation effects, potentially beneficial in COVID-19 patients
  - Inhibits MASP-2-mediated cleavage of prothrombin to thrombin, activation of kallikrein, and activation of factor XII to XIIa; blocks thrombus formation
  - No prolongation of PT, aPTT or bleeding time



Compassionate Use of Narsoplimab for COVID-19 Patients in Bergamo, Italy

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- 6 patients, each included for presence of ARDS requiring mechanical ventilation (4 on CPAP, 2 intubated) have been treated with narsoplimab
- Narsoplimab was administered through IV twice weekly for 2 to 4 weeks
- All patients fully recovered, survived and were discharged
- 2 patients also had massive bilateral pulmonary thromboses that resolved following narsoplimab treatment
- Temporal patterns of laboratory markers (CEC, IL-6, IL-8, CRP, LDH, AST and D-dimer) were consistent with the observed clinical improvement; in particular, CEC counts appear to be a reliable tool to evaluate endothelial damage and treatment response
- All patients received routine supportive care (prophylactic enoxaparin, azithromycin, hydroxychloroquine, darunavir/cobicistat) and, during the study, steroids (patient #1 did not receive steroids and patients 2 and 3 likely received no benefit from steroids given timing of initiation)
- Two control groups with similar entry criteria and baseline characteristics were selected for retrospective comparison showing substantial mortality rates of 32 percent and 53 percent
- Manuscript accepted for publication in peer-reviewed journal *Immunobiology*

Data from the COVID-19 Study in Italy

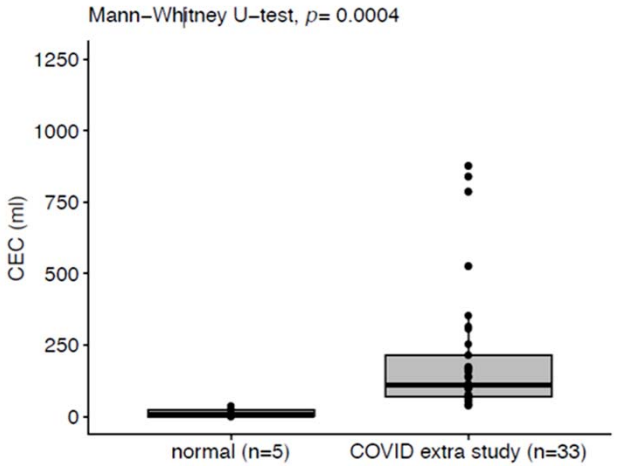
Demographics and Treatment Summary

Demographic	Median (range) or n (%)
Age	57 years (47-63)
Male sex	5 (83%)
Weight	86 Kg (82-100 Kg)
Comorbidities	Diabetes (n=1); Hypertension (n=1); Dyslipidemia (n=2); Obese/Overweight (n=6)

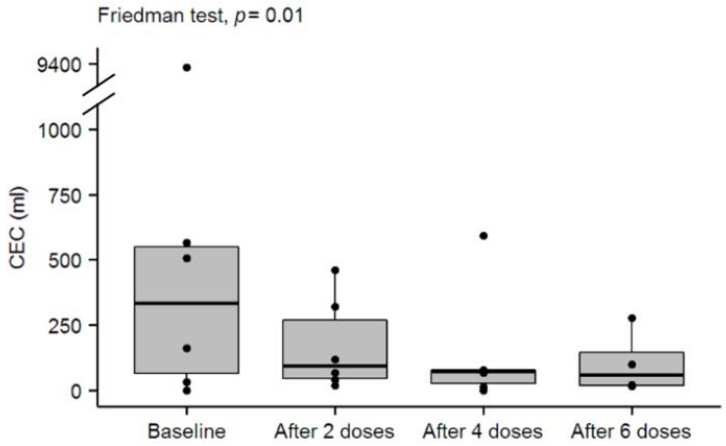
Treatment Summary	n (%) or Median (range)
Timing of narsoplimab treatment from start of CPAP oxygen support	
Within 24 hours	4 (67%)
Within 48 hours	2 (33%)
Time from hospital admission to treatment	2 days (1-4)
Duration of follow-up (to date) after first dose	27 days (16-90)

Data from the COVID-19 Study in Italy

Evidence of Endothelial Damage (CEC Counts) in COVID-19

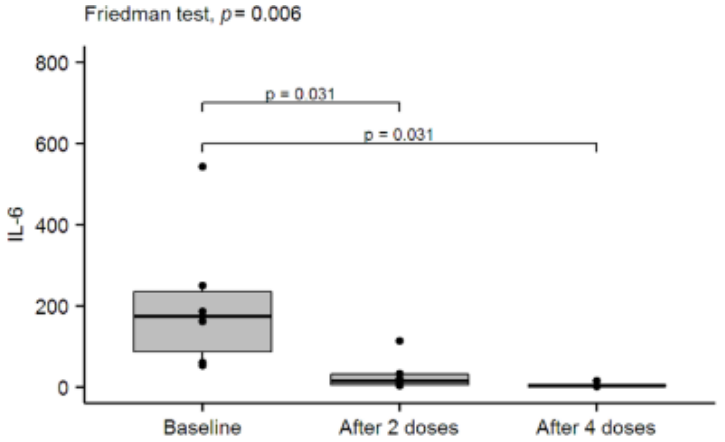


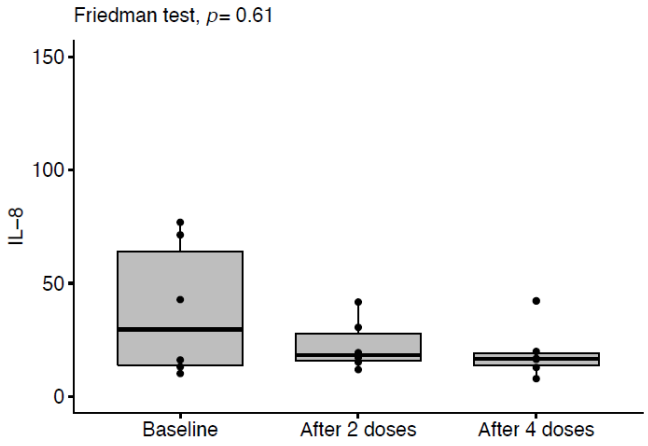
5 normal (uninfected) and 33 infected patients without Narsoplimab



6 infected patients treated with Narsoplimab

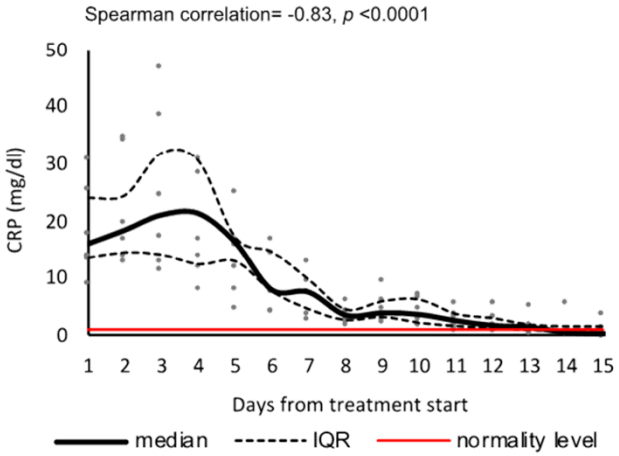
IL-6 / IL-8 Levels Improved in 6 Patients Treated with Narsoplimab



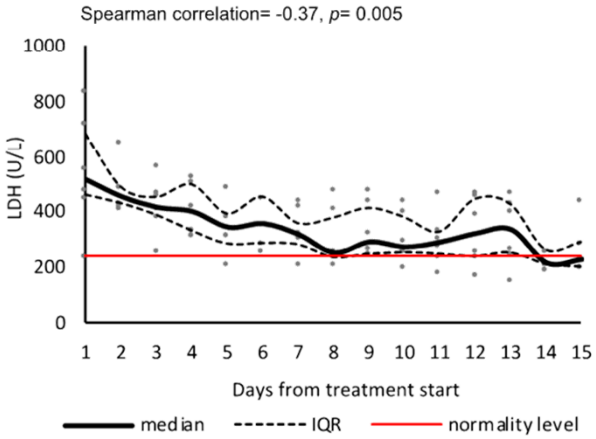


Data from Narsoplimab-treated COVID-19 Patients

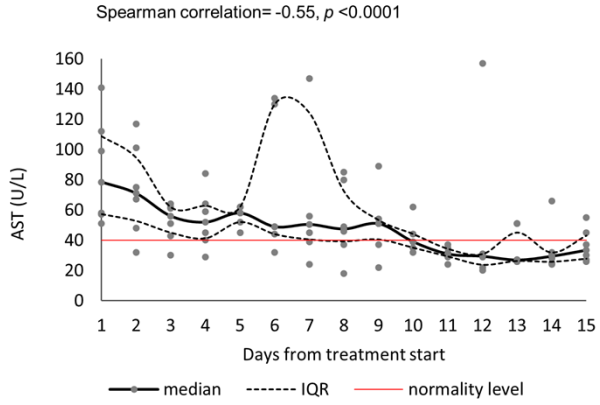
C-Reactive Protein
Improved in all 6 Patients



