Omeros Corporation (OMER)
Rating: Buy

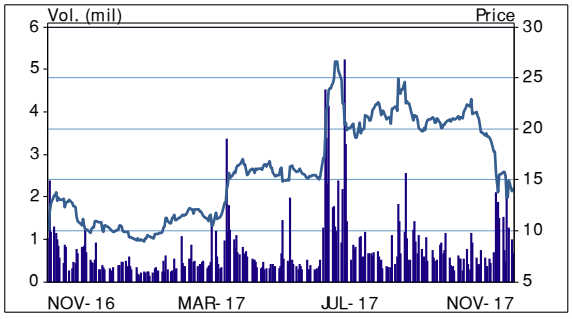
Raghuram Selvaraju, Ph.D.
212-916-3966
rselvaraju@hcwresearch.com

An Underrated Biopharma with a GPCR Engine; Initiating at Buy with $30 Price Target

Stock Data	11/07/2017
Price	$14.08
Exchange	NASDAQ
Price Target	$30.00
52-Week High	$27.09
52-Week Low	$8.71
Enterprise Value (M)	$726.1
Market Cap (M)	$674
Public Market Float (M)	42.2
Shares Outstanding (M)	47.9
3 Month Avg Volume	830,038
Short Interest (M)	8.44
Balance Sheet Metrics	
Cash (M)	$29.67
Total Debt (M)	$81.76
Total Cash/Share	$0.62
Book Value/Share	$(1.23)

EPS Diluted			
Full Year - Dec	2016A	2017E	2018E
1Q	(0.54)	(0.34)A	(0.31)
2Q	(0.32)	(0.33)A	(0.30)
3Q	(0.34)	(0.33)	(0.24)
4Q	(0.45)	(0.31)	(0.12)
FY	(1.65)	(1.30)	(0.96)

Revenue ($M)			
Full Year - Dec	2016A	2017E	2018E
1Q	7.4	12.3A	19.7
2Q	10.0	17.2A	20.5
3Q	11.3	17.5	23.9
4Q	12.9	18.9	27.6
FY	41.6	65.8	91.7



Undervalued based on marketed drug alone; pipeline a bonus. We are initiating coverage of Omeros Corporation, an emerging biopharmaceutical company headquartered in Seattle, WA, with a Buy rating and 12-month price target of $30.00 per share. In our view, the Street fails to appreciate even the value in Omeros's sole marketed product, the ophthalmology drug OMIDRIA (ketorolac + phenylephrine ophthalmic solution) for use in conjunction with cataract surgery and intra-ocular lens (IOL) implantations and replacements. While Omeros currently trades at an enterprise value of under $700M, we believe the net present value of the OMIDRIA franchise is over $800M. OMIDRIA could generate top-line revenue of nearly $66M this year, rising to over $90M next year, with peak annual sales of roughly $400M in 2022. In addition, we feel that the market does not currently give Omeros any credit whatsoever for its extensive clinical-stage pipeline. We have conservatively elected to solely project and value one of these candidates, the anti-MASP-2 monoclonal antibody OMS721, which Omeros is developing in three orphan indications: (1) atypical hemolytic uremic syndrome (aHUS); (2) hematopoietic stem cell transplant-associated thrombotic microangiopathies (HSCT-TMA); and (3) IgA-associated nephropathy (IgAN), also known as Berger's disease. In our view, OMS721 constitutes a pipeline in a single product candidate, and could generate peak sales of over $1.5B in the U.S. alone if approved in these three indications. The drug is currently in a Phase 2/3 trial for aHUS and is slated to enter pivotal testing in the other two indications within the coming months.

Pass-through expiry a bump in the road, not a brick wall. Despite the recent indication that pass-through status for OMIDRIA—which had effectively set pricing at $465 per vial—is slated to expire at the end of this year, we believe that lower pricing is likely to be counterbalanced by increased volume and that the product should continue to gain traction among ocular surgeons, who are favorably disposed towards its differentiated profile that makes surgeries easier without exposing patients to systemic side effects. Accordingly, we expect the product to keep growing even if the price per vial is substantially reduced, and believe that this could even prove an incentive to spur wider use. While we have projected a reduction in the per-vial price to the $300 range, it is also possible that the price would stay closer to where it is currently, which may thus drive upside to our estimates.

Sizzle plus the steak. We like the fact that OMS721 is a monoclonal antibody targeting a well-validated area—namely, complement pathway activation. The most well-known drug on the market that is positioned in this domain is Soliris (eculizumab) from Alexion Pharmaceuticals, which is one of the world's most expensive drugs, costing over $500,000 per patient annually. We note that Alexion's annual sales of Soliris—despite its being used in only very rare disease indications—have already exceeded $3 billion. OMS721 appears to be a potential competitor on a variety of fronts; it is more potent, has thus far demonstrated a benign safety profile (while Soliris is associated with infections, pulmonary edema and several other potentially life-threatening side effects) and also provides dosing and route of administration flexibility (subcutaneous and intravenous injection, while Soliris can only be given via intravenous infusion in the hospital setting).

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Multiple pipeline catalysts that could be value drivers near-term. Conservatively, we do not model or assign value to any of Omeros's other pipeline candidates, including OMS824, which is in Phase 2 testing for Huntington's disease and schizophrenia; OMS405, in mid-stage development for treatment of opioid and nicotine addiction; and OMS527, which is slated to enter clinical development next year and that could target a range of addictive and compulsive behavioral disorders. Omeros also has a preclinical antibody targeting a different element of the same pathway as OMS721, designated OMS906. This agent could be aimed at broader indications, including various autoimmune diseases like rheumatoid arthritis (RA) and multiple sclerosis (MS).

G-protein coupled receptor (GPCR) drug discovery technology platform slated to feed pipeline going forward. Omeros possesses what we would characterize as almost large pharma-strength drug discovery capabilities, thanks to its proprietary GPCR screening platform. This technology permits the company to efficiently and accurately identify ligands to so-called "orphan" GPCRs, which may thus be candidate compounds active against hitherto-undruggable targets. We note that Omeros currently has an extensive preclinical pipeline in the GPCR-modulating domain, which could yield future clinical programs for years to come. Currently, we do not ascribe any value to this platform, which thus could prove a source of future upside. Multiple GPCR-targeting drugs have achieved blockbuster status over the course of the history of the pharmaceutical industry, including products like Zyprexa (olanzapine) from Eli Lilly & Co., an atypical anti-psychotic drug deployed to treat schizophrenia and bipolar disorder; Clarinex (desloratadine) from Schering-Plough, now part of Merck & Co., which was used to treat allergy symptoms; and Zantac (ranitidine HCl) from GlaxoSmithKline, used to treat heartburn and ulcers.

Significantly undervalued relative to earlier-stage rare disease-focused companies. We note that Omeros trades at a very modest market cap relative to other companies with orphan drugs, which do not have any of the other attributes that Omeros possesses, namely a marketed product generating revenue, an extensive pipeline aimed at non-orphan diseases as well as rare disorders, and two technology platforms enabling the generation of additional preclinical programs. Examples of firms that trade at a significant premium to Omeros with less critical mass, in our opinion, include: Amicus Therapeutics, valued at $2.4B; AveXis, valued at nearly $3.3B; Blueprint Medicines, valued at $2.8B; and Ultragenyx, valued at $2B. Our valuation methodology utilizes a risk-adjusted net present value (rNPV) approach that ascribes a total firm value of $1.7B to Omeros based solely on OMIDRIA and OMS721. We have utilized a 12% discount rate for OMIDRIA and a 15% discount rate for OMS721, along with a 60% probability of success for OMS721. This translates into a price target of $30.00 per share, given 58M fully-diluted shares outstanding projected as of end-2018.

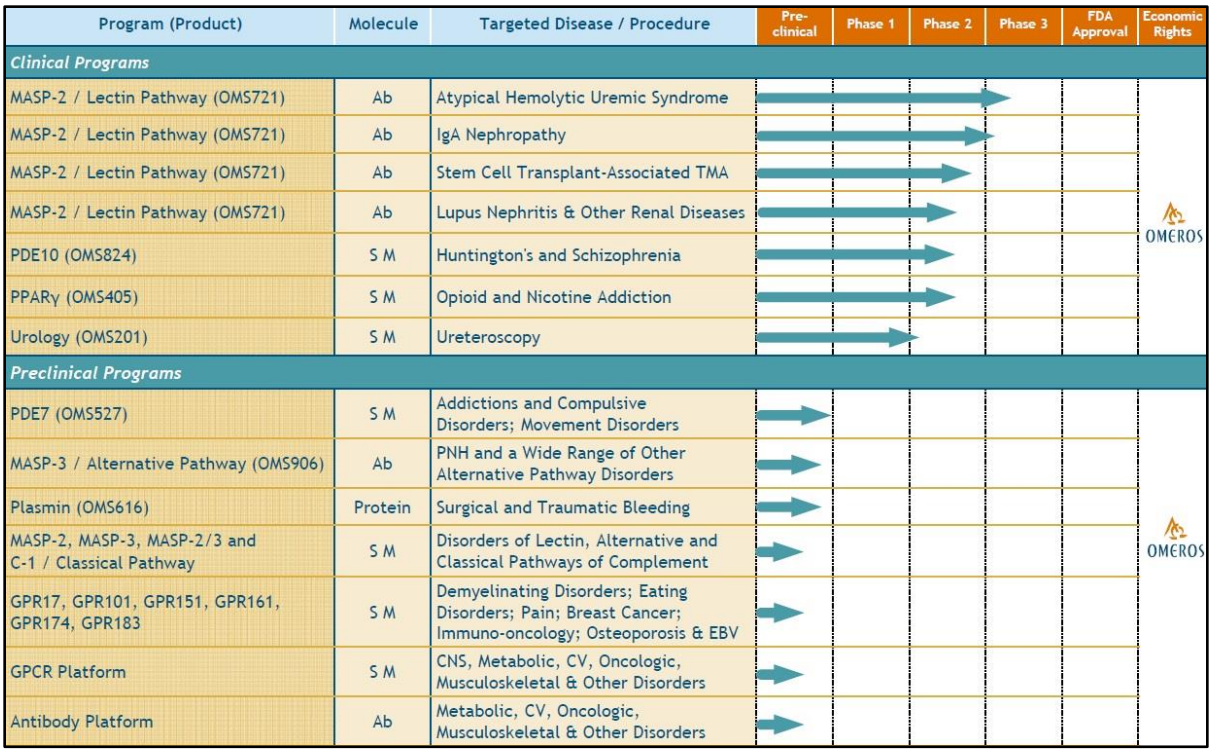
Company Overview

Omeros Corporation is a Seattle, WA-headquartered biopharmaceutical company discovering, developing and commercializing both small molecule and protein therapeutics for mass market as well as orphan indications targeting inflammation, coagulopathies and disorders of the central nervous system (CNS). The company also possesses a single marketed product, OMIDRIA® (phenylephrine 1%/ketorolac 0.3% injection), which is approved in the U.S. and various European countries for the maintenance of pupil size in patients undergoing cataract surgery or intra-ocular lens (IOL) replacement. The Omeros clinical-stage pipeline comprises three principal assets:

- OMS721, a human monoclonal antibody targeting mannan-binding lectin-associated serine protease-2 (MASP-2), which is in pivotal testing for atypical hemolytic uremic syndrome (aHUS) and in Phase 2 testing for both hematopoietic stem cell transplant-associated thrombotic microangiopathies (HSCT-TMA) as well as IgA nephropathy (IgAN) and other renal diseases;
- OMS824, a proprietary small molecule inhibitor of phosphodiesterase 10 (PDE10), which is being assessed in Phase 2 trials for treatment of Huntington's disease and schizophrenia;
- OMS405, a proprietary small molecule peroxisome proliferator-activated receptor gamma (PPARγ) agonist in Phase 2 studies for opioid and nicotine addiction, plus cocaine use disorder;
- OMS201 is an agent for use in urological applications—primarily uroendoscopic procedures—that has completed a Phase 1/2 trial in 2010 but is not currently in active development.

We are initiating coverage of OMER with a Buy rating and a 12-month price target of $30.00 per share, based on a discounted cash flow-driven risk-adjusted net present value (rNPV) methodology. An investment in Omeros may entail significantly above-average risk and volatility, in our view. The company's development-stage pipeline behind OMIDRIA is shown below.

Figure 1: Omeros Corporation Development-Stage Pipeline



Source: Company presentations.

Investors should also note that, as shown above, Omeros possesses a broad preclinical-stage pipeline and at least two drug discovery technology platforms, notably a G-protein coupled receptor (GPCR) screening platform and a proprietary antibody screening platform. Conservatively, we have not attributed any value to the company's preclinical pipeline or its drug discovery technologies.

Investment Highlights

1. A cascade of catalysts over the next 12 – 18 months; rapid clinical pathway for OMS721

We believe that upcoming events for Omeros constitute key reasons to own the stock in the near term. In our view, the Phase 3 trial of OMS721 in aHUS could complete enrollment in 2018 and also generate a readout from an interim analysis conducted on the first 40 patients—both catalysts that could potentially occur in 1H18—while both the IgAN and TMA indications for the drug could be advanced even more rapidly because of what we consider to be a significantly greater unmet medical need in these conditions vs. aHUS. In our view, further progress with OMS721 could position Omeros as a leader in the domain of lectin pathway disorders, specifically within the context of regulating aberrant complement pathway activation. The table below depicts recently-achieved and future milestones. Bolded items represent particularly critical stock catalysts.

Table 1: Near-Term Catalysts and Upcoming Events

Event	Timing	Importance
Completed		
Initiation of enrollment in pivotal Phase 3 trial of OMS721 monoclonal antibody in aHUS	**March 2017**	**High**
Completion of IgA nephropathy cohort in Phase 2 trial of OMS721	May 2017	Medium
Breakthrough Therapy status accorded to OMS721 in IgA nephropathy (IgAN)	**June 2017**	**High**
Orphan Drug Designation accorded to OMS721 for IgA nephropathy (IgAN)	August 2017	Medium
Anticipated		
Initiation of enrollment in pivotal Phase 3 trial of OMS721 for HSCT-TMA	Late 2017	Medium
Initiation of enrollment in pivotal Phase 3 trial of OMS721 for IgAN	Late 2017 / Early 2018	Medium
Investigational New Drug (IND) filing for OMS527 (lead indication as yet undisclosed)	Early 2018	Low
Completion of enrollment of first 40 patients in pivotal Phase 3 trial of OMS721 for aHUS	Early to mid-2018	Medium
Interim analysis of pivotal Phase 3 trial of OMS721 in aHUS	**Mid-2018**	**High**
Initiation of enrollment in first-in-human (FIH) study of OMS527	Mid-2018	Low
Top-line data from Phase 2 trials of OMS824 in Huntington's disease and schizophrenia	**Mid- to late 2018**	**High**
Completion of enrollment in pivotal Phase 3 trial of OMS721 for HSCT-TMA	**Mid- to late 2018**	**High**
Completion of enrollment in pivotal Phase 3 trial of OMS721 for aHUS	**Early to mid-2019**	**High**
Completion of enrollment in FIH study of OMS527	Early 2019	Low
Top-line data from FIH study of OMS527	Mid-2019	Medium
Completion of enrollment in Phase 3 trial of OMS721 in IgAN	**Mid- to late 2019**	**High**
Final top-line data for OMS721 in pivotal trial for HSCT-TMA	**Late 2019**	**High**
Final top-line data for OMS721 in pivotal trial for aHUS	**Late 2019**	**High**
Final top-line data for OMS721 in pivotal trial for IgAN	**Late 2019 / Early 2020**	**High**
Submission of regulatory filing for OMS721 for HSCT-TMA and/or IgAN	**Mid-2020**	**High**
Regulatory approval of OMS721 for HSCT-TMA and/or IgAN	**Mid-2020**	**High**
Submission of regulatory filing for OMS721 for aHUS	**Late 2020**	**High**
Regulatory approval of OMS721 for aHUS	**Mid-2021**	**High**