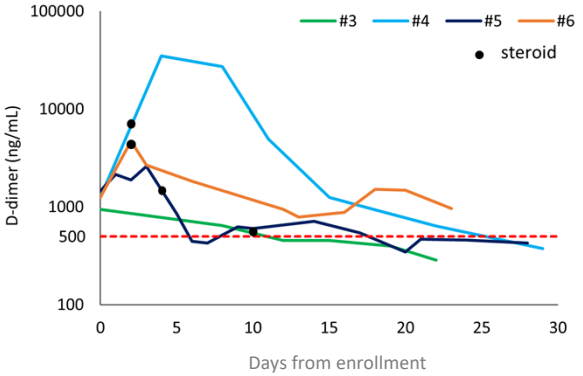
Lactate Dehydrogenase
Improved in all 6 Patients



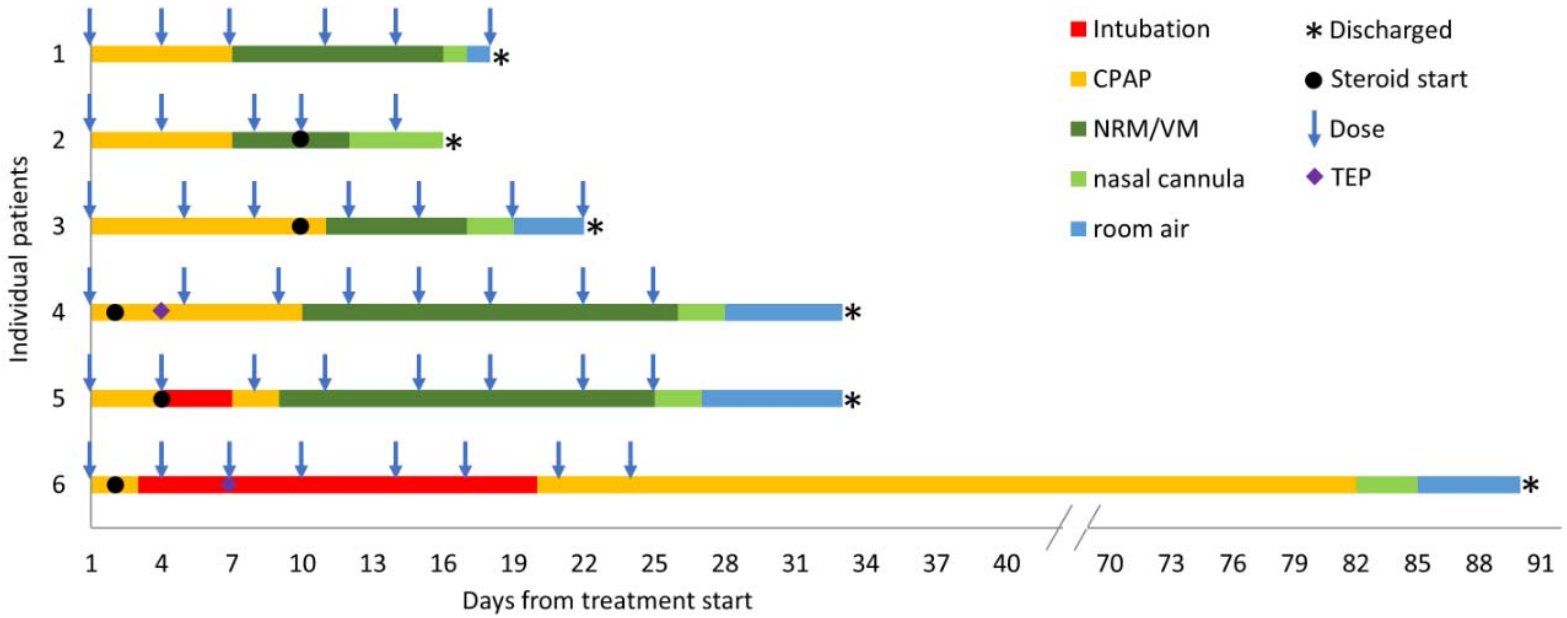
Aspartate Aminotransferase (AST)
Improved in all 6 Patients



D-Dimer
Improved in all Assessed Patients



Clinical Outcomes of COVID-19 Patients Treated with Narsoplimab



- The bar colors indicate the different oxygen support (CPAP = yellow; mechanical ventilation with intubation = red; non-rebreather oxygen mask/Venturi mask = green; low-flow oxygen by nasal cannula = light green; room air = blue)
- Narsoplimab doses are marked by blue arrows
- Black circle indicates the beginning of steroid treatment
- CPAP = continuous positive airway pressure; NRM = non-rebreather oxygen mask; VM = Venturi mask; TEP = pulmonary thromboembolism

Narsoplimab in HSCT-TMA

HSCT-TMA – Serious and Potentially Fatal Complication of HSCT Caused by Endothelial Injury

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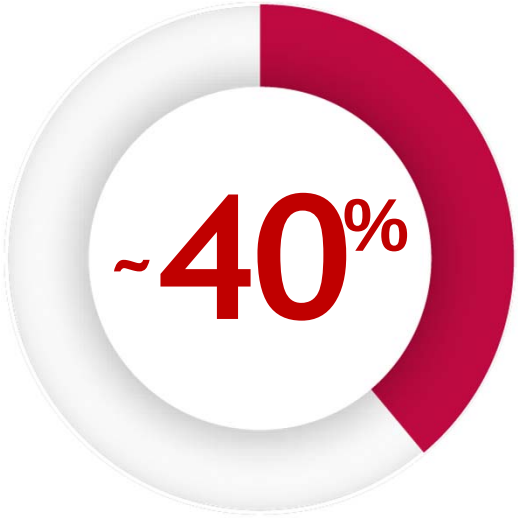
25,000 – 30,000
annual allogeneic HSCT in the US and EU

No approved therapies in HSCT-TMA



incidence
of TMA in allogeneic HSCT

of patients with HSCT-TMA display at least one **high-risk** feature

of severe cases of HSCT-TMA can be **fatal**

Narsoplimab In HSCT-TMA: Pivotal Study

Study Population

- Single-arm, open-label study of **high-risk** HSCT-TMA patients
- Protocol specified that patients receive narsoplimab once weekly for **≥ 4 weeks**
- 93% of the trial population had **multiple** risk factors for poor outcomes

Demographics	N=28
Mean age (years)	48
Male Gender, n (%)	20 (71%)
Malignant underlying disease	96%
Risk factors:	
Presence of GVHD, n (%)	18 (64%)
Significant infection, n (%)	21 (75%)
Non-infectious pulmonary complications (IPS or DAH), n (%)	4 (14%)
Neurological signs, n (%)	14 (50%)

Note: GVHD, graft versus host disease; IPS, idiopathic pulmonary syndrome; DAH, diffuse alveolar hemorrhage.