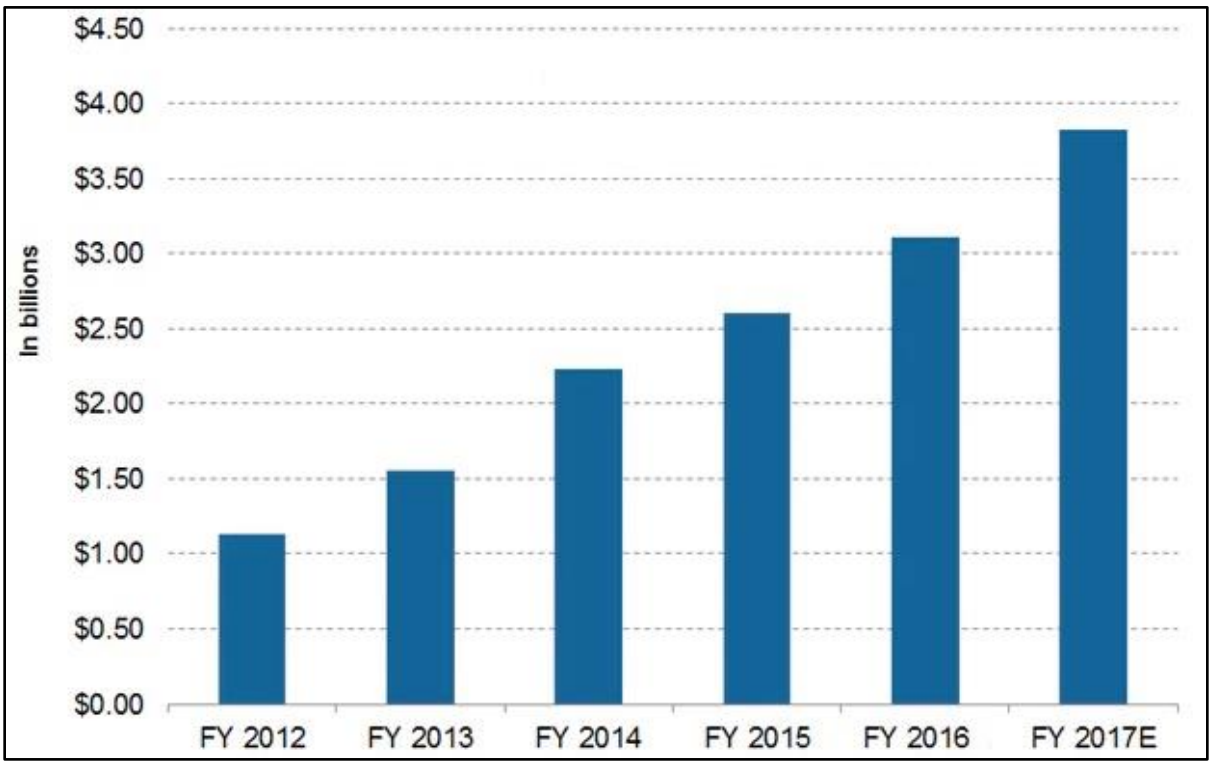
Source: Company reports and H.C. Wainwright & Co. estimates.

We feel that the market is giving Omeros insufficient credit for its sole marketed product, OMIDRIA, because of concerns over the potential timing of generic introductions and ongoing uncertainty regarding the speed with which the company could achieve further market penetration, as well as the viability of long-term reimbursement for the product. Investors should note, however, that the company recently reached a settlement with one potential generic entrant, Par Pharmaceutical, under which Par is slated to be enjoined from launching a generic version of OMIDRIA until April 1, 2032. Furthermore, we believe that the market is undervaluing the most advanced Omeros clinical-stage candidate, OMS721, because of a lack of understanding regarding the nature of the indications that OMS721 is targeting, the size of the commercial opportunities represented by these indications—even given the fact that these are niche markets—and the mechanistic data that demonstrate why OMS721 should be considered a risk-mitigated candidate. Finally, we believe the market does not currently ascribe any value to the company's earlier-stage pipeline, which includes a pair of Phase 2 assets (OMS824 and OMS405) and a late-stage preclinical agent (OMS527) that could enter clinical development early in 2018. OMS824 and OMS405 are both targeting CNS conditions that are widely considered areas of significant unmet medical need, while OMS527 could potentially be aimed at treating movement-related disorders, such as Parkinson's disease (PD), which have recently attracted a substantial amount of attention and also been the focus of several M&A transactions.

2. Potential best-in-class in the complement wars—an unwarranted valuation discrepancy

Investors should note that Omeros is competing against very few other companies in the complement activation pathway modulation space, and that one of these firms, the biotech bellwether Alexion Pharmaceuticals, currently has a marketed product called Soliris (eculizumab) for the treatment of paroxysmal nocturnal hemoglobinuria (PNH) and aHUS that generates over $3 billion in annual revenue despite being one of the most expensive drugs in the world and having only modest efficacy.

Figure 2: Soliris Revenue Trajectory Illustrates OMS721 Potential



Source: Market Realist, Alexion Pharmaceuticals SEC filings.

In our view, OMS721 could prove even more effective than Soliris in clinical testing because of the following key properties:

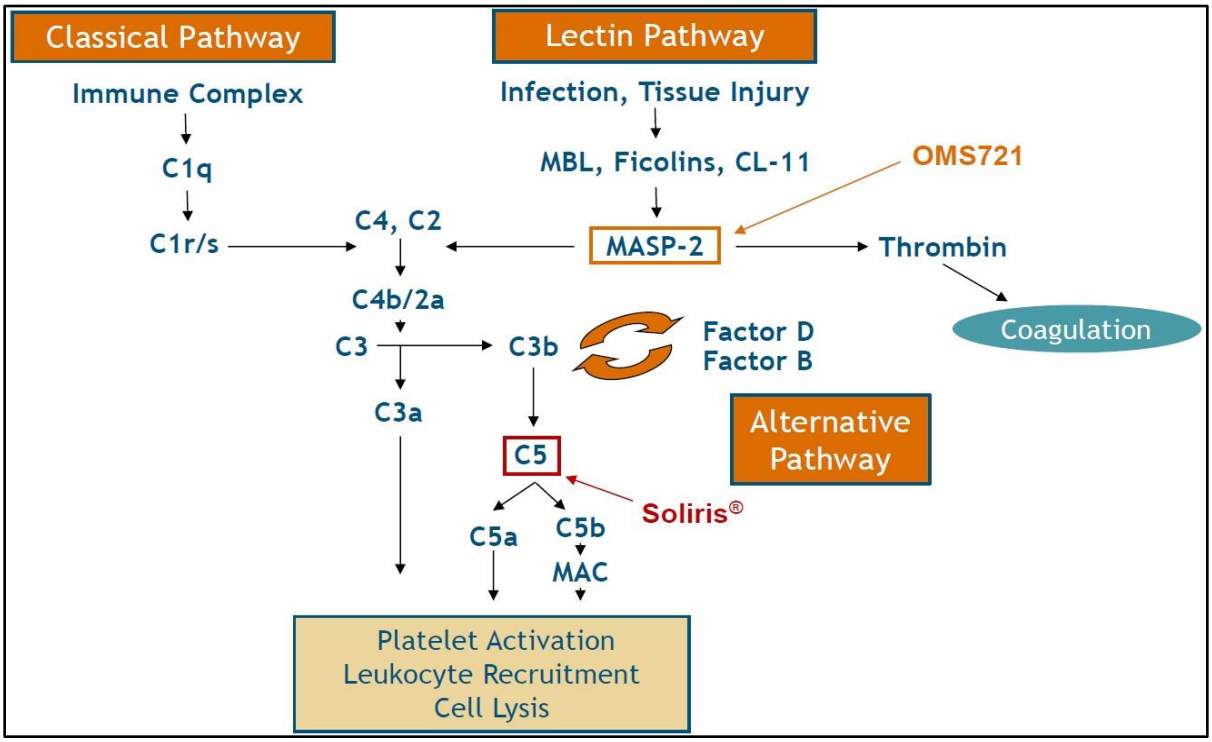
1) The ability to block complement activation without targeting the so-called “classical” pathway, which is critical for combating infection—this might enable OMS721 to demonstrate a superior safety profile to Soliris because its use may be associated with lower opportunistic infection risk;
2) Potential for administration via subcutaneous injection, enabling OMS721 to be delivered in an outpatient or at-home setting vs. Soliris, which requires intravenous infusion in a hospital setting;
3) Similar or even superior efficacy achieved with substantially lower dosing, due to the fact that the complement target for Soliris, the so-called C5 protein, is present in human plasma at 150-fold higher levels than MASP-2, the target for OMS721 (75 vs. 0.5 µg/ml)—this implies that the active dosage range for OMS721 could be in the 0.5 – 2µg/ml range vs. 100 – 200 µg/ml for Soliris. Such a difference in dosing could result in a significantly reduced cost of goods, allowing Omeros to position OMS721 as a lower-cost alternative to Soliris without sacrificing gross margins.

While we are not attempting to suggest that for the next few years Omeros should trade anywhere close to Alexion’s current valuation, which we believe is reflective of the lengthy commercial history for Soliris—which was originally launched as a treatment for PNH in 2007 in both the U.S. and Europe—as well as a nascent rare disease-focused commercial portfolio, we do believe that the valuation discrepancy between Omeros and Alexion should narrow somewhat in the mid- and long-term as Omeros continues to generate validating data in support of OMS721. Given Alexion’s current market cap of roughly $27 billion, we believe that in the mid-term the market cap of Omeros could

approach $2 billion and may reach roughly $3 billion upon the potential market entry of OMS721, which could occur in 2020. We also note that several other rare disease-focused companies that do not have any marketed products currently trade at valuations that are multiples of Omeros's current market capitalization. These include firms such as Amicus Therapeutics, with a market cap of $2.3 billion; AveXis, which has a market cap of $3.3 billion; bluebird bio, with a market cap of nearly $7 billion; Blueprint Medicines, with a market cap of $2.6 billion; Loxo Oncology, with a market cap of $2.6 billion; and Ultragenyx, with a market cap of roughly $2 billion.

The figure below demonstrates how Omeros could position itself as a best-in-class entrant in the complement wars, because of the unique mechanism of action that it targets. OMS721 represents a novel approach to modulating complement activation, since it avoids interfering with the so-called "classical" pathway involving proteins recruited to generate C3 convertase (C4bC2a), which cleaves the C3 protein. The C3b component of the cleaved C3 binds to C3 convertase (C4bC2a) to generate C5 convertase, which cleaves the C5 protein. The cleaved products attract phagocytes to the site of infection and tags target cells for elimination by phagocytosis. In addition, the C5 convertase initiates the terminal phase of the complement system, leading to assembly of the membrane attack complex (MAC), which creates a pore on the target cell's membrane, inducing cell lysis and death. While most of the other companies working on complement activation pathway-targeting drugs are solely focused on binding or inhibiting C5, Omeros specifically chose the MASP proteins as its target because these do not block the classical pathway and compromise the body's ability to fight infections.

Figure 3: OMS721 Modulates Complement Activation Uniquely, Avoiding Classical Pathway



Source: Omeros Corporation.

Investors should also note that Omeros has repeatedly pointed out how MASP protein-targeting drugs, including OMS721, could be applicable across a broad array of clinical indications. These include glomerulonephropathies (various kidney diseases), age-related macular degeneration (AMD), transplant, disseminated intravascular coagulation (DIC), traumatic brain injury, stroke, diabetic complications, and joint disease. Accordingly, while OMS721 could potentially constitute a pipeline in a single product, we have conservatively elected to project future sales in only the three indications for which Omeros is currently conducting active clinical development.

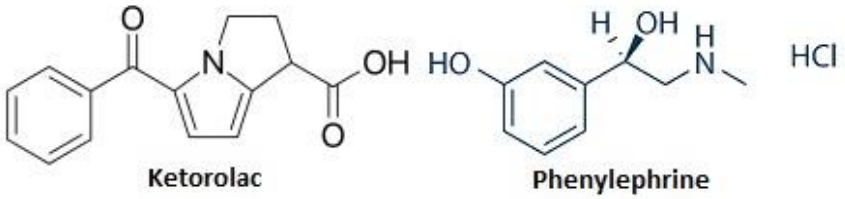
3. OMIDRIA: an underrated franchise with a longer commercial window than most expect.

OMIDRIA, a proprietary combination of ketorolac (anti-inflammatory) and phenylephrine (a mydriatic agent that functions via the adrenergic pathway to prevent pupil constriction), is added to the standard irrigation solution used in intraocular replacement surgeries and delivered intracamerally (inside the anterior segment of the eye) to maintain intraoperative mydriasis, prevent surgically induced miosis (excessive pupillary constriction), and reduce post-operative pain and irritation. Mydriasis—maintenance of pupil dilation—is essential for these procedures. If it is not maintained throughout the surgical procedure, the likelihood of adverse outcomes because of damage caused to intra-ocular tissue increases markedly. Moreover, miosis may extend the surgical procedure, leading to additional complications.

Ketorolac has been available for nearly three decades in the U.S. and is considered a broad-spectrum non-steroidal anti-inflammatory drug (NSAID) because of its ability to block prostaglandin production via inhibition of both COX-1 and COX-2 cyclooxygenase enzymes. It is a potent analgesic, and was the first NSAID approved in the U.S. for parenteral analgesic use. In addition, clinical trials involving more than 5,000 patients showed that ketorolac's analgesic efficacy was similar to that of parenteral opiates in patients undergoing general and oral surgery. Several hundred million injections of the product have been administered since it was first introduced into the U.S. in 1989. An eye drop formulation of ketorolac was first approved in 1992. Ketorolac tromethamine ophthalmic solution is indicated for the reduction of ocular pain and burning/stinging following cataract and refractive surgery. In this context, ketorolac has shown efficacy in reducing both pain and inflammation. The drug has also demonstrated effectiveness in treating cystoid macular edema, inhibits miosis and may prevent cystoid macular edema when used both pre- and post-operatively.

Phenylephrine is a powerful vasoconstrictor that can be employed to relieve nasal congestion, dilate the pupils, and maintain blood pressure during anesthesia. Phenylephrine is a mydriatic drug—these are classified as agents capable of increasing human pupillary diameter to approximately 5mm or larger. Topically applied, they are used to dilate the pupil in preparation for cataract surgery and intra-ocular lens (IOL) replacement surgeries. These are sympathomimetic drugs, which act directly or indirectly on the dilator muscle of the iris. Ophthalmic phenylephrine in strengths of 2.5% and 10% is used to dilate (enlarge) the pupil. It is used before eye examinations, before and after eye surgery, and to treat certain eye conditions. It is structurally similar to epinephrine and ephedrine, and causes little or no irritation, with primarily α-adrenergic effects. Phenylephrine acts locally within the eye to constrict ophthalmic blood vessels and the radial muscle of the iris. Phenylephrine hydrochloride ophthalmic solution is preferred as a mydriatic agent due to its rapid effect and moderate window of activity (well-suited to the surgical context), as well as to the fact that it produces no compensatory vasodilation. Local administration is also desirable, since systemic exposure to phenylephrine can cause elevated blood pressure and reflexive atropine-sensitive bradycardia (slowed heart rate).

Figure 4: Ketorolac and Phenylephrine Chemical Structures



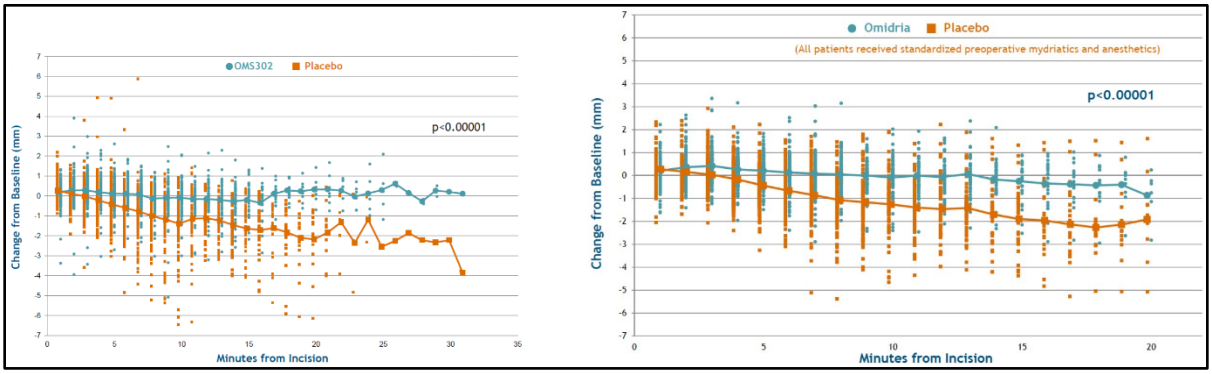
Source: Corporate presentations.