Efficacy Measures

- **Primary Endpoint:** Response as assessed by clinically meaningful improvement in TMA laboratory markers and organ function
  - *15% complete response rate is the FDA-agreed threshold for primary endpoint*
- **Secondary Endpoints:** 100-day survival and change from baseline in TMA lab measures

Key Findings

- Most narsoplimab-treated patients achieved a complete response, exceeding the targeted threshold for the primary efficacy endpoint
- 100-day survival was similarly impressive across all groups (responders, per-protocol, and intent-to-treat)
- Narsoplimab was well tolerated in this very sick population with multiple comorbidities
- No safety signal observed: observed adverse events were comparable to those typically seen in post-transplant population
- 21% of patients died during the trial due to causes common in HSCT

Narsoplimab In HSCT-TMA: Pivotal Study Results

Population	Complete Response Rate (%)
All treated patients (N=28) (95% CI)	54% (15/28) (34% to 72%)
Patients treated per protocol (≥ 4 weeks of dosing) (n=23) (95% CI)	65% (15/23) (43% to 84%)

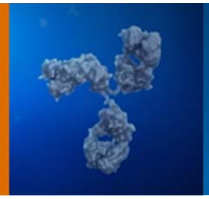
Population	100-Day Survival*
All treated patients (N=28)	68% (19/28)
Patients treated per protocol (≥ 4 weeks of dosing) (n=23)	83% (19/23)
Treatment responders (n=15)	93% (14/15)

*from date of HSCT-TMA diagnosis

Regulatory Milestones for Narsoplimab in HSCT-TMA

Narsoplimab in HSCT-TMA: Moving Rapidly Toward Global Regulatory Approvals

- Breakthrough therapy designation from FDA
- Orphan Drug designation from FDA and from EMA
- Pivotal trial enrollment closed
- FDA has agreed that the number of patients treated in the clinical trial (n = 28) is sufficient for BLA filing and review for approval
- Nonclinical and CMC portions of rolling BLA submitted; targeting 3Q 2020 for completion
- MAA submission is in preparation for submission to EMA; targeting 1H 2021 for completion
- Drug substance and drug product process validation lots successfully completed



Narsoplimab in HSCT-TMA Launch Readiness Milestones

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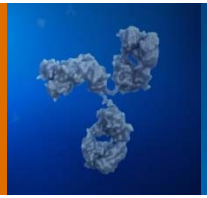
Engagement	Comprehensive engagement plan with top leaders from US and international transplant centers ✓ Introduce Omeros as a potential new partner in the transplant market ✓ Increase awareness of HSCT-TMA ✓ External steering committee establishing guidelines for diagnosis and treatment
Education	✓ Initiation of educational disease awareness campaign focusing on HSCT-TMA pathogenesis and unmet need ✓ International digital and print campaign ✓ Significant 2020 presence at US/EU hematology and transplant congresses
Value	Robust value framework to demonstrate clinical and financial value to global payers and providers ✓ Pricing strategy to ensure broad access across provider segments ✓ HEOR/RWE plan - reduction of post-HSCT complication costs; improved outcomes ✓ Convenient route of administration in inpatient and outpatient settings ✓ Pursuing coding strategy to ensure seamless access to narsoplimab, if approved (ICD-10, NTAP, J-code, etc.)
Operations	Organizational launch readiness ✓ Heads of national sales, medical science liaisons and advocacy already hired ✓ US Sales force hiring process initiated ✓ Long-term commercial manufacturing agreement with Lonza executed ✓ Process validation lots successfully manufactured; available for commercial use, if approved

Narsoplimab in IgA Nephropathy



Narsoplimab Clinical Program in IgAN

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Phase 2 Clinical Trial

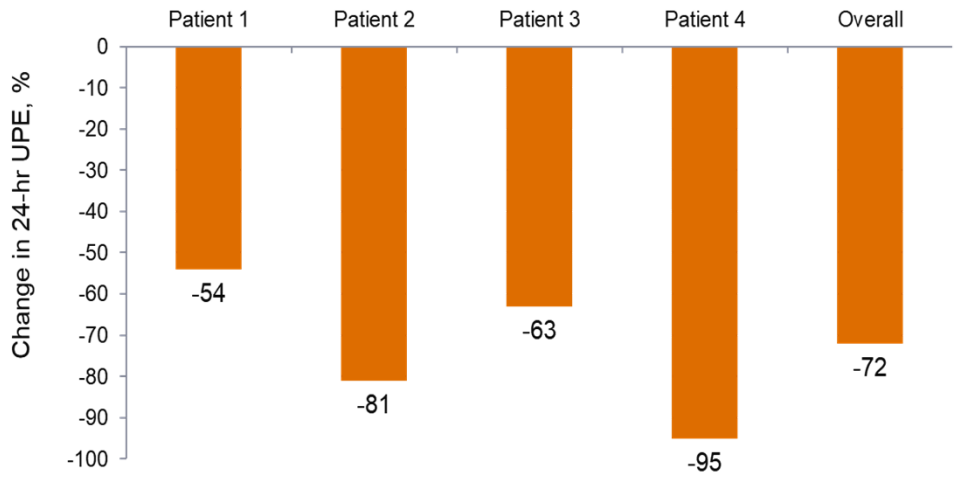
- Substudy 1: Narsoplimab in patients with IgAN, lupus nephritis, C3 glomerulopathy and membranous nephritis, all who were receiving treatment with corticosteroids
- Substudy 2: Narsoplimab in patients with IgAN who were not receiving corticosteroids

Phase 3 Clinical Trial: ARTEMIS-IGAN

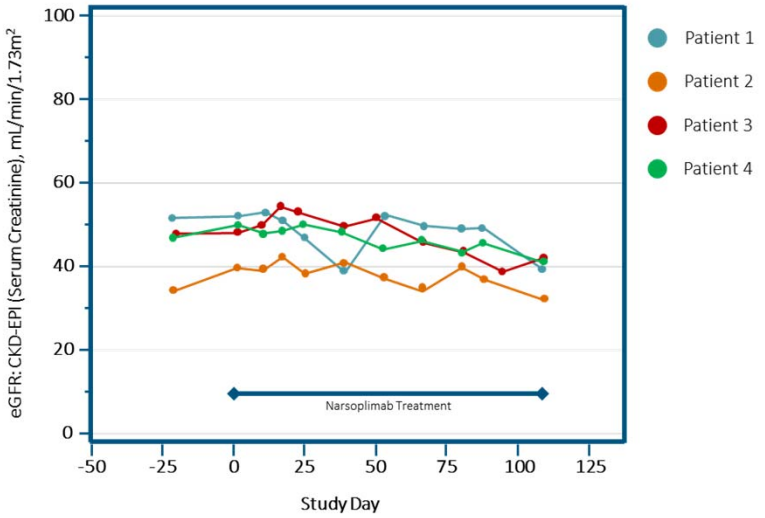
- Randomized, double-blind, placebo-controlled study of safety and efficacy of narsoplimab in patients with IgAN
- Reached consensus on trial design with FDA
- Based on 36-week assessment, could support a BLA seeking either full OR accelerated approval in EITHER:
  - Entire population (>1g/day proteinuria)
  - High-risk subset of patients (≥2g/day proteinuria)

Phase 2: Substudy 1 Results

Percent change in proteinuria at Week 18 compared with baseline in individual patients and overall



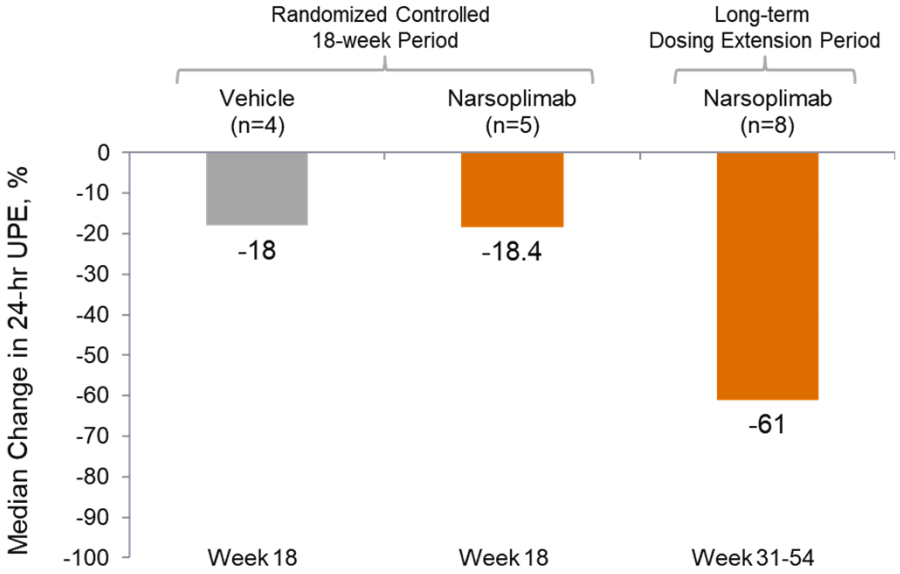
eGFR over time for individual patients in Substudy 1 from screening through Week 18