We provide this background to illustrate the tried and tested nature of both components of OMIDRIA, and why their combination makes sense. From our perspective, combinations of known agents—each with its own specific therapeutic mechanism—is a well-established product development strategy in the biopharmaceutical arena. We consider the OMIDRIA product profile favorable and attractive given its applicability in ophthalmology, which we view as a rapidly-growing segment of substantial size. Clinical development of OMIDRIA was also less expensive than would have been the case for chronic use-destined therapeutic agents, since OMIDRIA is designed only for surgical setting applications.

In July 2010, Omeros conducted a Phase 2b study of OMIDRIA (formerly known as OMS302) in patients undergoing cataract surgery. The parallel-design, double-blinded, multicenter trial included 221 patients randomized 1:1:1:1 across four arms. The first arm (n=55) received OMS302, the second arm (n=55) received only phenylephrine, the third arm (n=54) received only ketorolac and the fourth arm (n=57) received standard irrigation solution absent any active agent. The co-primary endpoints of the trial included maintenance of mydriasis (pupil dilation) and reduction in post-operative ocular pain. On an intent-to-treat (ITT) basis, the OMIDRIA group showed statistically significant mydriasis maintenance vs. both the ketorolac- (p<0.0001) and vehicle-treated (p<0.0001) groups. OMIDRIA also prevented clinically meaningful miosis when compared to each of the other three arms (p=0.0005 vs. ketorolac, p=0.0404 vs. phenylephrine and p<0.0001 vs. vehicle), again on an ITT basis. Furthermore, the OMIDRIA-treated group showed a statistically significant reduction in pain vs. both phenylephrine- (p=0.0089) and vehicle-treated (p=0.0418) groups. From our perspective, these data illustrate how each constituent of OMIDRIA furnishes a component of the drug's activity, with both phenylephrine and ketorolac additively providing intraoperative mydriasis and ketorolac alone responsible for the impact on post-operative pain. The drug was also seen to be well-tolerated.

In 2012, Omeros reported positive data from two pivotal Phase 3 trials of OMIDRIA in patients undergoing intraocular lens replacement surgery. These were multi-center, double-blinded, placebo-controlled trials that enrolled 405 and 416 patients, respectively, randomized 1:1 to receive drug or placebo. The primary endpoint in both studies was maintenance of intra-operative mydriasis, while the main secondary endpoint was reduction of post-operative ocular pain. The drug met the primary endpoint by showing statistically significant (p<0.00001) maintenance of intra-operative mydriasis in both studies. As shown below, progressive pupillary constriction occurred in placebo subjects, while OMIDRIA-treated patients maintained pupil dilation throughout the surgical procedure. The left panel depicts primary outcome data from the first pivotal trial and the right panel from the second study.

Figure 5: OMIDRIA Sustains Pupillary Diameter—Phase 3 Trial Primary Outcome Data



Source: Corporate presentations.

The effect of a decrease in pupil diameter by a very small objective amount (≥2.5mm) had a substantial impact on the effective size of the operative field. Operating with a <6mm pupil diameter significantly increases the risk of complications, such as posterior capsule tears, retained lens fragments and vitreous loss. A very small (1-3%) number of patients experienced reductions in pupil diameter ≥2.5mm in the treatment arms of the pivotal trials, vs. roughly 30% in the control group. OMIDRIA also showed statistical superiority versus placebo in reduction of ocular pain in the early post-operative period. When the two trials were combined, significantly more patients (26%) reported no post-operative pain and significantly fewer OMIDRIA-treated patients reported moderate-to-severe pain vs. placebo (17% and 14%). This was corroborated by the finding that more patients on placebo (35%) used analgesics on the day of surgery vs. individuals in the treatment groups (25%). In summation, therefore, we believe that the mid- to late-stage trial results for OMIDRIA paint a picture of a safe, well-tolerated and highly effective product, with meaningful impact from the viewpoint of ocular surgeons. While there are many unapproved combination ophthalmic solutions being used today, we believe that OMIDRIA's status as an FDA-authorized product with controlled clinical data and a reliable manufacturing process with appropriate quality control should make it a drug of choice.

In a July study 2015 published in *Clinical Ophthalmology*, Lola Elizabeth Lawuyi and Avinash Gurbaxani described OMIDRIA as a combination phenylephrine/ketorolac injection that "maintains intraoperative mydriasis, prevents miosis and reduces post-operative pain. When miosis develops during cataract surgery, it compromises the visualization of the surgical field and working space for surgeons, which may cause posterior capsular tear and associated vitreous loss, longer surgical time and post-operative inflammation."

Dr. James Loden, who runs three vision centers in Nashville, TN, has been quoted as saying that "we see eyes every day that start out well-dilated where the pupil constricts throughout the case; OMIDRIA keeps the pupil better dilated, which makes the surgery easier for me and makes the surgery go faster." While Dr. Loden uses OMIDRIA in its combined, injectable form—a 4-ml vial of OMIDRIA is mixed with a 500 cc-bottle of balanced salt solution—he says some surgeons have had success removing the medication from the bottle to insert into the anterior chamber, where normally they would use a lidocaine epinephrine mixture. Dr. Loden has also found Omidria to be "very synergistic" with triamcinolone/moxifloxacin (Tri-Moxi, Imprimis Pharmaceuticals) injections used in drop-less cataract surgery. He opined that "Tri-Moxi has an 8.5% breakthrough rate where the patient has to be started on drops; however, only one out of >70 patients that had Tri-Moxi with OMIDRIA needed breakthrough medications—a fairly big difference that is going to improve patient satisfaction."

Reimbursement for the drug, however, has been one of the principal issues since its introduction. Under the pass-through payment principle, the cost of OMIDRIA is reimbursed separately instead of being bundled with other supplies as part of the facility fee. For OMIDRIA, pass-through payment was originally implemented with a deadline of end-2017. Pass-through status has been described as a "honeymoon period", so surgeons can become acquainted with a new technology drug that took more than 10 years to develop and get FDA approved. This approach comes from the implementation of the Outpatient Prospective Payment System (OPPS) in the year 2000, as authorized by Congress in the Balanced Budget Act of 1997. Prior to OPPS, hospitals operated under a cost-based Medicare payment system, with dramatically different payment rates for cataract surgery from place to place. The Centers for Medicare and Medicaid Services (CMS) retired the cost-based system and instituted a prospective payment system and announced it was only going to give hospitals a $1,500 flat fee for cataract surgery. Efficient hospitals would keep the difference in Medicare payment and their costs, while inefficient hospitals would lose money on cataract surgery. In 2000, ambulatory surgery centers (ASC) already received reimbursement under a prospective payment system but much less than hospitals received for the same service. Subsequently, this also changed. The ASCs payment rates were linked to hospital rates in a 3:2 ratio (i.e., if the hospital received $3, the ASC received about $2). The new OPPS payment rates were derived from historical Medicare payments. However, those historical payments did not account for new products after 2000. Congress and CMS created the pass-through payments to handle new devices and drugs that were never accounted for in the OPPS payment rates. Under CMS pass-through regulation, outpatient hospitals and ambulatory surgery centers are reimbursed for the manufacturer's wholesale price of OMIDRIA (currently $465) plus a 6% handling fee subject to the usual deductible. Copayments apply to ASCs but not hospitals. This obscure regulation affects about 70% of all Medicare beneficiaries those that are covered by Part B Medicare. Those with Part C Medicare or Medicare Advantage are likewise covered, but the amount of payment (if any) is a matter for contract negotiation. The collective reimbursement for all drugs and devices subject to the pass-through payment is a tiny part of the Part B Medicare program—about 0.01% of the $500 billion Medicare budget.

After expiration of pass-through for OMIDRIA, a readjustment of the OPPS payment rate for cataract surgery is slated to occur, and separate payment for the product is slated to cease. While loss of pass-through status is slated to impact Medicare reimbursement for the product in 2018, we believe that two other options exist: the first option is average selling price (ASP) + 6%, while the second is to have two separate ambulatory payment classifications (APCs), one APC reimbursed at a higher rate than the other—the one with a higher rate would contain OMIDRIA and the other would be for a procedure in which OMIDRIA is not utilized. Despite the lack of formal clarity on the actual price per vial once pass-through status expires, we believe that the underlying demand for the product should be revealed as relatively strong if the price per vial were to drop significantly.

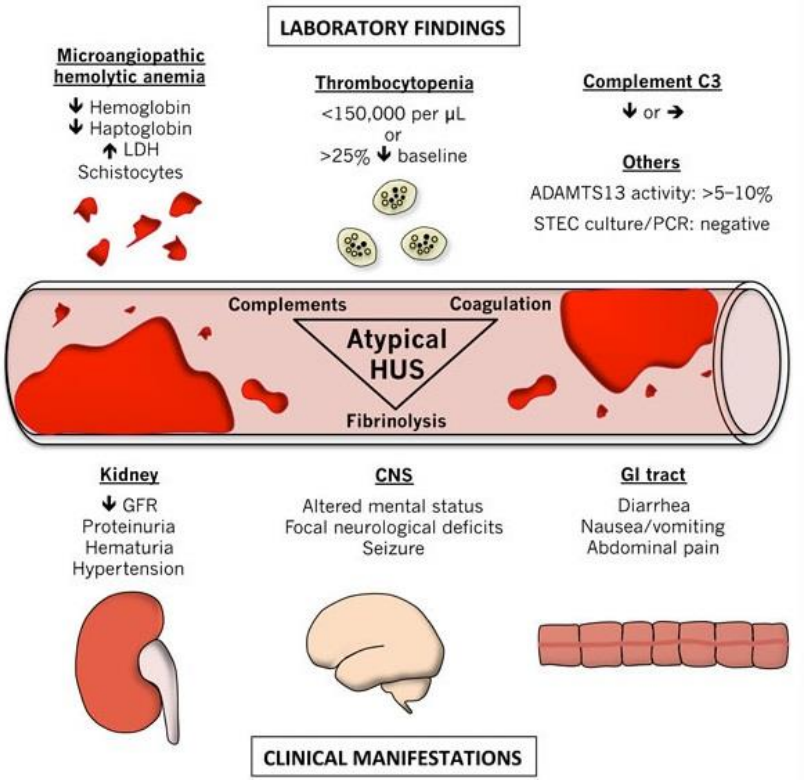
4. OMS721 may provide a best-in-class solution for complement activation-related disorders.

In our view, OMS721 could become a leading product in the field of treatment for complement activation-related disorders. We have chosen to focus in our assumptions on its applicability in aHUS, HSCT-TMA, and IgAN, although it may have potential in a broad array of other diseases, because these indications represent the areas in which the candidate is currently being tested clinically. We note that all of these disorders typically involve some degree of renal involvement, and that OMS721 could benefit from being positioned as a specialty drug aimed primarily at nephrologists.

OMS721 in aHUS

Atypical hemolytic-uremic syndrome (aHUS) is a systemic disease that primarily affects the kidneys. The rare condition is associated with genetic mutations or polymorphisms that result in chronic, uncontrolled activation of the alternative complement pathway. The dysregulated alternative complement pathway activation causes blood clots (thrombi) to form in the glomerular capillaries (groups of small blood vessels of the kidneys) and thickening of the glomerular capillary walls. Other possible changes include fibrinoid necrosis of the vessels that supply blood to the kidneys (afferent arterioles), mucoid intimal hyperplasia accompanied by narrowing of the vessel lumen, and fibrin deposits in the glomeruli and vessel walls. Hemolytic anemia and thrombocytopenia are typical with aHUS. The prevalence of aHUS is approximately two per million, and it accounts for an estimated 10% of aHUS diagnoses. Although many gene abnormalities have been implicated in the pathogenesis of aHUS, 40% of patients have no known mutation. The most prevalent mutation is CFH, which is identified in 25% to 30% of cases. Other more common mutations are CFI, MCP, and C3. Only 20% of mutations are inherited; the rest are acquired. Patients may have more than a single mutation. Environmental triggers also appear to play a role in aHUS, as those with mutations at birth often do not develop symptoms until adulthood. Onset of aHUS in middle-age is common. Respiratory and gastrointestinal infections, certain medications, chronic diseases, cancer, pregnancy, or other conditions are believed to provoke unregulated, continual activation of the complement system in 50% of genetically susceptible individuals. Diagnosis of aHUS involves the parameters illustrated below.

Figure 6: Atypical Hemolytic Uremic Syndrome (aHUS) Diagnosis Paradigm



Source: Asif et al., The Open Urology and Nephrology Journal 7: 91 – 94 (2014).

The current prognosis for patients with aHUS is poor. Patients whose aHUS is not diagnosed and treated within the first year of onset have a high risk of morbidity and mortality. One-half of patients with aHUS ultimately develop end-stage renal disease (ESRD), which occurs in approximately 65% of adults and 36% to 48% of children within three to five years after diagnosis. Prognosis appears to correlate with the genetic subtype of disease: Patients with an MCP mutation have the best outcomes, and patients with a CFH, CFI, or C3 mutation have poorer outcomes.

About 10 – 20% of patients have extra-renal manifestations. In these individuals, aHUS may affect the cardiovascular, gastrointestinal, or CNS. The most frequent extra-renal complications are neurological in nature, with studies suggesting that 16% of patients have neurologic involvement. Delayed treatment has been implicated in neurologic manifestations. We note that aHUS, classified as a type of major thrombotic microangiopathy (TMA), can be difficult to distinguish from other TMAs such as thrombotic thrombocytopenic purpura (TTP) and typical HUS due to the Shiga-like toxins produced by *Escherichia coli* (*E. coli*). Since these conditions need to be treated differently, conducting the appropriate tests is essential. Microbiological analysis can be used to rule out Shiga-associated HUS, and testing for thrombospondin type 1 motif, member 13 (ADAMTS13) activity can distinguish between TTP and aHUS. If ADAMTS13 activity is >10%, aHUS would be the diagnosis.

The Omeros Phase 1 clinical program for OMS721 involved the evaluation of safety, tolerability, pharmacodynamics and pharmacokinetics of OMS721 in healthy subjects. Notably, both intravenous and subcutaneous administration yields sustained lectin pathway (LP) inhibition. Omeros management have indicated that the maximum tolerated dose (MTD) for OMS721 has not yet been reached. The company's toxicology program supports chronic clinical administration via multiple routes for all potential indications. No adverse findings were identified in a chronic toxicology study in primates up to and including the highest doses administered, and there is FDA and EMA concurrence regarding OMS721's non-clinical development to date and findings from preclinical toxicology work.

The company's Phase 2 trial of OMS721 in thrombotic microangiopathies actually spans both aHUS and HSCT-TMA. This is an open-label, three-stage trial in adult patients with either condition. The objectives are to evaluate safety, tolerability, pharmacokinetics, pharmacodynamics, and clinical activity; the study is currently ongoing in the U.S., EU, and Asia Pacific regions. The design parameters shown below are delineated in Omeros's corporate presentations.

Stage 1 (dose escalation)
- → IV weekly x 4 weeks
- → Three cohorts (low-, mid-, and high-dose)

Stage 2 (cohort expansion)
- → aHUS cohort (12 weeks)
- → HSCT-TMA cohort (4 weeks)

Stage 3 (extended treatment)
- → aHUS cohort (12 weeks)
- → HSCT-TMA cohort (4 weeks)

Improvements in TMA markers (mean platelets, LDH and haptoglobin) were observed in aHUS patients enrolled in this trial. Three aHUS patients were able to discontinue dialysis. Three others on chronic dialysis were deemed eligible for renal transplant, with one successfully transplanted to date. OMS721 has been well-tolerated thus far, with a clean safety profile. The interim data from this open-label Phase 2 study were presented at the World Congress of Nephrology, Mexico City, in April 2017.

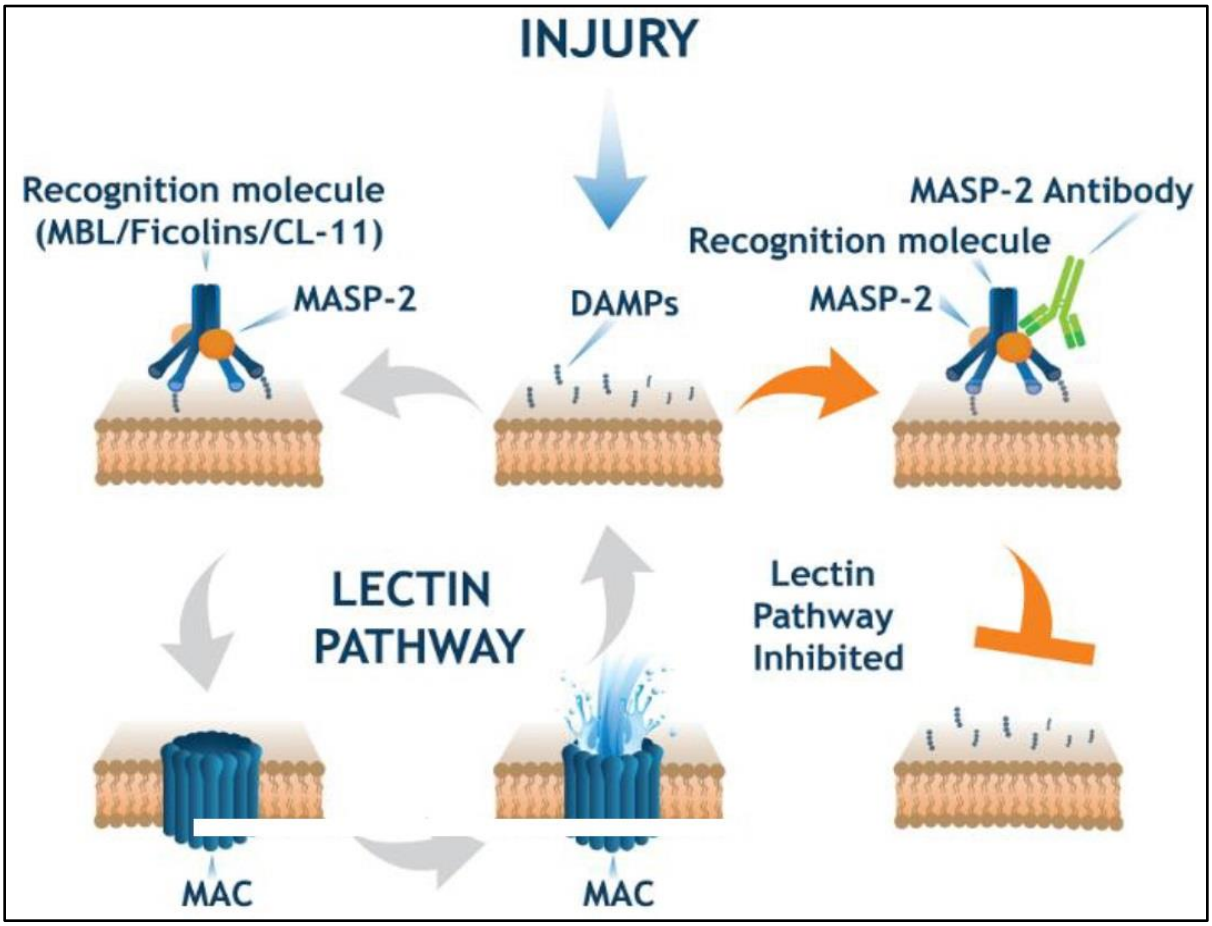
In March 2017, Omeros announced that it had formally initiated a Phase 3 trial in newly-diagnosed or ongoing aHUS. The company has also obtained Fast Track and Orphan Drug designations from the FDA for OMS721 in this indication. We note that Omeros has reached agreement with both the FDA and EMA on one single-arm (i.e., no control group), open-label trial to satisfy both agencies on filing requirements for OMS721 in aHUS. The clinical package for the envisaged Biologics License Application (BLA) is similar to that which formed basis of approval for Soliris. We believe that one of the most critical aspects of the concession package from the regulators is the acknowledgment that

safety can be demonstrated for OMS721 across a range of diseases, without Omeros having to show it specifically in aHUS patients alone. Since the disease is so rare and Soliris has already been approved to treat it, finding enough patients to establish an appropriate safety database with HUS sufferers alone would probably not have been feasible. We note that Omeros has indicated its intent to pursue accelerated approval of OMS721.

OMS721 in HSCT-TMA

As mentioned above, OMS721 entered clinical development for HSCT-TMA based on the incorporation of patients suffering from this condition into the open-label Phase 2 study that was originally intended to evaluate the drug's activity profile in patients with aHUS. HSCT-TMA is a serious and potentially fatal complication of HSCT caused by endothelial injury.[1] In 2015, more than 20,000 HSCT procedures were performed in the US and more than 42,000 were done in Europe. The incidence of HSCT-TMA has been reported to be as high as approximately 40%. Mortality can be >90% in severe cases. Survivors have increased risk of renal impairment and ESRD. No treatments are currently approved for HSCT-TMA, but eculizumab treatment has been reported to be effective.[2] As shown below, OMS721 is purported to have therapeutic potential in HSCT-TMA through blockade of the formation of the membrane attack complex (MAC), which prevents cell lysis and destruction.

Figure 7: OMS721 Proposed Mechanism of Action in HSCT-TMA



Source: Omeros Corporation.

Omeros has initiated a Phase 2 trial of OMS721 in HSCT-TMA, designed as a three-stage study. The first stage comprises a dose-escalation format, with the drug being administered intravenously on a weekly basis four times a week. Three cohorts (low-, mid- and high-dose) are involved.

[1] Jodele *et al*., Blood 122: 2003 – 2007 (2013)
[2] Brocklebank and Kavanagh. Clinical Kidney Journal 10: 600 – 624 (2017)

Improvements in TMA markers were observed in seven of nine HSCT-TMA patients in this trial to date. Two non-responders discontinued treatment after only a few weeks and were given palliative care. OMS721 was generally well-tolerated with a benign safety profile. Data were presented at the American Society for Blood and Marrow Transplantation (ASBMT) meeting held in Orlando, FL in February 2017. The company continues to enroll patients in the U.S., EU, and Asia Pacific.

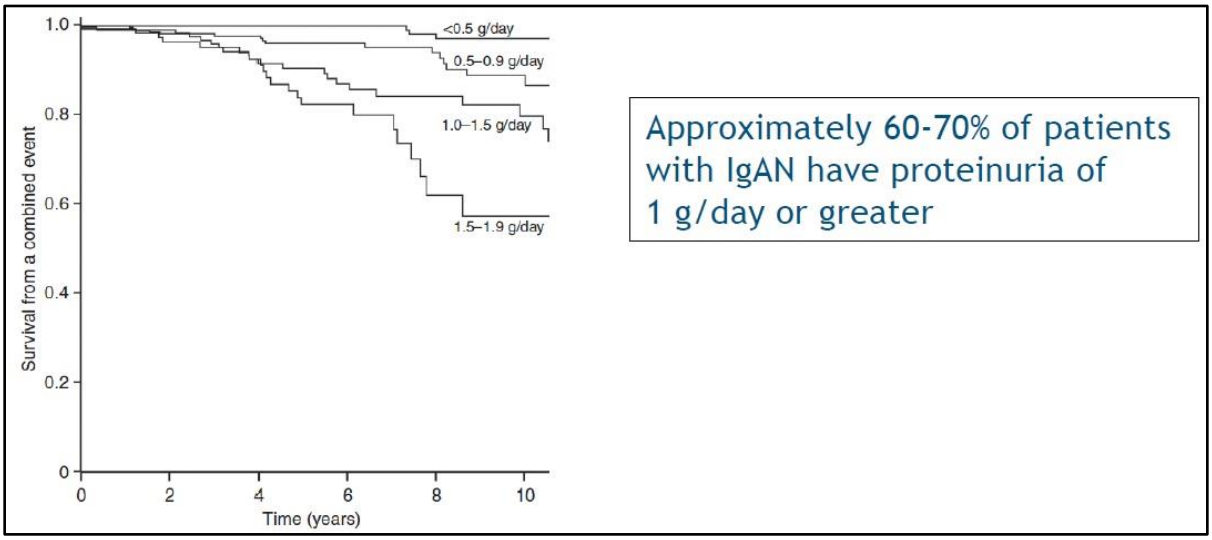
One compassionate use case report appears to be worth highlighting here. This involved a 15-year-old girl who was administered HSCT to treat Diamond-Blackfan anemia. She originally developed TMA and responded to eculizumab treatment. Subsequently, she developed pulmonary edema associated with eculizumab, which was then discontinued. The TMA relapsed and was retreated with low-dose eculizumab; however, this again resulted in pulmonary edema. The patient required daily platelet transfusions and thrice-weekly hemodialysis for several months. She was then treated with OMS721 three times a week and she discontinued dialysis after a few weeks of OMS721 treatment. The patient's platelets recovered; transfusions were tapered, then halted entirely. During the tapering of OMS721, the patient developed a viral infection that reactivated her HSCT-TMA; this was successfully treated with restoration of original OMS721 dose (evidence of retreatment effectiveness). This patient is currently doing well, having remained free of both dialysis and transfusions. From our perspective, this information—while anecdotal—points to some of the background regarding why Omeros management believes that HSCT-TMA could potentially leapfrog the HUS indication and also seems to justify the effort to advance OMS721 into a pivotal trial for this indication, with Breakthrough Therapy designation being sought for the drug specifically in HSCT-TMA. Investors should note that OMS721 already has Breakthrough Therapy status in IgAN.

OMS721 in IgAN (Berger's disease)

We believe that IgAN could be considered the largest opportunity for OMS721, simply because its prevalence is far higher than that of aHUS or HSCT-TMA. The condition is caused by deposition of galactose-deficient IgA1 molecules in the glomerulus causing inflammation and progressive kidney disease. IgAN is considered the most common form of primary glomerulonephritis and responsible for 10% of patients on dialysis. According to some estimates, approximately 150,000 – 180,000 people have IgAN in the U.S. alone. It is possible that the prevalence is even greater in Europe.

Up to 40% of patients with IgAN develop end-stage renal disease and require dialysis within 20 years of diagnosis. No therapies are currently approved to treat this disease. Notably, renal biopsies from IgAN sufferers demonstrate evidence of lectin pathway activation, providing some justification for the potential therapeutic applicability of OMS721 in this indication.

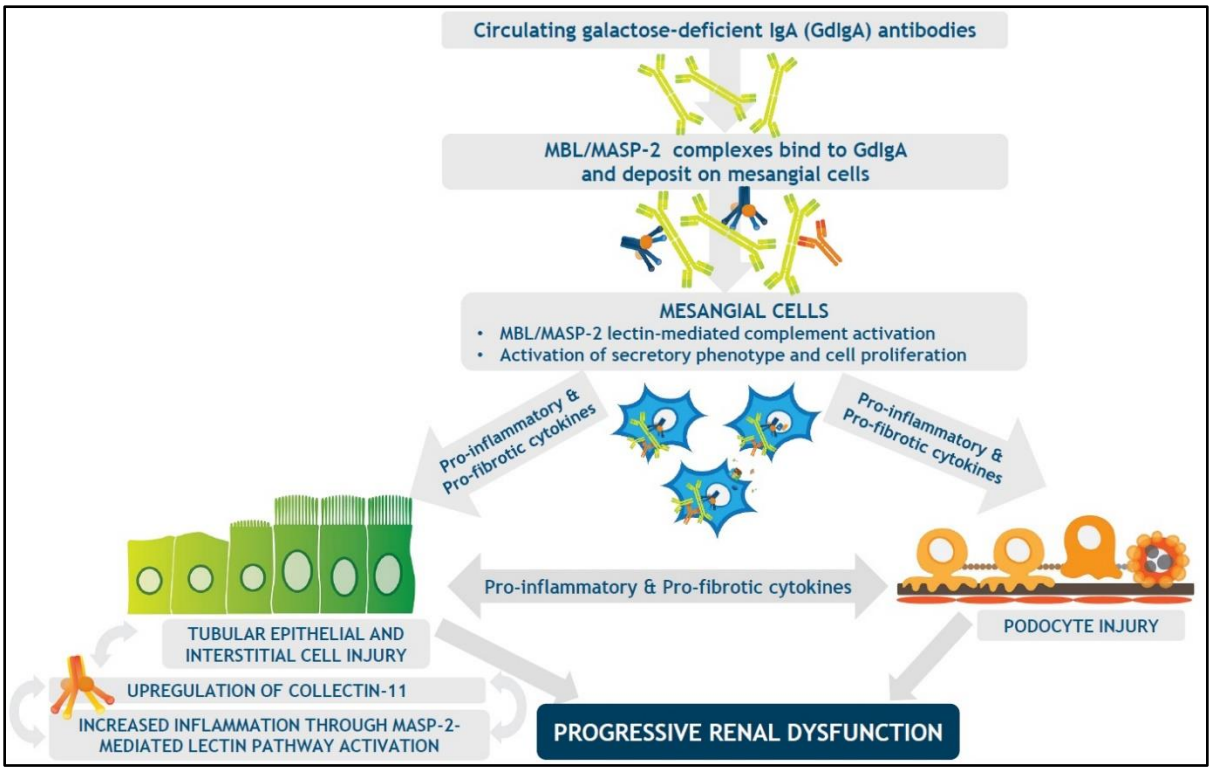
Figure 8: Proteinuria Levels Correlate with Poor Outcomes in Renal Disease Patients



Source: Cappo et al, Kidney International 86: 828 – 836 (2014).

From a histopathological perspective, IgAN seems to be a particularly intriguing target for OMS721. Predominance of IgA deposits (alone or with IgG/IgM) occurs within the glomerular mesangium of the kidneys in IgAN sufferers. Abnormally glycosylated IgA1 binds MBL and activates MASP-2, the target of OMS721. Complement C4, MBL, MASP-2, and the terminal complement complex (C5b–C9) are often detected in biopsies and are indicative of lectin pathway activation. MBL staining at a biopsy level is associated with more severe proteinuria. Patients with high urinary MBL levels have an unfavorable prognosis. Interestingly, the absence of C1q suggests that the classical pathway is not activated, which implies that agents targeting complement component 5 (C5)—e.g., Soliris—are not likely to demonstrate therapeutic efficacy in this disease.

Figure 9: Purported Mechanism of Action for OMS721 in IgAN



Source: Omeros Corporation.

Omeros is currently conducting a Phase 2 study of OMS721 in glomerulonephritis. The aim is to characterize the drug's safety and tolerability in patients with renal disease. Patients being enrolled into this trial are suffering from a range of kidney disorders, including:

- IgAN
- Membranous nephropathy
- Lupus nephritis
- C3 glomerulopathy

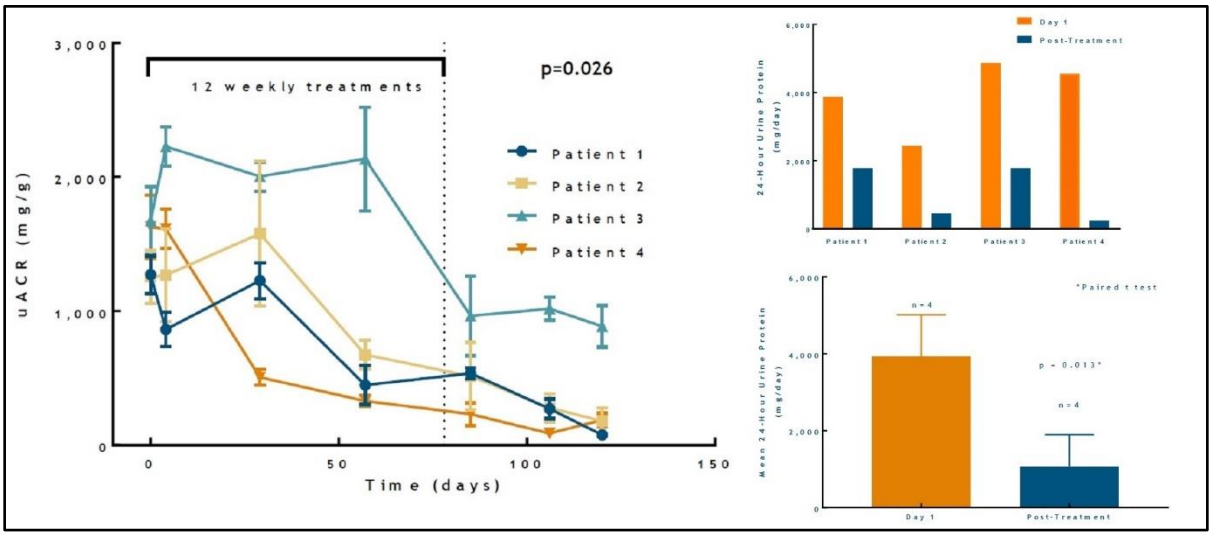
Patients were on steroids and RAS blockade. The study design is as follows:

- One-month run-in to establish baseline
- 12-week treatment period including corticosteroid tapering
- Six-week follow-up period
- Urine albumin/creatinine ratio (uACR) change from baseline by disease
- 24-hour urine protein change from baseline by disease
- Corticosteroid dose needed to maintain stable renal function by disease

Initial data from this study were presented at the ERA-EDTA Congress in Madrid, Spain in June 2017. The findings thus far from the Phase 2 glomerulonephritis trial can be summarized as follows:

- Clinically and statistically significant treatment effect in IgAN as assessed by both uACR and 24-hour urine protein
- Steroids completely eliminated for all patients
- Four of five lupus nephritis patients had massive—mean 69%—decrease in 24-hour urine protein
- The majority of patients were able to taper steroid dosing
- Mixed results were seen in membranous nephropathy, considered an inherently variable disease
- OMS721 was well-tolerated with a benign safety profile
- Positive post-trial follow-up data (up to approximately one year)
- Proteinuria in three of four patients remained reduced at 14%, 23% and 24% of pre-treatment baseline values
- Glomerular filtration rate (GFR) slightly improved in two patients, 57% increase in third
- Continuing enrollment of IgAN patients (no steroids) in double-blinded, vehicle-controlled cohort

Figure 10: OMS721 Initial Proof-of-Concept Trial Data—Clear Improvement in Kidney Function



Source: Company presentations.