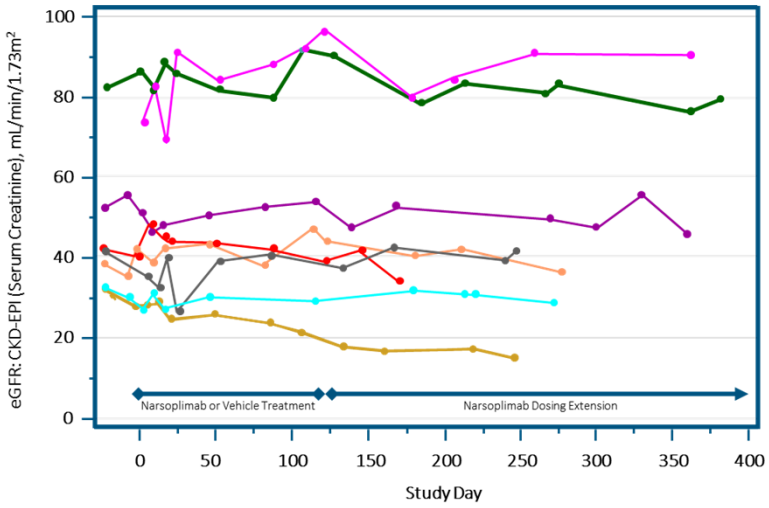
- No treatment-related serious adverse events (SAEs) were observed, and most of the reported AEs were mild or moderate in severity and reversible including fatigue, upper respiratory infection, headache, and alopecia
- All patients were able to taper completely off corticosteroids by the end of the substudy
- 4 of 5 lupus nephritis patients showed substantial (mean of 69%) decrease in 24-hour urine protein
- Response in membranous nephropathy was mixed

Phase 2: Substudy 2 Results

Median percent change in proteinuria at Week 18 compared with baseline in individual patients and overall



eGFR over time for individual evaluable patients from screening through last measurement during the ongoing dosing extension



- Further reductions were observed in the narsoplimab dose-extension period with a median reduction of 61% assessed at 31 weeks to 54 weeks post-baseline
- Narsoplimab was well tolerated in Substudy 2 with no drug-related AEs reported; 3 patients in Substudy 2 had a total of 5 Grade 3 AEs, all of which were reversible*

*1 patient randomized to vehicle experienced 2 Grade 3 AEs (urinary tract infection and acute kidney injury), a second patient randomized to narsoplimab had a known psychiatric illness and experienced an acute psychotic event that required hospitalization, and a third patient randomized to vehicle and continuing with narsoplimab in the dosing extension was diagnosed with hyperkalaemia at the beginning of narsoplimab treatment.

Phase 2: Substudy 2 Results (Cont'd)

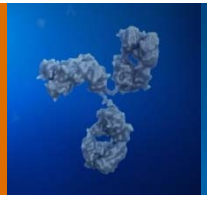
- Baseline demographics were not balanced between narsoplimab and vehicle groups (evaluable patients) – narsoplimab group was sicker with longer-standing disease
  - Study population had multiple co-morbidities

Baseline Characteristics	Vehicle	Narsoplimab
Median age, years	33	44
Median time since IgAN diagnosis, years	7	17
Median 24-hr UPE, g/day	4.0	2.4

- Manuscripts published or accepted for publication
  - J. Barratt and R. Lafayette, *MASP-2 inhibition as a potential strategy for the management of IgA nephropathy,* Drugs of the Future 2020, 45(6): 389-396
  - R. Lafayette, et. al., *Safety, Tolerability, and Effect of Narsoplimab (OMS721), a Novel MASP-2 Inhibitor for the Treatment of IgA Nephropathy (*accepted for publication in Kidney International Reports)

Summary of ARTEMIS-IGAN Phase 3 Trial; Enrolling at over 100 Sites Globally

Overview	• Phase 3 randomized, double-blind, placebo-controlled trial of narsoplimab vs. placebo in patients with IgA nephropathy • Planned enrollment primary endpoint = 280 patients (140/arm) ➢ High Risk Subset (≥2g UPE) = 156 patients (78/arm)
Inclusion Criteria	• Biopsy-confirmed diagnosis of IgAN within 8 years prior to screening • Proteinuria of >1 g/day within 6 months prior to screening or uPCR >0.75 by spot urine at screening • Mean of two proteinuria measurements >1 g/day at baseline • eGFR of ≥ 30mL/min/1.73 m^2 at screening and baseline
Efficacy Measures	• Primary efficacy endpoint: Change from baseline 24-hour urine protein excretion (UPE g/day) at 36 weeks from baseline for **EITHER** the entire population or the subset of "high-protein" spillers • Secondary efficacy endpoints include rate of change from baseline in eGFR



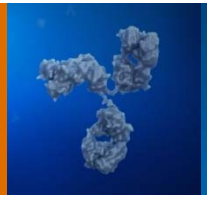
Regulatory Milestones for Narsoplimab in IgAN

Narsoplimab: Advancing Toward Global Regulatory Submissions in IgAN

- Breakthrough Therapy designation from FDA
- Orphan Drug designation from FDA and from EMA
- First and only IgAN investigational treatment to receive breakthrough therapy designation
- Potential to seek full or accelerated approval on proteinuria alone in either of the overall or high-protein-spiller populations
- Over 100 trial sites activated and enrolling for Phase 3 trial in US, EU, Australia, Canada and Asia; additional sites being activated
- Data readout on proteinuria endpoint targeted for next year

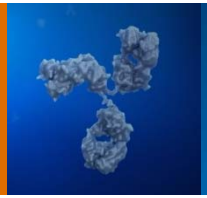
OMEROS

Narsoplimab in Atypical Hemolytic Uremic Syndrome (aHUS)



Narsoplimab
Summary of aHUS Phase 2 Results

- Improvements in TMA markers (platelets, LDH and haptoglobin)
- 3 aHUS patients were able to discontinue dialysis
- 3 others on chronic dialysis were deemed eligible for renal transplant, with one successfully transplanted to date
- Narsoplimab was well tolerated with predictable safety profile



Narsoplimab
Phase 3 Clinical Program in aHUS

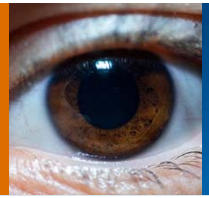
OMEROS

- Fast track and orphan designations from FDA
- Phase 3 trial in newly diagnosed or ongoing aHUS
- Agreement with FDA and EMA on one single-arm (i.e., no control group), open-label trial to satisfy both agencies
  - ~40 patients for EMA full and US accelerated approvals
  - ~80 patients for US full approval
- Clinical package for biologics license application (BLA) similar to that which formed basis of approval for Soliris® in aHUS
- Safety can be demonstrated across range of diseases
- FDA and EMA agreement on CMC and nonclinical safety/tox plans
- Pursuing US accelerated approval and European full approval
- Enrollment ongoing at sites in US, Europe and Asia

OMIDRIA® (phenylephrine and ketorolac intraocular solution) 1% / 0.3%

OMIDRIA®
Opthalmological Surgery

- First and only FDA-approved intraocular product to prevent miosis and to reduce postoperative ocular pain
  - Approved for both adult and pediatric patients
- Used in over 1 million cataract procedures without any safety concerns
- Strong post-launch ("real-world") clinical data
- On VA National Formulary and continuing to expand reimbursement across commercial and Medicare Advantage payers
- Issued patents through 2033 (2035 if pending patents issue)
- Nearly 4 million cataract procedures performed annually in US (~45-50% Med Part B; ~25-30% Med Advantage; ~20-30% commercial)
- Pass-through reimbursement status reinstated by Congress through September 30, 2020
- Pursuing administrative and legislative remedies for continued separate payment
  - NOPAIN Act introduced in House and Senate with broad and growing bipartisan co-sponsorship and leadership/committee-member support
  - Strong case for continued separate payment, like Exparel®, in ASC setting – OMIDRIA meets all criteria



Real-World Evidence — OMIDRIA® Improves Outcomes

OMEROS

Peer-reviewed publications detailing post-launch studies in adults and pediatric patients demonstrated that the use of OMIDRIA statistically significantly:

- ✓ Prevented IFIS[1]
- ✓ Prevented iris prolapse[1]

*Compared to steroids:**

- ✓ Reduced cystoid macular edema[2,3]
- ✓ Decreased breakthrough iritis[3]
- ✓ Reduced pain[3]

Compared to epinephrine:

- ✓ Decreased complication rates[4]
- ✓ Decreased use of pupil-expanding devices[4-8]
- ✓ Enabled performance of surgery and postoperative care without the use of steroids[2,3,9]
- ✓ Shortened surgical times[4,6,8]
- ✓ Reduced need for opioids (*i.e.*, fentanyl) during surgery while decreasing VAS pain scores[10]
- ✓ Prevented miosis during femtosecond laser-assisted surgery[7]
- ✓ Improved uncorrected visual acuity on day after surgery[4]

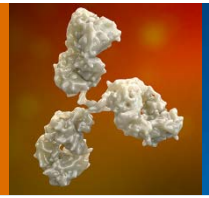
*OMIDRIA used intraoperatively with postoperative NSAIDs (no steroids) when compared to postoperative steroids with or without NSAIDs (no OMIDRIA)

1. Silverstein SM, et al. J Cataract Refract Surg. 2018;44(9):1103-1108. 2. Walter K, et al. J Cataract Refract Surg. 2020;46:350-354. 3. Visco DM, et al. Effect of intracameral phenylephrine and ketorolac 1.0%/0.3% on postoperative cystoid macular edema, iritis, pain, and photophobia following cataract surgery. J Cataract Refract Surg. In press. 2020. 4. Rosenberg ED, et al. Clin Ophthalmology. 2018;12:21-28. 5. Bucci FA, et al. Clin Ophthalmology. 2017;11:1039-1043. 6. Visco D. Clin Ophthalmol. 2018;12:301-305. 7. Walter K, et al. J Cataract Refract Surg. 2019;45(4):465-469. 8. Data on file. Clinical outcomes of phenylephrine/ketorolac intraocular solution versus epinephrine in cataract surgery in a real-world setting. 9. Al-Hashimi S, et al. J Cataract Refract Surg. 2018;44:1032-1041. 10. Donnenfeld, E et al. Clin Ophthalmol. 2019;13:2143-2150.