Earlier this month, Omeros announced that extended follow-up data from patients with IgAN treated with OMS721 were presented at the American Society of Nephrology (ASN) Meeting in New Orleans, LA. These data describe up to one-year follow-up of patients treated with OMS721 in the ongoing glomerulonephropathy Phase 2 trial. During the Phase 2 clinical trial, which consisted of 12 weeks of treatment and 6 weeks of follow-up, all patients with IgAN demonstrated clinically meaningful improvement in proteinuria. The data were statistically significant. These data, which are limited to the duration of the clinical trial, were previously presented at the 54th European Renal Association-European Dialysis and Transplant Association (ERA-EDTA) Congress in June 2017. The additional data presented at the ASN meeting describe the course of these Phase 2 patients after the trial and while under standard care for up to approximately a year after the completion of OMS721 treatment.

As previously reported, all four IgA nephropathy patients demonstrated a substantial reduction in proteinuria during the clinical trial, including 24-hour urine protein levels and albumin/creatinine ratios (uACRs). In the extended follow-up after completion of the Phase 2 trial, urine protein/creatinine ratios (uPCR) were measured according to investigator practice standard. For purposes of *post-hoc* comparisons of proteinuria during and after the clinical trial, each post-trial uPCR value was converted to uACR.[3] Proteinuria reduction was maintained in three of the four patients. In these three patients, uACRs during extended follow-up remained decreased. Specifically, those three patients maintained partial remission relative to baseline during available follow-up (75.8%, 85.9%, and 76.8%

[3] Zhao *et al.*, Clinical Journal of the American Society of Nephrology 11: 947 – 955 (2016)

uACR decreases at 12, 11, and 3 months after cessation of OMS721 dosing, respectively). Following a substantial drop in uACR during dosing and trial follow-up, the fourth patient's uACR returned to approximately 88% of baseline at four months after the end of treatment with OMS721. Numerical improvement in estimated glomerular filtration rate (eGFR), a measure of renal function, was observed in three of the four patients after the trial. Post-treatment eGFR increases ranged from 7 to 17 mL/min/1.73 m^2 relative to screening values. The patient with the most severe loss of kidney function demonstrated eGFR improvement from 30 mL/min/1.73 m^2 to 47 mL/min/1.73 m^2, a 57% improvement. The fourth patient demonstrated stable eGFR relative to screening.

OMS721 was well-tolerated in the clinical trial with fatigue and anemia the most commonly reported adverse events. All adverse events were mild to moderate; some were considered possibly related but most were considered unrelated to OMS721. Following the clinical trial, all patients have remained well. In our view, the long-term safety of OMS721—albeit shown in the IgAN context for only a small cohort of subjects—is a particularly encouraging facet of the drug's overall profile; in our view, this bodes well for future testing in larger trials. There were no major adverse events (i.e., nothing Grade 3 or 4 in severity). Essentially, three of the four patients continued to show improvement in proteinuria even after the trial was over, long after OMS721 dosing had ended. Anemia and fatigue were some of the most commonly reported side effects, but were generally mild. In our view, from an efficacy perspective, the punchline is this: the OMS721 data from long-term follow-up in the IgAN indication looks quite impressive. As such, with no products currently on the market for IgA nephropathy, there appears to be an opportunity to obtain Orphan Drug exclusivity and possibly a pricing advantage.

We believe that the main items to bear watching going forward would be the primary and secondary endpoints in the clinical trial program that would be acceptable to the FDA for registration. The ultimate yardstick used to measure the efficacy profile of OMS721 is what could determine the eventual acceptance of the product among nephrologists and the total size of the market opportunity. While various other pivotal programs in nephropathy have utilized biomarkers as efficacy readouts, we believe that the FDA would be unlikely to give biomarkers priority over hard endpoints (e.g., proteinuria, progression to end-stage renal failure, and the like). Proteinuria reduction could potentially represent a reasonable primary endpoint in pivotal development for OMS721, which would permit a relatively rapid readout on the drug's efficacy to be obtained while also enabling the monitoring of long-term efficacy. As such, therefore, if OMS721 can demonstrate sustainable improvement in proteinuria as an outcome measure while continuing to show acceptable safety, we believe it would be considered approvable in IgAN and could achieve significant traction in this arena.

Finally, investors should be aware that Omeros has successfully obtained Breakthrough Therapy designation (BTD) and Orphan Drug designation from the FDA, which we consider to be very significant advances for OMS721. We note that OMS721 is the first IgAN investigational treatment to receive BTD. Also, based on the magnitude, rapidity, and persistence of proteinuria reduction in OMS721-treated patients, the FDA has indicated a willingness to consider proteinuria reduction as the primary endpoint for full approval of the drug. This would require fewer patients and a shorter study duration. Glomerular filtration rate (GFR) is slated to be a safety measure. Omeros is conducting ongoing discussions with regulatory agencies, and anticipates opening enrollment in the IgAN pivotal trial program before the end of this year.

We summarize OMS721's potential best-in-class properties below:

1) Safety: a potentially superior safety profile due to lower risk of opportunistic infections;
2) Efficacy: greater potency based on a higher degree of pathway specificity and the ability to target a complement mediator that is present in human plasma at lower concentrations than, for example, the target of Soliris;
3) Convenience: subcutaneous injection, which should permit administration in an outpatient or in-home setting, vs. Soliris, which requires intravenous (IV) infusion in a hospital setting.

5. Proven management with expertise in both commercialization and GPCR drug discovery.

We have known Omeros management for several years and consider the company's leadership to be defined by two core characteristics: dedication to high-quality science on multiple fronts, and determination to advance clinical programs even if the pathway is arduous and challenging. Dr. Greg Demopulos founded the company and has served as President, Chief Executive Officer and Chairman of the Board of Directors since June 1994. Prior to founding Omeros, Dr. Demopulos completed his residency in orthopedic surgery at Stanford University and his fellowship training in hand and microvascular surgery at Duke University. Dr. Demopulos currently serves on the board of trustees of the Smead Funds Trust, an open-end mutual fund company registered under the Investment Company Act of 1940, and on the board of directors of Onconome, Inc., a privately-held company developing biomarkers for early cancer detection. He received his M.D. from the Stanford University School of Medicine and his B.S. from Stanford University. We note that Dr. Demopulos appears to possess a rare combination of scientific acumen, medical expertise, and commercial nous. His extended tenure at the helm of Omeros, during which the company has gone from a discovery-stage GPCR platform-focused entity to a forward-integrated entity with a marketed product, a rich clinical-stage pipeline, multiple preclinical candidates and two powerful drug discovery platforms, constitutes an impressive track record. We believe Dr. Demopulos should be more than capable of leading Omeros through the next several value inflection points in the company's future.

Leonard M. Blum joined Omeros as the company's Chief Business and Commercial Officer in April 2016. He possesses nearly 30 years of executive and management experience in the pharmaceutical industry. Mr. Blum served as Senior Vice President and Chief Commercial Officer from 2007 until March 2016 at Theravance, Inc., a publicly-traded biopharmaceutical firm (now called Innoviva), and its spin-off Theravance BioPharma, also a publicly-traded biopharmaceutical firm. Prior to that, Mr. Blum founded and led the commercial functions at the biotech firm ICOS Corp.—ultimately as Senior Vice President of sales and marketing—from 2000 until the company's acquisition by Eli Lilly & Co. in 2007. ICOS and Lilly together brought Cialis, one of the world's leading erectile dysfunction drugs, to market globally; despite being a third-in-class agent, Cialis eventually topped $1 billion in annual sales. Mr. Blum began his career in the pharma industry at Merck & Co. Inc., where he spent thirteen years in positions of increasing responsibility in marketing and business unit leadership in the U.S. and Europe. During his tenure at Merck, Mr. Blum was responsible for over a dozen product launches in several therapeutic categories and markets, including multiple blockbuster drugs. Before beginning his career in the pharmaceutical industry, he served as an officer in the U.S. Army Special Forces.

Timi Edeki, M.D., Ph.D. joined Omeros as Vice President, Clinical in May 2017. Dr. Edeki is a pharmaceutical physician-scientist with experience in developing drugs in multiple therapeutic areas including nephrology, hematology, cardio-metabolic, immunology, neuroscience, gastroenterology, and oncology. From 2006 through 2016, he served as principal physician for AstraZeneca and during most of that period also served as Senior Director of R&D. Prior to AstraZeneca, Dr. Edeki was associate director at Abbott Laboratories (now AbbVie) from 2003 to 2006. Dr. Edeki is a fellow of the American College of Clinical Pharmacology and currently holds academic appointments as adjunct Professor of Pharmacology, Physiology, and Internal Medicine at Drexel University and previously as Clinical Professor of Medicine at the Chicago Medical School. In our view, his prior tenure in Big Pharma provides Omeros with valuable in-house expertise in drug development.

Catherine Melfi, Ph.D. joined Omeros as Vice President of Regulatory Affairs and Quality Systems in October 2012 and has served as the company's Chief Regulatory Officer since April 2016. Dr. Melfi previously worked from 1996 to 2012 at Eli Lilly & Co., where she held technical and leadership roles of increasing scope and responsibility, including as Senior Director and Scientific Director in Global Health Outcomes and Regulatory Affairs, respectively. Prior to joining Lilly, Dr. Melfi held various faculty and staff positions at Indiana University. In our view, the experience of individuals like Dr. Edeki and Dr. Melfi within established pharmaceutical firms provides Omeros with a degree of experience and knowhow that is rarely encountered among development-stage biotech companies, and should allow Omeros to continue advancing its proprietary pipeline candidates through the entire gamut of clinical studies necessary to reach regulatory submission.

6. Extensive and long-lived intellectual property portfolio protecting lead candidates

As of February 28, 2017, Omeros owned or held worldwide exclusive licenses to a total of 63 issued patents and 73 pending patent applications in the U.S. and 492 issued patents and 313 pending patent applications in foreign markets directed to therapeutic compositions and methods related to our development programs. We view the company's intellectual property (IP) estate as extensive, multi-faceted and long-lived, thus constituting an important asset to Omeros.

- OMIDRIA is covered by the company's PharmacoSurgery patent portfolio. The relevant patents and patent applications in this portfolio are directed to combinations of agents, generic and/or proprietary to Omeros or to others, drawn from therapeutic classes such as pain and inflammation inhibitory agents, mydriatic agents and agents that reduce intraocular pressure, delivered locally and intraoperatively to the site of ophthalmological procedures, including cataract and lens replacement surgery. Omeros owns six issued U.S. patents and four pending U.S. applications and 36 issued patents and 46 pending applications in foreign markets that are directed to OMIDRIA. The company's OMIDRIA patents have terms that are slated to expire as late as October 23, 2033 and—if pending claims are issued—as late as November 30, 2035.
- OMS721—the MASP-2 program—is covered by worldwide exclusive licenses to rights in connection with MASP-2, the antibodies targeting MASP-2 and therapeutic applications for those antibodies from the University of Leicester, MRC and Helion. Omeros thus exclusively controls 15 issued patents and 23 pending applications in the U.S., and 163 issued patents and 86 pending applications in foreign markets, related to the MASP-2 program.
- OMS906—MASP-3 program—is covered by IP that is controlled by Omeros under a license from the University of Leicester in the U.K., which covers all methods of treating various disorders and diseases by inhibiting MASP-3. The company exclusively controls four pending applications in the U.S. and 37 pending applications in foreign markets directed to these therapeutic methods.
- OMS824—the PDE10 program—is covered by eight issued patents and six pending applications in the U.S., and 22 issued patents and 41 pending applications in foreign markets.
- OMS405—the PPARγ agonist program—is covered by one issued patent and three pending patent applications in the U.S., and 25 issued patents and 19 pending patent applications in foreign markets, directed to the company's discoveries linking PPARγ and addictive disorders.
- OMS527—the PDE7 program—is covered two issued patents and one pending patent application in the U.S., and 21 issued patents and 12 pending applications in foreign markets directed to the company's discoveries linking PDE7 to movement disorders, as well as one issued patent and two pending applications in the U.S., and seven issued patents and 23 pending applications in foreign markets directed to the link between PDE7 and addiction and compulsive disorders.
- OMS616—the company's plasmin program—is covered by worldwide exclusive licenses to IP on a series of antifibrinolytic agents from The Regents of the University of California. This estate encompasses three issued patents and two pending applications in the U.S. and 29 issued and 11 pending applications in foreign markets.

Omeros owns seven issued patents and 13 pending applications in the U.S. and 52 issued patents and four pending applications directed to the following: (1) previously unknown links between specific molecular targets in the brain and a series of CNS disorders; (2) a proprietary cellular redistribution assay and other GPCR screening-related tools; and (3) orphan GPCRs and other GPCRs for which Omeros has identified functionally interacting compounds via its proprietary cellular redistribution assay. The Omeros antibody platform is covered by an IP estate based on worldwide exclusive license rights from the University of Washington to five issued patents and four pending patent applications in the U.S., and seven issued patents and 10 pending patent applications in foreign markets, directed to Omeros's antibody platform and antibodies generated using this platform. We note that, in addition to the above, Omeros holds IP on its OMS103 candidate via issued patents slated to expire as late as September 24, 2022 and pending claims that may, if granted, expire as late as August 3, 2032. OMS103 is covered by three issued U.S. patents and four pending U.S. patent applications, together with 32 issued patents and 13 pending patent applications in foreign markets. The company's OMS201 candidate is covered by IP expiring as late as July 16, 2029; this IP estate currently consists of one issued U.S. patent, two pending U.S. patent applications, and an additional 12 issued patents and one pending patent application in foreign markets.

7. Multiple other value drivers and technological advantages not factored into our valuation.

Investors should be aware that we are not currently ascribing any value to the extensive Omeros clinical-stage pipeline focused on CNS and neuropsychiatric disorders. Among these are the programs in Huntington's disease and schizophrenia with OMS824, which is slated to report data from two ongoing Phase 2 studies in 2018; OMS405, which was licensed in from the Japanese firm Asubio and which is being explored in opioid and nicotine addiction; and OMS527, which may enter clinical development in early 2018 for an indication yet to be selected but that is likely to be chosen from a range of addictive and compulsive behavioral disorders. In our view, the prudent and conservative route to take would be to assign value to these programs once clinical proof-of-concept efficacy data has been generated from one or more of the ongoing or envisaged clinical trials.

We would also draw investors' attention to the Omeros GPCR technology platform. The company's screening methodology is based on modulating GPCR localization, so that the unbound receptor is inside the cell and only localizes to the cell membrane and remains there after ligand binding has occurred. In this format, using the assay does not require secondary messenger monitoring, which was the standard approach in GPCR deorphanization conducted at most pharmaceutical companies. Furthermore, maintaining the bound GPCR receptor on the cell surface, while others are internalized, increases the signal-to-noise ratio for the desired target, further enabling the investigation of less common GPCRs. Utilizing this technique, Omeros can quickly screen GPCRs against a library of synthetic compounds, and potentially uncover GPCR ligands that could not have been discovered with traditional methods.

Omeros possesses a substantial library of compounds for running high-throughput screening (HTS) campaigns on GPCRs. The library comprises about 320,000 compounds, 20,000 of which are believed to be structurally oriented towards GPCR binding. Thus far, the company has screened over 50 orphan GPCRs. While we cannot definitively predict how likely Omeros would be to discover new drug-like compounds with novel pharmacology that could then be optimized as therapeutics, we believe that the company is able to operate in a sparsely populated space and focus on hitherto-undruggable targets, making its platform powerful and potentially valuable.

Downstream functional assays are also conducted in a differentiated manner within Omeros's R&D organization. Once the initial ligand for the orphan GPCR at hand has been pinpointed, Omeros confirms the GPCR-ligand interaction with more tests and advances the compound into functional studies using proprietary mouse mutant knockout models. To date, Omeros has generated mutant mouse strains for 60 different GPCRs. These models enable swift characterization of the biological significance of the desired GPCR receptor, and can utilize the newly discovered ligand to confirm the biological activity of the target receptor and its relevance to disease situations. Examples of early-stage GPCR-targeting programs currently in development at Omeros include: GPR17, which has been profiled as a possible stimulator of remyelination (e.g., with applicability in multiple sclerosis); GPR101, with potential for use in treating appetite and eating disorders; GPR151, with functionality that may be relevant to treatment of schizophrenia and certain cognitive impairment-related disorders; GPR161, aimed at triple-negative breast cancer (TNBC); GPR174, implicated in cytokine and regulatory T cell (T reg) modulation; and GPR183, for treatment of osteoporosis and Epstein-Barr virus (EBV)-related diseases. We do not currently give Omeros any credit for any of these programs.

Our DCF Valuation Suggests Significant Upside Potential vs. Current Levels

We have utilized a risk-adjusted net present value (rNPV) approach, which deploys a discounted cash flow (DCF) methodology to quantifying the value of the marketed product OMIDRIA and the late-stage candidate OMS721. Given the current absence of a local corporate tax rate in Washington state, we have utilized a 35% effective corporate tax rate, reflecting the statutory federal tax rate in the U.S. In our projections, we have utilized a 12% discount rate to future cash flows associated with OMIDRIA, which we consider reasonable given the well-established nature of the market and accepted use of mydriatic agents, and a 15% discount rate to those for OMS721 based on the uncertainty surrounding incidence and prevalence of some of the target indications. Conservatively, as we have previously stated, we do not assign any value to the company's clinical-stage pipeline beyond OMS721, nor do we ascribe value to the company from its platforms in GPCR-modulating compound or monoclonal antibody discovery. Our valuation methodology is summarized below.

Table 2: Risk-Adjusted NPV-Based Composite Valuation

OMS721 - Global	
Total patients suffering from aHUS, HSCT-TMA, and IgAN[1]	1MM
Patients seeking treatment[2]	690,000
Peak market share[3]	3.8%
Treatment revenue/prescription/course of therapy[4]	$75,000
Peak sales[5]	$1.3B
Launch[6]	2020
Peak sales year	2031
Protection expires[7]	2032
Discount rate	12%
Probability of success[8]	60%
Risk-adjusted NPV[9]	$900MM
NPV per share	$16.00
Additional value drivers (OMIDRIA)	$830MM
Total firm value	$1730MM
Shares Outstanding (MM; end-2018)	58MM
Present value-derived price target	$30.00

Notes on assumptions:

[1] Total patients with aHUS, HSCT-TMA, and IgAN - worldwide (only includes U.S. and European Union)
(Source: National Institute of Health, Rare Disease Report)

[2] Patients seeking therapy
(Source: H.C. Wainwright & Co. estimates)

[3] Peak market share - blended; factoring in competition from other complement inhibitors

[4] Revenue/year/patient - blended across all three indications

[5] Peak sales - treatment revenue/year x treated patients x peak market share

[6] Launch in 2020 (US) / 2021 (EU)

[7] Biologic drug exclusivity for 12 years in United States; 10 years as New Chemical Entity in Europe

[8] Probability of success - OMS721 is in Phase 2/3 testing (complement inhibition is a validated mechanism)

[9] Cash flow fully taxed at 35% (only federal tax as Washington state has no state corporate income tax)

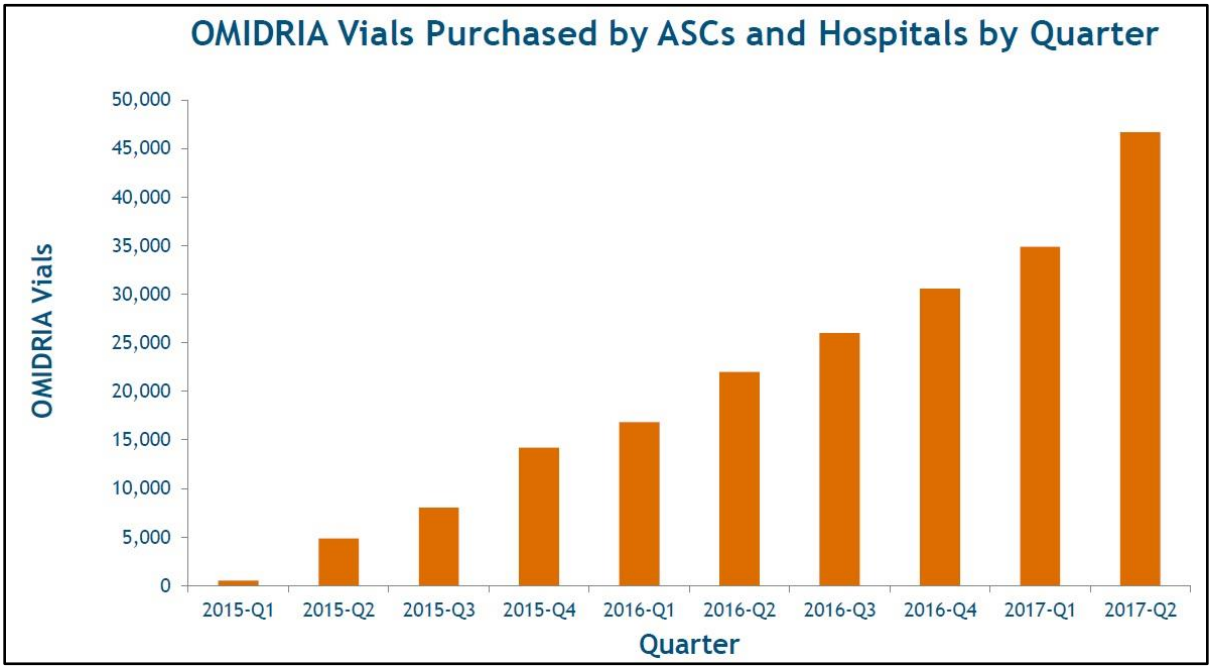
Source: Company reports and H.C. Wainwright & Co. estimates.

In the case of OMS721, we utilize a probability of success at 60% for the aHUS, HSCT-TMA, and IgAN indications, which we believe is conservative given prior data validating the mechanism of action and the fact that the drug is currently in pivotal development for aHUS and may begin pivotal testing near-term in the other two indications. The market presence of Soliris, an existing complement activation pathway-targeting product, for aHUS constitutes a further risk-mitigating factor, in our view. Conservatively, we have not projected OMS721 sales in any other diseases, nor have we modeled sales outside the U.S. and European markets for any of the three modeled indications. While we believe that Omeros could conceivably commercialize OMS721 in the U.S. independently across all three indications, given their specialty nature, we have modeled out-licensing of the drug in Europe and payment of tiered double-digit royalties on net sales to Omeros.

OMIDRIA Market Model

We have continued to model OMIDRIA sales solely in the ocular surgery setting, specifically in conjunction with cataract surgeries and intra-ocular lens implantation or replacement procedures. According to our estimates, there are roughly eight million of these candidate procedures occurring annually in the U.S. and Europe alone, with about four million in the U.S. The figure below depicts the sales trajectory for the drug since its launch in early 2015—following approval on May 30, 2014—in terms of the number of vials purchased on a quarterly basis by both ambulatory surgical centers (ASCs) and hospitals. The product's growth in terms of vial volume appears to be accelerating after a relatively slow start, which we believe should point to significantly increased adoption rates in the coming quarters as ocular surgeons have started to gain familiarity with the drug. We note that several surgical professionals with whom we have spoken indicated that OMIDRIA was their drug of choice to maintain pupillary dilation.

Figure 11: OMIDRIA Sales Trajectory Since Launch—Purchased Vial Volume



Source: Company reports.

Our market model assumes continued volume growth in 2018 and beyond, while the price per vial undergoes a substantial decrease next year based upon our assumption of loss of pass-through reimbursement. Originally, OMIDRIA was authorized for reimbursement at $465 per vial; however, pass-through status, which allows payment for new products on a temporary basis, typically runs for only two to three years to establish claims history. The pass-through price is first determined by the wholesale acquisition cost (WAC) plus 6%, then transitions to average selling price plus 6%. The Centers for Medicare and Medicaid Services (CMS) granted pass-through status for OMIDRIA in October 2014. Although we acknowledge that the price per vial is likely to go down starting next year, we also believe that vial volumes are likely to continue growing because of increased awareness and the potential for the lower cost to actually encourage broader uptake. Accordingly, we feel that market sentiment seemingly indicating a loss of confidence in OMIDRIA's durability as a franchise appears somewhat misplaced. Our model assumes reduction in OMIDRIA pricing in the U.S. from $465 per vial to $300 per vial, with 3% annual price increases thereafter to account for inflation. In Europe, we have assumed pricing at $200 per vial. We expect Omeros to partner the drug in Europe, receiving a flat 20% royalty on net sales. We continue to believe that OMIDRIA could show annual sales growth until at least 2023, when claims in the first patent family protecting the drug expires. Our peak annual sales assumption for the U.S. stands at roughly $380 million, while in Europe we believe the drug could achieve peak annual sales of over $150 million in the hands of a suitable partner.

Table 3: OMIDRIA® Market Model in Cataract and Intra-Ocular Lens (IOL) Replacement Surgery (2017 – 2035E)

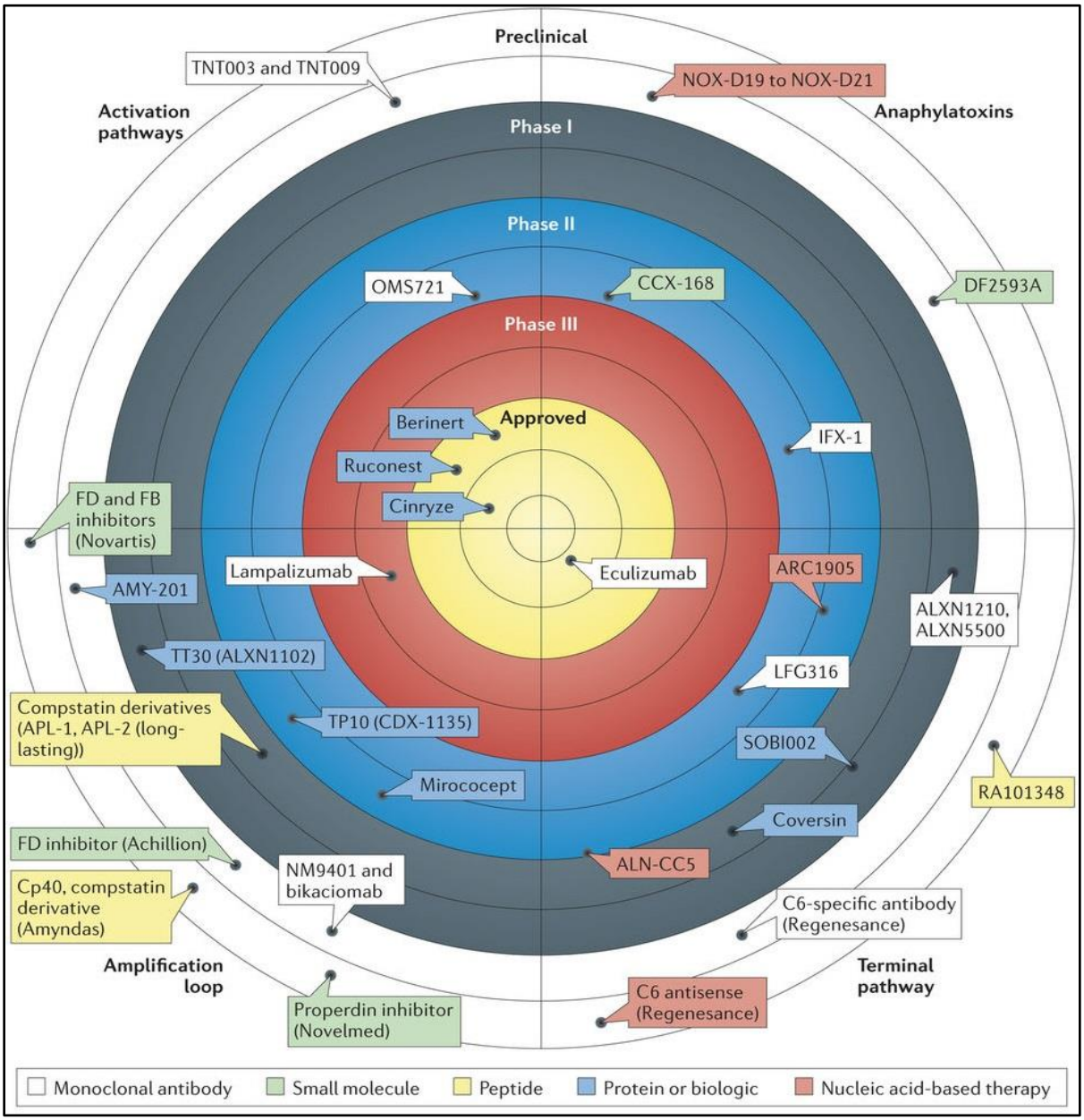
	2017	2018	2019	2020	2021	2022	2023	2024	2025	2026	2027	2028	2029	2030	2031	2032	2033	2034	2035
U.S. market																			
Total cataract surgery/IOL implantation procedures	4,040,000	4,161,200	4,286,036	4,414,617	4,547,056	4,683,467	4,823,971	4,968,690	5,117,751	5,271,284	5,429,422	5,592,305	5,760,074	5,932,876	6,110,862	6,294,188	6,483,014	6,677,504	6,877,830
Growth rate	*3.0%*	*3.0%*	*3.0%*	*3.0%*	*3.0%*	*3.0%*	*3.0%*	*3.0%*	*3.0%*	*3.0%*	*3.0%*	*3.0%*	*3.0%*	*3.0%*	*3.0%*	*3.0%*	*3.0%*	*3.0%*	*3.0%*
Penetration Rate	4%	7%	12%	16%	20%	24%	21%	17%	14%	12%	10%	9%	8.5%	7.8%	7.5%	6.5%	3.2%	2.1%	1.2%
Patients administered OMIDRIA	141,400	300,077	514,324	706,339	909,411	1,124,032	1,013,034	844,677	716,485	632,554	542,942	503,307	489,606	462,764	458,315	409,122	207,456	140,228	82,534
OMIDRIA cost per vial ($)	$465	$300	$309	$318	$328	$338	$348	$358	$369	$380	$391	$403	$415	$428	$440	$454	$467	$481	$496
U.S. OMIDRIA revenue ($MM)	**$66**	**$90**	**$159**	**$225**	**$298**	**$379**	**$352**	**$303**	**$264**	**$240**	**$212**	**$203**	**$203**	**$198**	**$202**	**$186**	**$97**	**$67**	**$41**
Europe market																			
Total cataract surgery/IOL implantation procedures	4,150,000	4,274,500	4,402,735	4,534,817	4,670,862	4,810,987	4,955,317	5,103,977	5,257,096	5,414,809	5,577,253	5,744,571	5,916,908	6,094,415	6,277,247	6,465,565	6,659,532	6,859,318	7,065,097
Growth rate	*3.0%*	*3.0%*	*3.0%*	*3.0%*	*3.0%*	*3.0%*	*3.0%*	*3.0%*	*3.0%*	*3.0%*	*3.0%*	*3.0%*	*3.0%*	*3.0%*	*3.0%*	*3.0%*	*3.0%*	*3.0%*	*3.0%*
Penetration %		1%	4%	7%	11%	15%	14%	12%	11%	9%	7%	5%	3%	2%	1.5%	1.2%	0.4%	0.2%	0.15%
Patients administered OMIDRIA		42,745	176,109	317,437	513,795	721,648	693,744	612,477	578,281	487,333	390,408	287,229	177,507	121,888	94,159	77,587	26,638	13,719	10,598
OMIDRIA cost per vial ($)		$200	$203	$206	$209	$212	$215	$219	$222	$225	$229	$232	$236	$239	$243	$246	$250	$254	$258
European OMIDRIA sales ($MM)	$0	$9	$36	$65	$107	$153	$149	$134	$128	$110	$89	$67	$42	$29	$23	$19	$7	$3	$3
Omeros Royalty Rate on OMIDRIA European sales	**20%**	**20%**	**20%**	**20%**	**20%**	**20%**	**20%**	**20%**	**20%**	**20%**	**20%**	**20%**	**20%**	**20%**	**20%**	**20%**	**20%**	**20%**	**20%**
Omeros Revenue on OMIDRIA European sales	**$0**	**$2**	**$7**	**$13**	**$21**	**$31**	**$30**	**$27**	**$26**	**$22**	**$18**	**$13**	**$8**	**$6**	**$5**	**$4**	**$1**	**$1**	**$1**
Total OMIDRIA Revenue to Omeros ($MM)	**$66**	**$92**	**$166**	**$238**	**$320**	**$410**	**$382**	**$329**	**$290**	**$262**	**$230**	**$216**	**$212**	**$204**	**$206**	**$189**	**$98**	**$68**	**$41**

Source: Company reports; Decision Resources; EvaluatePharma; H.C. Wainwright & Co. estimates.