OMIDRIA Returning to Pre-COVID-19 Sales Highs

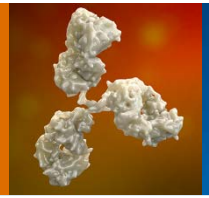
- 1Q 2020 and 2Q 2020 net sales of $23.5 million and $13.5 million, respectively, reflect COVID-19-related shutdown of cataract surgery and other elective procedures beginning in mid-March
  - Cataract surgeries resumed beginning in the second half of May and
  - By the end of June 2020 the run rate of weekly OMIDRIA sales had recovered to levels near those seen prior to pandemic
  - Many facilities operating at reduced capacity as a result of new safety protocols; OMIDRIA's clinical benefits appear to resonate well in this environment
- Throughout 2019, net revenues had been increasing at double-digit rates quarter-over-quarter driven by growth in number of accounts and deeper penetration within accounts across ASCs, hospitals and government payers
- On formulary at approximately half of the top 25 academic hospitals nationally
- Permanent J-code became effective October 1, 2019 and is expanding reimbursement, particularly by commercial and Med Advantage insurers

MASP-3 Development Program



OMS906: MASP-3 Inhibitor Targeting the Alternative Complement Pathway

- Omeros' lead MASP-3 inhibitor, OMS906, an investigational, fully-human monoclonal antibody designed to have high potency and selectivity for MASP-3 and potential to treat multiple alternative pathway-driven diseases with infrequent subcutaneous delivery
- By inhibiting MASP-3, OMS906 blocks conversion of pro-Factor D to Factor D
- Recently filed both CTA and IND; expect to begin enrollment this quarter in Phase 1 SAD/MAD study
- The initial targeted indication is paroxysmal nocturnal hemoglobinuria (PNH), a rare, acquired, life-threatening disease of the blood
- Targeting monthly subcutaneous dosing



OMS906 Inhibits MASP-3, Considered the Premier Target in the Alternative Pathway

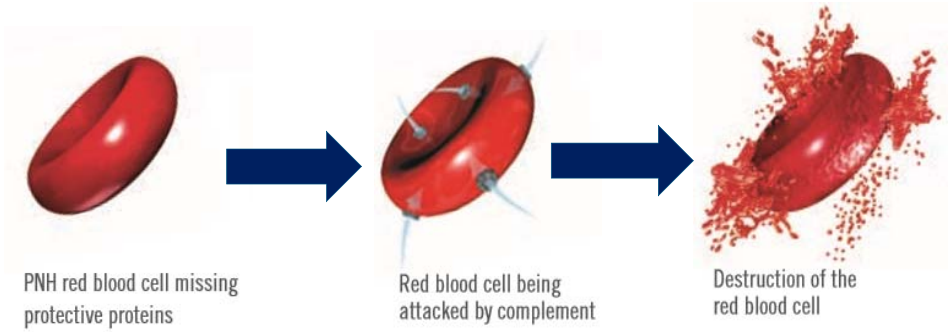
OMEROS

- MASP-3 is the key activator of the alternative pathway ("AP")
- MASP-3 is the premier target within the AP
  - Has the lowest concentration of all AP proteins
  - Has low relative clearance of AP targets
    - Example: ~50% of systemic CFD is cleared per hour
  - Unlike C5 and C3 blockers, leaves intact the lytic arm of the classical pathway, important in fighting infection

PNH is a Rare, Chronic, Life-Threatening Complement-Mediated Blood Disorder

OMEROS

PNH is characterized by intravascular and extravascular hemolysis



Unmet Need Persists

~70%

of PNH patients continue to have **low hemoglobin levels while on a C5 inhibitor**[1,2]

~1/3

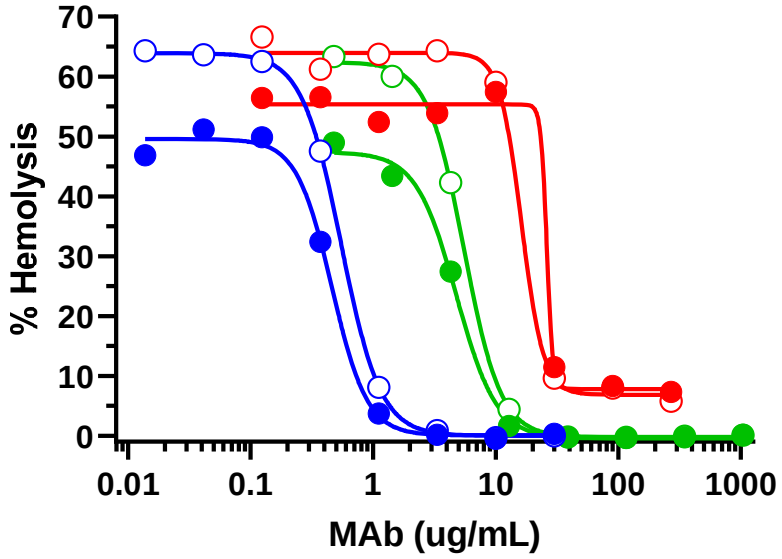
of PNH patients require **one or more transfusions a year while on a C5 inhibitor**[3]

1. Risitano AM, Marotta S, Ricci P, et al. (2019) Anti-complement Treatment for Paroxysmal Nocturnal Hemoglobinuria: Time for Proximal Complement Inhibition? A Position Paper From the SAAWP of the EBMT. Front. Immunol. 10:1157. doi: 10.3389/fimmu.2019.01157. 2. Risitano AM, Notaro R, Marando L, et al. (2009) Complement fraction 3 binding on erythrocytes as additional mechanism of disease in paroxysmal nocturnal hemoglobinuria patients treated by eculizumab. Blood. 2009 Apr 23;113(17):4094-100. 3. McKinley C. Extravascular Hemolysis Due to C3-Loading in Patients with PNH Treated with Eculizumab: Defining the Clinical Syndrome. Blood. 2017;130:3471.

Model of Intravascular Lysis of PNH RBCs (Human): OMS906 Compared to C3 and C5 Inhibitors

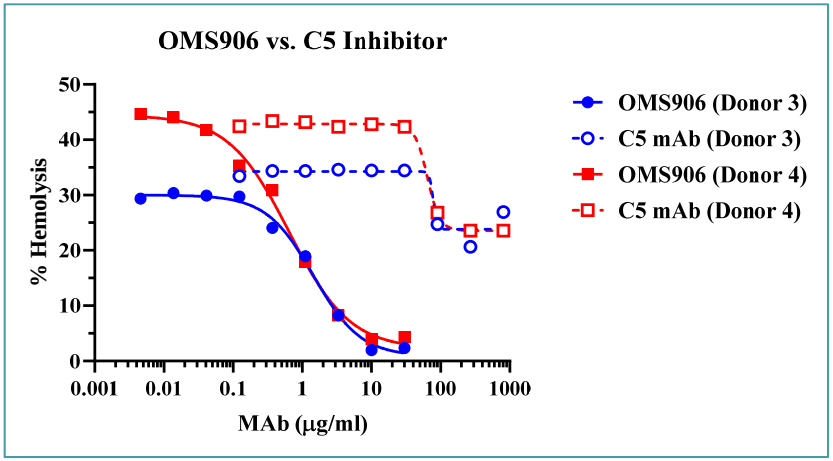
OMEROS

OMS906 vs. C5 and C3 Inhibitors



- OMS906 (Donor 1)
- OMS906 (Donor 2)
- C5 mAb (Donor 1)
- C5 mAb (Donor 2)
- Compstatin (Donor 1)
- Compstatin (Donor 2)