Complement Activation Pathway Modulators—Background and Overview

We believe that it is important to profile the current status of the field as it pertains to complement activation pathway modulators. A great deal of drug development has been occurring in this arena over the course of the past decade. As shown in the figure below, multiple agents targeting different components of the complement activation cascade are currently in various stages of development. Although only a handful have actually succeeded in reaching the market so far, we believe that this arena is likely to become significantly more populated in the coming years.

Figure 12: Complement Activation Pathway Modulators



Source: Nature Reviews Drug Discovery (2016).

The table overleaf lists the drugs and drug candidates that we consider most directly comparable to and competitive with OMS721. However, investors should note that: (1) OMS721 is being developed in settings where most, if not all, of its direct competitors are not being targeted; and (2) OMS721 has advantages from a convenience standpoint (e.g., not requiring infusion or intra-vitreal injection).

Table 4: OMS721 Competitive Landscape

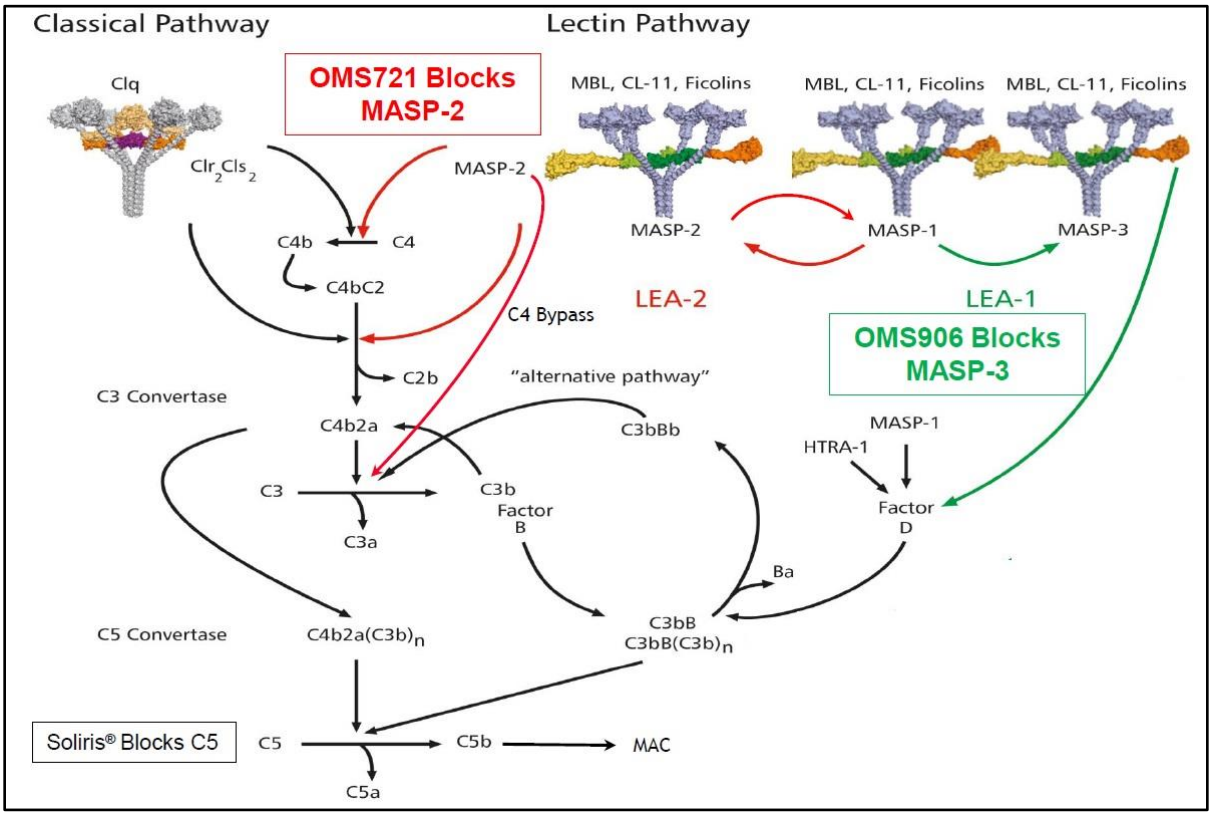
Trade Name	Generic Name	Sponsor	Indication	Mechanism of Action	Dosing Route	Development Stage	Approval Date
Soliris	eculizumab	Alexion Pharmaceuticals	Paroxysmal nocturnal hemoglobinuria (PNH) Atypical hemolytic uremic syndrome (aHUS)	Complement component 5 (C5) inhibitor	Intravenous infusion	Marketed	March 2007
ABP 959	eculizumab	Amgen	Paroxysmal nocturnal hemoglobinuria (PNH) Atypical hemolytic uremic syndrome (aHUS)	Complement component 5 (C5) inhibitor	Intravenous infusion	Pharmacokinetic Study	NA
RG-7417	lampalizumab	Roche/Genentech	Age-related macular degeneration (AMD)	Complement Factor D (CFD) binder	Intravitreal injection	Phase 3	NA
Zimura	avacincaptad pegol	Ophthotech	Age-related macular degeneration (AMD)	Complement component 5 (C5) inhibitor	Intravitreal injection	Phase 2/3	NA
OMS721	NA	Omeros Corporation	Atypical hemolytic uremic syndrome (aHUS) Thrombotic microangiopathy (TMA) IgA nephropathy (IgAN or Berger's disease)	MASP-2 inhibitor	Subcutaneous injection	Phase 2/3 Phase 2 Phase 2	NA
Coversin	NA	Akari Therapeutics	Paroxysmal nocturnal hemoglobinuria (PNH) Atypical hemolytic uremic syndrome (aHUS)	Complement component 5 (C5) inhibitor	Phase 2 (eculizumab resistant)	Phase 2 (eculizumab resistant)	NA
LFG316	NA	Novartis	Age-related macular degeneration (AMD) Choroiditis	Complement component 5 (C5) inhibitor	Intravitreal injection	Phase 2	NA
APL-2	NA	Apellis Pharmaceuticals	Age-related macular degeneration (AMD)	Complement component 3 (C3) inhibitor	Intravitreal injection	Phase 2	NA
IFX-1	NA	InflaRx	Hidradenitis suppurativa (HS) ANCA-associated vasculitis (AAV)	Complement component 5a (C5a) inhibitor	Intravenous injection	Phase 2 Phase 1	NA
ALN-CC5	cemdisiran	Alnylam Pharmaceuticals	Paroxysmal nocturnal hemoglobinuria (PNH)	Complement component 5 (C5) inhibitor	Subcutaneous injection	Phase 1/2	NA

[Publicly-traded companies mentioned in the above table comprise: Akari Therapeutics (AKTX; not rated); Alexion Pharmaceuticals (ALXN; not rated); Alnylam Pharmaceuticals (ALNY; not rated); Amgen (AMGN; not rated); Ophthotech (OPHT; not rated); Novartis AG (NVS; not rated); Roche Holding (RHHBY; not rated).]

Source: Company reports; ADIS R&D Insights; EvaluatePharma.

Omeros effectively owns the rights to real estate involving both lectin and alternative pathway inhibitors, with claims on modulation of activity on multiple members of the MASP protein sub-family. As shown below, the company has thus far discovered and characterized two molecules targeting the lectin pathway—OMS721, currently in mid- to late-stage clinical trials, and OMS906, which has not yet entered clinical development. OMS721 blocks MASP-2, while OMS906 blocks MASP-3.

Figure 13: Lectin Pathway Modulation of Complement Activation by MASP-2 and MASP-3



Source: Corporate presentations.

In our view, Omeros may be seeking to position OMS721 in rarer diseases, which may be indications for which the company could pursue commercialization independently (similar to the Alexion model), while OMS906—and/or small molecule inhibitors of MASP-3, on which Omeros is currently working as well—could be aimed at larger, non-orphan indications. The advantage of this strategy would be that the pricing leverage in smaller indications would not be weakened or destroyed by commercialization of a different drug targeting another component of the same pathway that is aimed at larger markets, for which premium pricing may not be feasible.

Preclinical data generated by Omeros with OMS906 has shown dose-dependent activity of the drug in reducing the incidence and severity of arthritis-like symptoms in the collagen antibody-induced arthritis (CAIA) mouse model, a canonical experimental system for assessing the activity of drugs that might be aimed at autoimmune conditions like rheumatoid arthritis (RA). OMS906 has also demonstrated the ability to improve red blood cell (RBC) or erythrocyte survival in a mouse model of PNH—something that a C5-targeting antibody (analogous to Soliris) was unable to achieve. OMS906 is able to exert this activity by blocking the initial opsonization step, in which C3b deposition on the surfaces of RBCs marks these cells for clearance. Anti-C5 antibodies can only block the complement pathway downstream of this stage, preventing them from being effective at inhibiting extravascular clearance. We believe that Omeros could potentially pursue development of MASP-3 inhibition strategies in both PNH and non-rare diseases, such as arthritis, multiple sclerosis (MS), age-related macular degeneration (AMD), and asthma. Going after PNH with an agent superior to Soliris might make Omeros a more urgent target for acquisition or licensing by a company like Alexion, in our view.

OMS721 Market Model

We have projected sales of OMS721 in only the three indications for which the drug is currently undergoing clinical development—namely, atypical hemolytic uremic syndrome (aHUS), hematopoietic stem cell transplant-associated thrombotic microangiopathies (HSCT-TMA), and IgA-associated nephropathy, also known as Berger's disease. Omeros is currently conducting a Phase 2/3 trial of OMS721 in aHUS, an 80-patient study that is slated to undergo an interim analysis based on the first 40 subjects enrolled. It is estimated that just one to four people per million have aHUS, implying that the global population of aHUS patients is only a few thousand individuals, including about 500 – 700 patients in America. We have projected the U.S. aHUS population starting at 650 individuals, which underscores the ultra-orphan nature of the disease. In patients with aHUS, genetic mutations cause an uncontrolled activation of the complement system that causes inflammation and leads to the formation of dangerous clots within the kidneys. Eventually, about 60% of aHUS patients suffer kidney damage or kidney failure. Soliris is the only FDA-approved therapy for aHUS, and it won accelerated FDA approval in 2011 after mid-stage trials showed that it improved kidney function and reduced disease activity. In one of two 26-week trials, the majority of aHUS patients on dialysis were able to discontinue dialysis treatment. In the second trial, the majority of patients on plasma therapy were similarly able to stop treatment. In 2015, a review of patients in Soliris trials showed that the benefits of Soliris treatment were maintained at a two-year follow-up point. Since Soliris improves kidney function and can reduce disease activity for a sustained period, it has become a standard aHUS treatment. We believe that, if OMS721 can eventually show improvement in kidney function, Omeros could quite easily position OMS721 as a lower-cost, more convenient (i.e., subcutaneous) alternative to Soliris, which must be administered via intravenous infusion. Since Soliris currently costs $537,000 per patient annually in the U.S., we have assumed that OMS721 for HUS could be priced in the $450,000 range in the U.S. market and $100,000 in Europe.

The HSCT-TMA indication has been projected to encompass roughly 35,000 individuals in the U.S. and 20,000 in Europe. We have assumed significantly higher annual growth for this disease, since we believe it to be under-diagnosed. While this is not an ultra-orphan disorder, we note that it still comfortably fits the canonical description of a rare condition. We have assumed that Omeros might actually be able to achieve regulatory approval for OMS721 in the HSCT-TMA indication slightly ahead of approval for the drug in aHUS, since patients are likely to be easier to recruit. We have assumed pricing starting at $75,000 in the U.S. and $40,000 overseas, with peak market penetration rates of roughly 20% in the U.S. and 8% in Europe.

Finally, patients with IgAN, or Berger's disease, are estimated to number over 100,000 in the U.S. alone according to data from the Rare Disease Report. We have assumed a starting population of 120,000 subjects in the U.S. and nearly 300,000 in Europe; while this indication would likely fit the definition of an orphan disease in the U.S., we are uncertain as to how it would be treated in Europe. However, we have projected pricing for this indication at only $30,000 in the U.S. and $15,000 in Europe on an annualized per-patient basis; accordingly, we consider our assumptions reasonable and reflective of the possibility that the condition may not be considered a classical orphan disorder. We have assumed very low market penetration rates for OMS721 in this condition, reaching only about 6% in the U.S. and 3.5% in Europe. These may turn out to be conservative, since OMS721 is currently the only complement activation pathway modulator in clinical testing for IgAN.

We have estimated that OMS721 could reach the market in both HSCT-TMA and IgAN in 2020, slightly ahead of its market entry for treatment of HUS. However, we believe that the drug could be branded differentially in each of the three indications, most likely using distinct routes of delivery and formulations, so as to avoid cannibalization of potential sales in the highest-priced indication by the products used to treat the broader populations. While we feel Omeros could independently commercialize the drug in the U.S., given the niche nature of the target markets, we expect the company to partner OMS721 in ex-U.S. territories. Conservatively, we have only modeled sales in the U.S. and Europe. Our European projections include the scenario in which Omeros receives tiered, double-digit royalties on net sales of the drug from a partner, with a 12 – 16% royalty range.