	C5 MAb	Compstatin
Relative OMS906 Potency	~30-fold	~1000-fold



OMS906 showed greater potency and greater degree of pathway inhibition

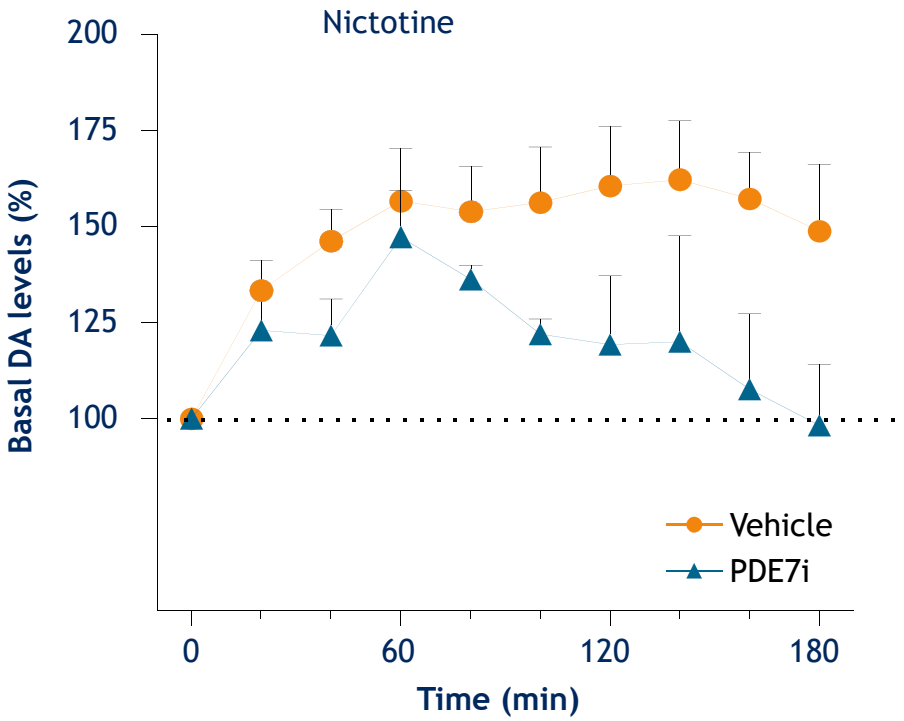
Addiction: OMS527

OMS527
PDE7 Inhibitor

OMEROS

- Novel target and novel mechanism for treating addiction
- Mechanism is highly conserved between humans and rodents
- Works through the dopamine system
- PDE7 inhibitors in animal models:
  - Have not appeared to alter reward system (no interference with other pleasurable activities)
  - Reduced both craving and relapse
  - Did not exhibit addictive properties
- Significant effects observed in animal models of:
  - Nicotine, cocaine, alcohol, and opioid addiction
  - Binge eating
- Broad issued and pending patents internationally cover any PDE7 inhibitor for treatment of any addiction or compulsive behavior
- Nicotine addiction selected as initial indication
- Manuscript detailing OMS527 data and PDE7 mechanism of action submitted for peer-reviewed publication

OMS527
Dopamine Levels in the "Liking" (or Craving) Phase

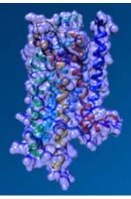


PDE7 inhibition reduced nicotine-induced increase of extracellular dopamine levels in the rat nucleus accumbens

OMS527
Clinical Development

- Phase 1 trial assessed the safety and pharmacokinetics of the study drug (OMS182399)
- Double-blind, randomized, placebo-controlled trial evaluated
  6 single-ascending-dose and 3 multiple-ascending-dose cohorts
- Met primary safety and tolerability endpoints
  - No significant adverse events were reported and OMS182399 was generally well-tolerated over the dose ranges tested – no meaningful difference from placebo
- Data showed a favorable and dose-proportional pharmacokinetic profile supporting once-daily dosing

G-Protein Coupled Receptors (GPCR) Platform

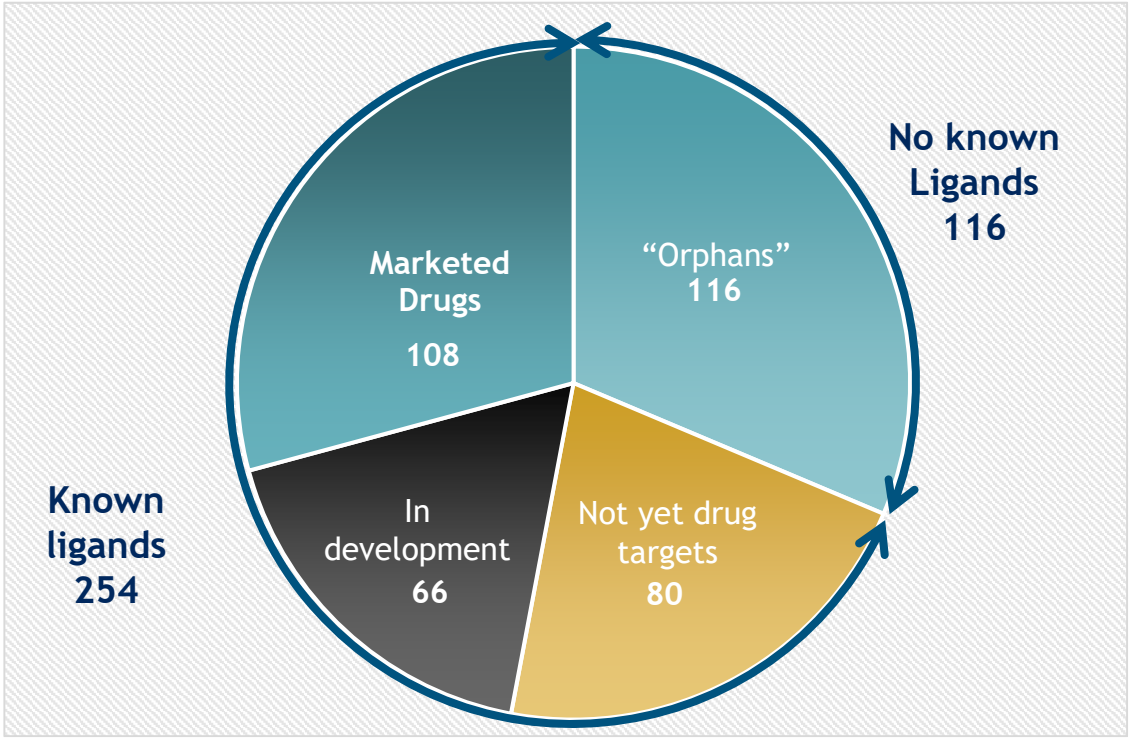


GPCRs - Attractive Drug Targets

OMEROS

GPCRs are promising drug targets, but there are challenges in drug discovery

370 Non-Sensory GPCRs

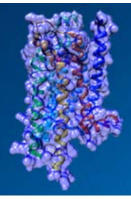


Challenges

- Ligand required for assay development
- Signaling pathway not known
- Laborious fractionation for natural ligand identification
- Current technologies limited only to agonist screening

Opportunities

Over 100 new drug targets



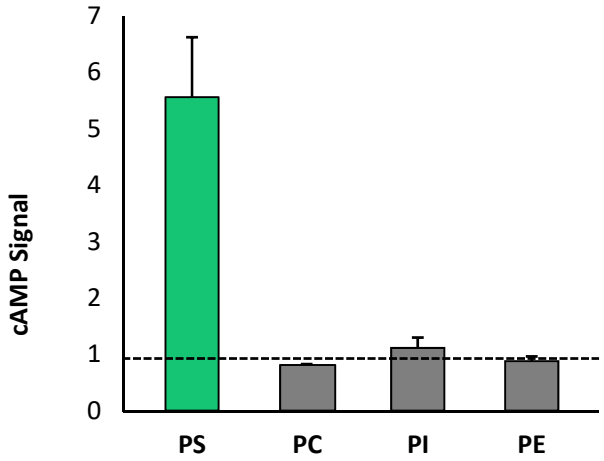
Immuno-Oncology: GPR174

Preclinical studies showed synergistic effects with checkpoint inhibitors and cellular therapies; enhancing anticancer effects

- GPR174 is an orphan GPCR expressed almost exclusively in immune cells
- GPR174 inhibition potentiated the immune system and suppresses tumor promoters
- Combined inhibition of GPR174 and the adenosine pathway synergistically enhanced anticancer effects
- Amenable to combination with check-point inhibitors and cellular therapies
- Potential to address non-responders to current therapies

GPR174 Is Activated by Phosphatidylserine (PS), a Product of Cell Death and Stress

GPR174 Signaling



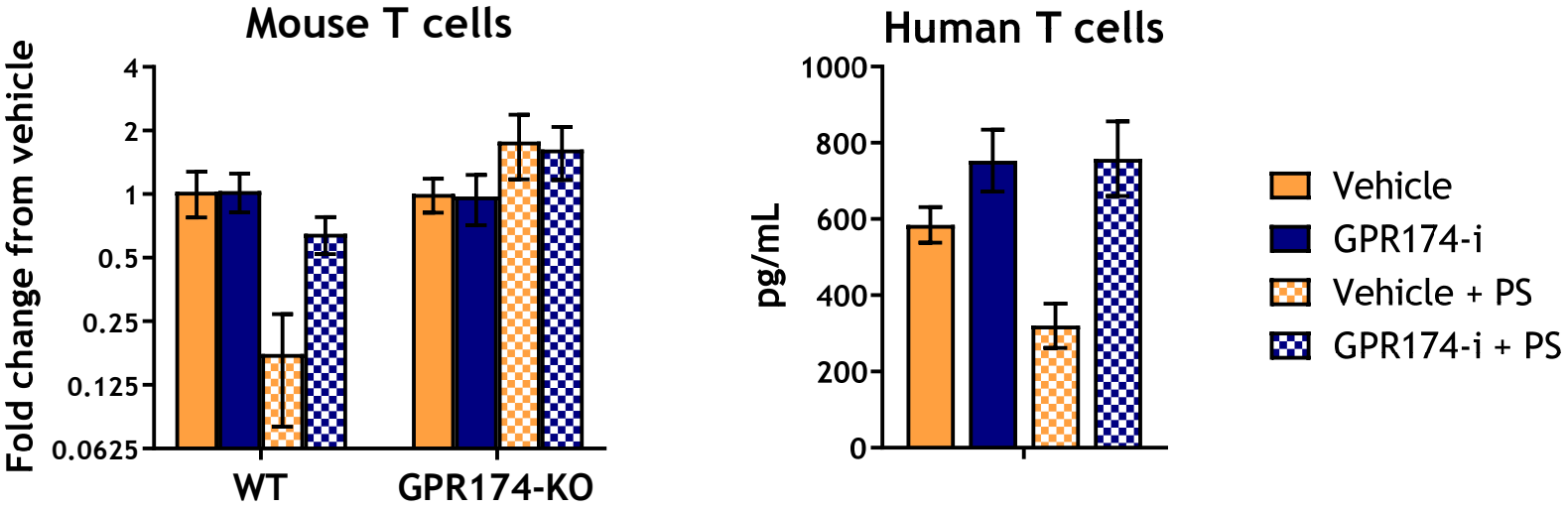
Liposomes made with:

- Phosphatidylserine (PS)
- Phosphatidylcholine (PC)
- Phosphatidylinositol (PI)
- Phosphatidylethanolamine (PE)

- PS is a global immunosuppressive signal in cancer

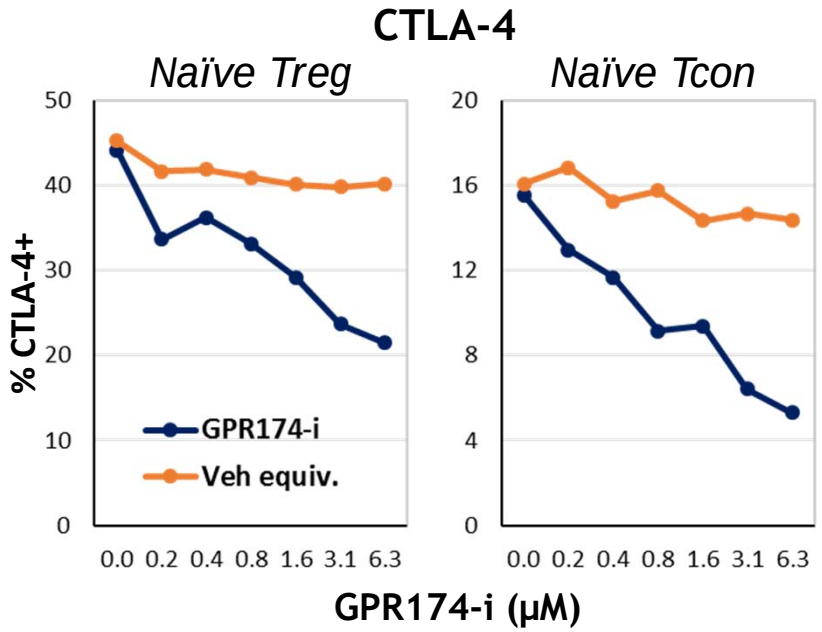
PS Activity on Purified T Cells Is GPR174-Dependent and Is Inhibited by GPR174-i

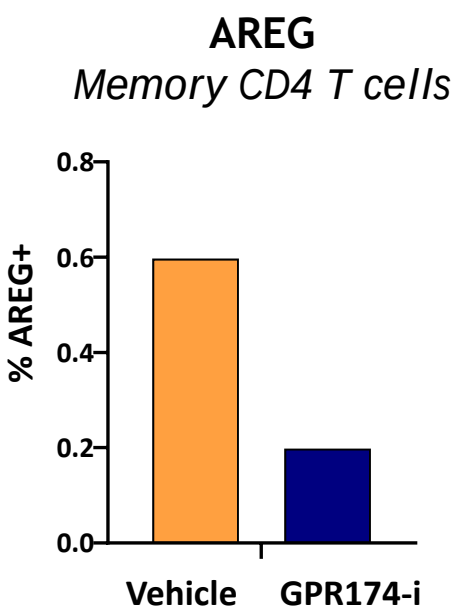
IL-2



- IFN-γ and TNF are also induced

GPR174 Inhibition Reduced Expression of Tumor Promoting Immunomodulators



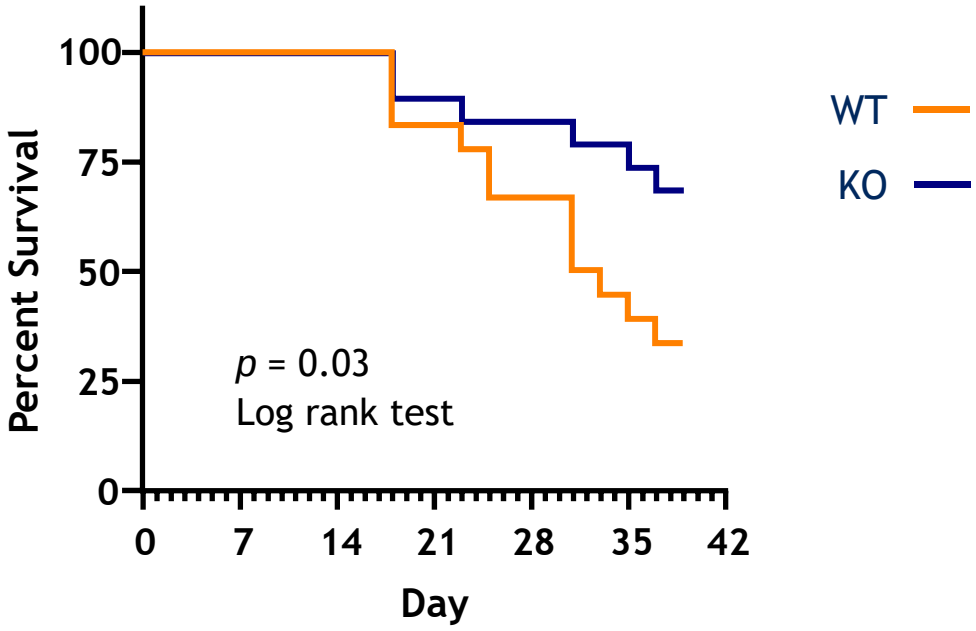


- CTLA-4 is an immune checkpoint targeted by YERVOY®
- Amphiregulin (AREG) is a tumor-promoting growth factor

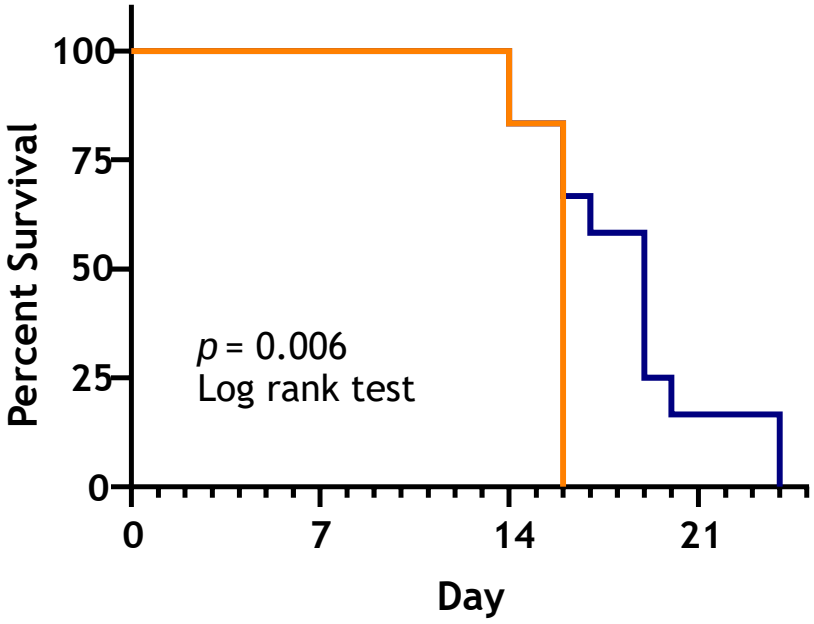
GPR174 Deficiency Synergizes With Anti-GITR-Mediated Treg Attenuation to Activate Anti-Tumor Immunity

OMEROS

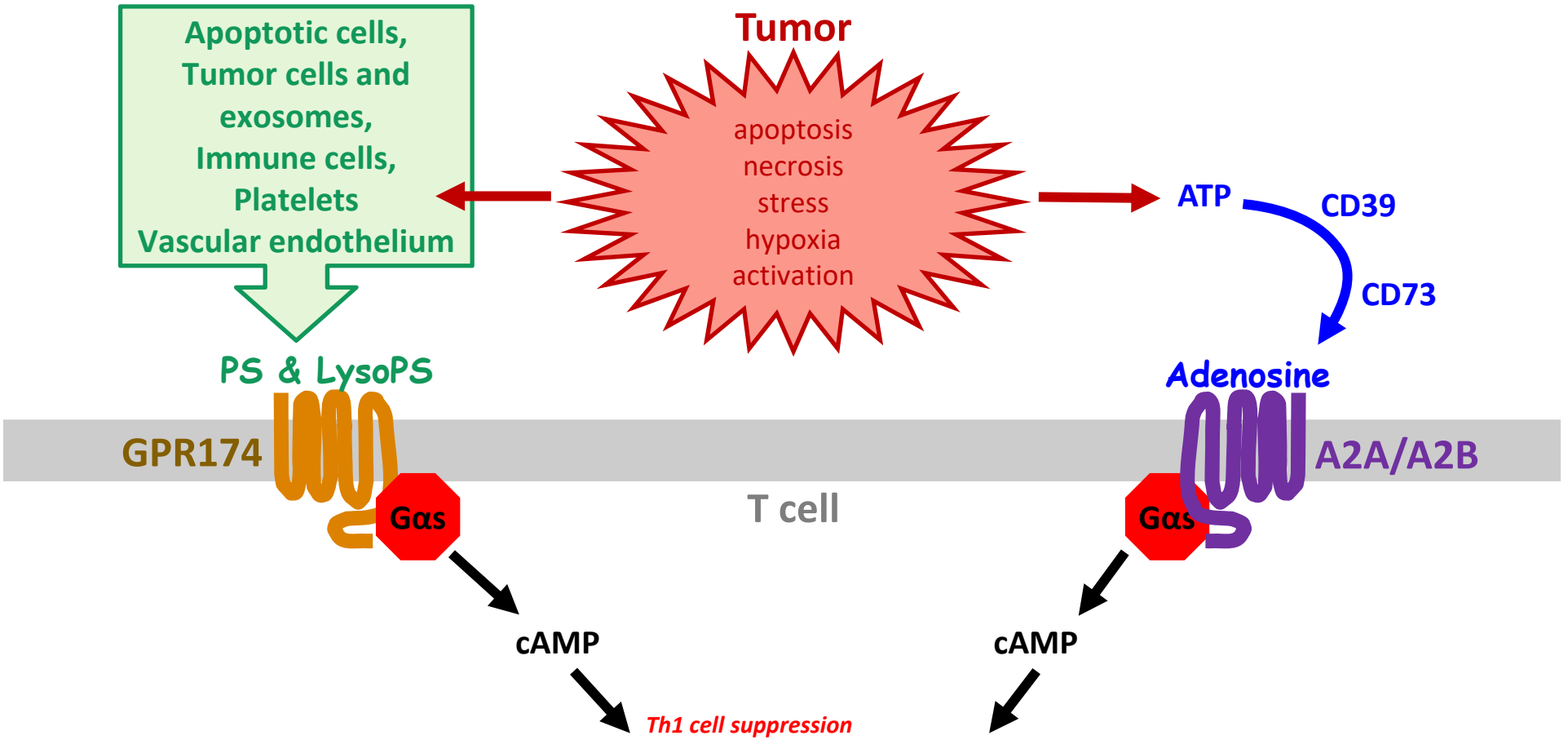
Colon Carcinoma



Melanoma



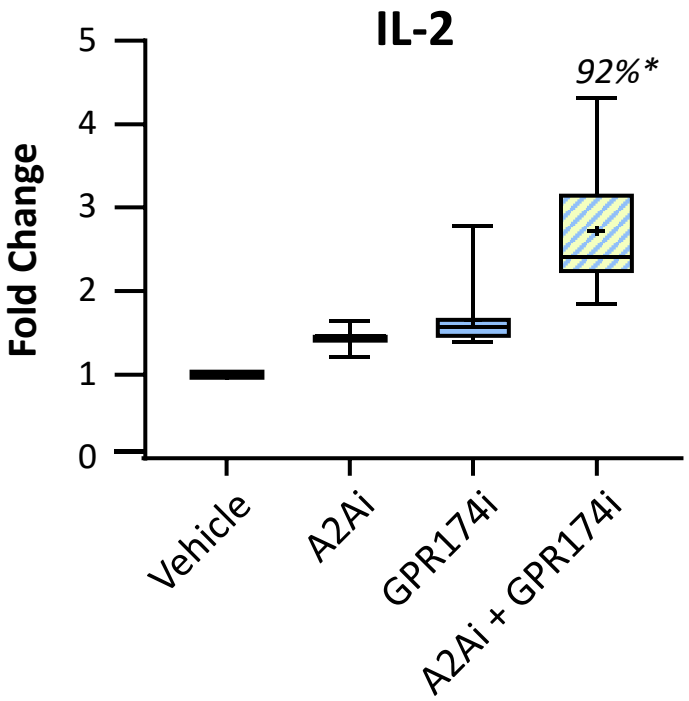
Activating Ligands for both GPR174 and Adenosine Receptor A2A/A2B Are Products of the Tumor Microenvironment



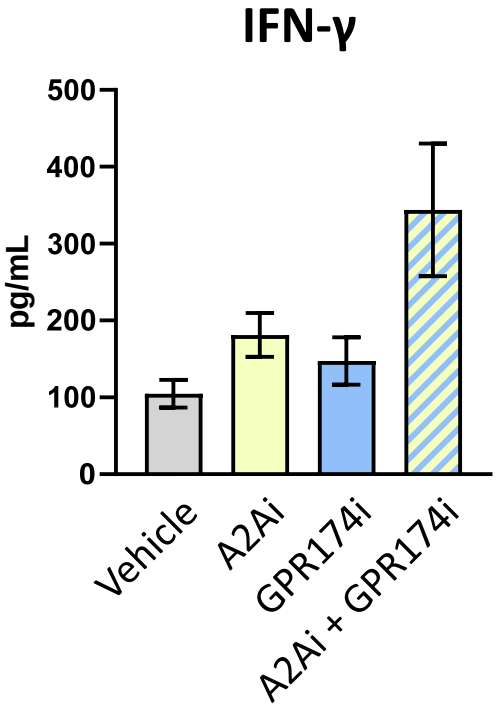
GPR174 and A2A/A2B adenosine receptors suppress T cells through the cAMP pathway

Inhibition of GPR174 and A2A Receptors Synergistically Activates Human T Cells

Total PBMC culture
High cell density, rich in PS and adenosine



CD8 T cell culture
Low cell density,
with supplemented PS and adenosine (NECA)



Normalized Data from 12 Human Donors

**Percent of donors exhibiting GPR174i/A2Ai synergy*