Table 5: OMS721 Market Model—Complement Pathway Activation-Related Disorders (2017 – 2035E)

	2017	2018	2019	2020	2021	2022	2023	2024	2025	2026	2027	2028	2029	2030	2031	2032	2033	2034	2035
U.S. market																			
Total patients with aHUS	650	657	663	670	676	683	690	697	704	711	718	725	732	740	747	755	762	770	777
Growth rate	*1%*	*1%*	*1%*	*1%*	*1%*	*1%*	*1%*	*1%*	*1%*	*1%*	*1%*	*1%*	*1%*	*1%*	*1%*	*1%*	*1%*	*1%*	*1%*
Total patients with HSCT-TMA	35,000	36,050	37,132	38,245	39,393	40,575	41,792	43,046	44,337	45,667	47,037	48,448	49,902	51,399	52,941	54,529	56,165	57,850	59,585
Growth rate	*3%*	*3%*	*3%*	*3%*	*3%*	*3%*	*3%*	*3%*	*3%*	*3%*	*3%*	*3%*	*3%*	*3%*	*3%*	*3%*	*3%*	*3%*	*3%*
Total patients with IgAN (Berger's disease)	120,000	123,600	127,308	131,127	135,061	139,113	143,286	147,585	152,012	156,573	161,270	166,108	171,091	176,224	181,511	186,956	192,565	198,342	204,292
Growth rate	*3%*	*3%*	*3%*	*3%*	*3%*	*3%*	*3%*	*3%*	*3%*	*3%*	*3%*	*3%*	*3%*	*3%*	*3%*	*3%*	*3%*	*3%*	*3%*
Penetration % - aHUS					3.5%	9.5%	18.9%	27.1%	36.8%	42.9%	47.6%	53.4%	57.8%	59.2%	61.1%	45.6%	37.4%	32.5%	25.8%
Penetration % - HSCT-TMA				0.5%	1.2%	3.5%	5.3%	8.2%	10.6%	12.7%	14.8%	16.4%	18.2%	19.5%	20.1%	17.2%	12.9%	8.5%	5.7%
Penetration % - IgAN				0.1%	0.7%	1.4%	2.1%	2.5%	2.9%	3.2%	3.7%	4.2%	4.7%	5.3%	5.8%	4.1%	2.3%	1.5%	1.2%
Patients on OMS721 - aHUS					24	65	130	189	259	305	342	387	423	438	457	344	285	250	201
Patients on OMS721 - HSCT-TMA				191	473	1,420	2,215	3,530	4,700	5,800	6,961	7,946	9,082	10,023	10,641	9,379	7,245	4,917	3,396
Patients on OMS721 - IgAN				131	945	1,948	3,009	3,690	4,408	5,010	5,967	6,977	8,041	9,340	10,528	7,665	4,429	2,975	2,452
Average cost per patient per year ($) - aHUS					$450,000	$463,500	$477,405	$491,727	$506,479	$521,673	$537,324	$553,443	$570,047	$587,148	$604,762	$622,905	$641,592	$660,840	$680,665
Average cost per patient per year ($) - HSCT-TMA				$75,000	$77,250	$79,568	$81,955	$84,413	$86,946	$89,554	$92,241	$95,008	$97,858	$100,794	$103,818	$106,932	$110,140	$113,444	$116,848
Average cost per patient per year ($) - IgAN				$30,000	$30,900	$31,827	$32,782	$33,765	$34,778	$35,822	$36,896	$38,003	$39,143	$40,317	$41,527	$42,773	$44,056	$45,378	$46,739
Total U.S. OMS721 sales ($MM)		**$0.0**	**$0.0**	**$18**	**$76**	**$205**	**$342**	**$515**	**$693**	**$858**	**$1,046**	**$1,234**	**$1,445**	**$1,644**	**$1,818**	**$1,545**	**$1,176**	**$858**	**$648**
Europe market																			
Total patients with aHUS	2,100	2,121	2,142	2,164	2,185	2,207	2,229	2,251	2,274	2,297	2,320	2,343	2,366	2,390	2,414	2,438	2,462	2,487	2,512
Growth rate	*1%*	*1%*	*1%*	*1%*	*1%*	*1%*	*1%*	*1%*	*1%*	*1%*	*1%*	*1%*	*1%*	*1%*	*1%*	*1%*	*1%*	*1%*	*1%*
Total patients with HSCT-TMA	20,000	20,600	21,218	21,855	22,510	23,185	23,881	24,597	25,335	26,095	26,878	27,685	28,515	29,371	30,252	31,159	32,094	33,057	34,049
Growth rate	*3%*	*3%*	*3%*	*3%*	*3%*	*3%*	*3%*	*3%*	*3%*	*3%*	*3%*	*3%*	*3%*	*3%*	*3%*	*3%*	*3%*	*3%*	*3%*
Total patients with IgAN (Berger's disease)	280,000	288,400	297,052	305,964	315,142	324,597	334,335	344,365	354,696	365,336	376,297	387,585	399,213	411,189	423,525	436,231	449,318	462,797	476,681
Growth rate	*3%*	*3%*	*3%*	*3%*	*3%*	*3%*	*3%*	*3%*	*3%*	*3%*	*3%*	*3%*	*3%*	*3%*	*3%*	*3%*	*3%*	*3%*	*3%*
Penetration % - aHUS						2.9%	7.5%	9.8%	11.9%	13.3%	15.8%	17.2%	18.5%	15%	10.6%	8.7%	6.3%	5.2%	4.4%
Penetration % - HSCT-TMA					0.3%	1.4%	2.1%	2.8%	3.6%	4.7%	5.5%	6.4%	7.6%	8.3%	3.5%	2.9%	2.2%	1.6%	1.1%
Penetration % - IgAN					0.1%	0.7%	1.2%	1.7%	2.4%	2.8%	3.1%	3.3%	3.5%	3.4%	0.8%	0.6%	0.5%	0.4%	0.2%
Patients on OMS721 - aHUS					-	64	167	221	271	305	367	403	438	358	256	212	155	129	111
Patients on OMS721 - HSCT-TMA		-	-	-	68	325	502	689	912	1,226	1,478	1,772	2,167	2,438	1,059	904	706	529	375
Patients on OMS721 - IgAN		-	-	-	315	2,272	4,012	5,854	8,513	10,229	11,665	12,790	13,972	13,980	3,388	2,617	2,247	1,851	953
Average cost per patient per year ($) - aHUS						$100,000	$103,000	$106,090	$109,273	$112,551	$115,927	$119,405	$122,987	$126,677	$130,477	$134,392	$138,423	$142,576	$146,853
Average cost per patient per year ($) - HSCT-TMA					$40,000	$41,200	$42,436	$43,709	$45,020	$46,371	$47,762	$49,195	$50,671	$52,191	$53,757	$55,369	$57,030	$58,741	$60,504
Average cost per patient per year ($) - IgAN					$15,000	$15,450	$15,914	$16,391	$16,883	$17,389	$17,911	$18,448	$19,002	$19,572	$20,159	$20,764	$21,386	$22,028	$22,689
Total Europe OMS721 sales ($MM)		**$0**	**$0**	**$0**	**$7**	**$55**	**$102**	**$149**	**$214**	**$269**	**$322**	**$371**	**$429**	**$446**	**$159**	**$133**	**$110**	**$90**	**$61**
Omeros Royalty on OMS721 European sales					**12%**	**13%**	**14%**	**14%**	**15%**	**15%**	**16%**	**16%**	**16%**	**16%**	**16%**	**16%**	**16%**	**16%**	**16%**
Royalty-Based Revenue on OMS721 in Europe ($MM)		**$0**	**$0**	**$0**	**$1**	**$7**	**$14**	**$21**	**$32**	**$40**	**$52**	**$59**	**$69**	**$71**	**$25**	**$21**	**$18**	**$14**	**$10**
Total OMS721-Derived Revenue to Omeros ($MM)		**$0**	**$0**	**$18**	**$77**	**$212**	**$357**	**$536**	**$725**	**$898**	**$1,097**	**$1,294**	**$1,514**	**$1,715**	**$1,843**	**$1,566**	**$1,194**	**$873**	**$658**

Source: EvaluatePharma; H.C. Wainwright & Co. estimates.

Investment Risks

Financial outlook. Omeros Corporation has incurred operating losses since its inception and may not achieve profitability for several years, in our view. Although the firm has been able to obtain capital in order to fund its operations, it is not known if Omeros would be able to continue this practice or be able to obtain other types of financing to meet its operating needs. We estimate that the firm would need to raise additional capital to complete the clinical development of candidates like OMS721, OMS824 and OMS405, as well as support the commercialization of these drugs. We have assumed that Omeros would seek to commercialize OMS721 independently in the U.S. and via partnerships in ex-U.S. territories. In our view, therefore, given the potential risks associated with dilution from future financing rounds, investors should recognize that Omeros shares may be subject to above-average risk and may experience excessive price volatility.

Clinical development risk. New therapeutics development is a multi-year process that requires human clinical trials prior to approval by the U.S. Food and Drug Administration (FDA) and other regulatory agencies, such as the European Medicines Agency (EMA). Clinical trials may face delays in obtaining the necessary regulatory permission, initiation of patient enrollment, achievement of target enrollment, and indeed in yielding favorable efficacy data. If the company's clinical programs with OMS721, OMS824 and OMS405 fail, the firm's innate value could be substantially impaired.

Regulatory risk. The process of obtaining regulatory approval from the FDA and other regulatory agencies, such as the EMA, is typically arduous and complex. Omeros seeks approval from U.S., European and other regulatory agencies for development, testing, manufacturing, labeling, storage, and sale of its products. These regulations are demanding and may change from time to time. There can be no guarantee that, even if Omeros were to successfully complete clinical trials in its chosen target disease indications, its product candidates would be granted regulatory approval. Without such permission, Omeros would not be able to successfully partner or self-commercialize its candidates.

Potential partnership dependency risk. OMS721 constitutes a product opportunity that could potentially be commercialized by Omeros independently, in our view, given the focused nature of the target indications. In our view, Omeros is likely to pursue external licensing arrangements for the commercialization of OMS721 outside the United States, since establishing commercial infrastructure globally is likely to be a challenge. However, the company could also seek a global partner to launch OMS721 in all markets, including the U.S. For OMS824, Omeros may elect to commercialize the drug via global partnerships; even though Huntington's disease is an orphan indication, other target markets like schizophrenia would be impossible to penetrate without an established partner. If Omeros elects to partner OMS721 globally, for example, its licensee would be responsible for commercialization. Market penetration would therefore be dependent on the licensee's commitment to timely clinical development and product rollout. While we believe that an established pharmaceutical firm—particularly one with a pre-existing commitment to and expertise in areas like complement pathway activation-related disorders, hematological diseases and CNS conditions—might contribute meaningfully to the commercial optimization of the company's products, a lack of commitment could also potentially hinder the company's ability to realize favorable economics from the partnering of its core assets.

Competitive landscape. Major competitors to Omeros include well-established companies such as Alexion Pharmaceuticals, Allergan plc, AstraZeneca, GlaxoSmithKline, Johnson & Johnson, Pfizer and Takeda. Firms active in the GPCR space also include smaller entities, such as Confo Therapeutics, Dimerix Ltd., and Heptares Therapeutics. We anticipate that Omeros could face competition from other companies targeting the complement activation pathway, such as Akari Therapeutics. There are also entities developing biosimilar competitors to Alexion's branded Soliris product, which might pose commercial challenges to OMS721 if and when it is commercialized. The most notable of these is Amgen, with its ABP 959 candidate. Alexion itself is also developing a longer-acting version of the Soliris antibody. Some of the potential competitors cited above are large, well-financed and experienced pharmaceutical and biotechnology firms or have been partnered with such entities, which may give them development, regulatory and marketing advantages over Omeros.

Intellectual property risk. Omeros relies on patents and trade secrets to protect its products from competition. The pharmaceutical industry is litigious, and lawsuits are considered a normal part of doing business. A court might not uphold the company's intellectual property rights, or it could find that Omeros infringed upon another party's property rights. In addition, generics firms could potentially find loopholes in Omeros's intellectual property estate, which might enable them to launch generic versions of the company's branded drugs prior to the expiration of patent protection on these products.

Reimbursement risk. Following the institutionalization of broad-based healthcare reform policy, reimbursement agencies have grown warier of systematically reimbursing for drugs that are either unnecessary or provide marginal benefit at an increased cost. If Medicare spending growth continues to outpace GDP growth, and the government's ability to fund healthcare becomes impaired, changes could be made to policy that would negatively affect the firm's business.

Industry risk. Emerging biotechnology and pharmaceuticals stocks are inheritably volatile and increasingly subject to development and regulatory risk. Meeting or missing commercialization milestones may result in a significant change in the perception of the company and its stock price. We do not expect volatility to subside near term.

For additional risk considerations, please refer to the company's SEC filings.

Table 6: Omeros Corporation (OMER)—Historical Income Statements, Financial Projections

FY end December 31
$ in thousands, except per share data

	2016A					2017E					
	1QA	2QA	3QA	4QA	2016A	1QA	2QA	3QE	4QE	2017E	2018E
Revenue											
Product revenue	7,246	10,004	11,289	12,905	41,444	12,257	17,151	17,500	18,900	65,808	90,000
Grant revenue	173	-	-	-	173	-	-	-	-	-	-
Royalties	-	-	-	-	-	-	-	-	-	-	1,710
Total revenue	7,419	10,004	11,289	12,905	41,617	12,257	17,151	17,500	18,900	65,808	91,710
Expenses											
Cost of product and service revenue	327	327	378	380	1,412	271	157	158	208	793	1,526
Research & development	15,434	10,231	12,492	12,542	50,699	12,240	13,137	13,500	13,800	52,677	58,300
Selling, general and administrative	11,110	10,375	10,457	11,840	43,782	12,471	15,796	16,500	17,000	61,767	70,600
Total expenses	26,871	20,933	23,327	24,762	95,893	24,982	29,090	30,158	31,008	115,237	130,426
Gain (loss) from operations	(19,452)	(10,929)	(12,038)	(11,857)	(54,276)	(12,725)	(11,939)	(12,658)	(12,108)	(49,429)	(38,716)
Other income/expense											
Interest income/expense	(1,375)	(1,857)	(2,135)	(2,452)	(7,819)	(2,663)	(2,723)	(2,750)	(2,780)	(10,916)	(8,420)
Loss on early extinguishment of debt	-	-	-	(5,595)	(5,595)	-	-	-	-	-	-
Other income/expense	288	174	211	272	945	299	303	300	300	1,202	1,200
Total investment income and other	(1,087)	(1,683)	(1,924)	(7,775)	(12,469)	(2,364)	(2,420)	(2,450)	(2,480)	(9,714)	(7,220)
Loss before provision for income taxes	(20,539)	(12,612)	(13,962)	(19,632)	(66,745)	(15,089)	(14,359)	(15,108)	(14,588)	(59,143)	(45,936)
Net loss/income	(20,539)	(12,612)	(13,962)	(19,632)	(66,745)	(15,089)	(14,359)	(15,108)	(14,588)	(59,143)	(45,936)
Net loss per share (basic)	(0.54)	(0.32)	(0.34)	(0.45)	(1.65)	(0.34)	(0.33)	(0.33)	(0.31)	(1.30)	(0.96)
Net loss per share (diluted)	(0.54)	(0.32)	(0.34)	(0.45)	(1.65)	(0.34)	(0.33)	(0.33)	(0.31)	(1.30)	(0.96)
Weighted average number of shares outstanding (basic)	38,317	39,179	41,059	43,231	40,446	43,829	44,037	45,973	47,523	45,340	47,648
Weighted average number of shares outstanding (diluted)	38,317	39,179	41,059	43,231	40,446	43,829	44,037	45,973	47,523	45,340	47,648

Source: Company reports and H.C. Wainwright & Co. estimates.

Table 7: Omeros Corporation (OMER)—Historical Balance Sheets, Financial Projections

FY end December 31
$ in thousands, except per share data

	2016A					2017E					
	3/31	6/30	9/30	12/31	12/31/16A	3/31A	6/30A	9/30	12/31	12/31/17E	12/31/18E
Assets											
Current assets:											
Cash and cash equivalents	978	4,589	10,297	2,224	2,224	1,282	3,386	55,517	44,398	44,398	14,739
Short-term investments	12,266	16,648	37,144	43,107	43,107	32,371	26,281	26,281	26,281	26,281	26,281
Restricted cash	-	-	-	-	-	-	-	-	-	-	-
Accounts receivable	6,646	8,806	10,457	12,037	12,037	13,481	19,075	19,075	19,075	19,075	19,075
Inventories	1,972	1,780	1,396	1,128	1,128	960	905	905	905	905	905
Other assets and prepaid expenses	2,460	2,282	1,355	1,766	1,766	3,260	3,446	3,446	3,446	3,446	3,446
Total current assets	24,322	34,105	60,649	60,262	60,262	51,354	53,093	105,224	94,105	94,105	64,446
Property and equipment	898	1,251	1,206	1,181	1,181	1,215	1,426	1,357	1,288	1,288	1,012
Intangible assets	-	-	-	-	-	-	-	-	-	-	-
Restricted cash	10,679	10,679	10,835	5,835	5,835	5,835	5,835	5,835	5,835	5,835	5,835
Other assets	73	73	73	-	-	-	-	-	-	-	-
Total Assets	35,972	46,108	72,763	67,278	67,278	58,404	60,354	112,416	101,228	101,228	71,293
Liabilities and shareholder equity											
Current liabilities											
Accounts payable	5,332	5,420	3,985	2,519	2,519	2,409	2,572	2,572	2,572	2,572	2,572
Accrued expenses	12,120	10,452	11,882	13,354	13,354	14,336	21,643	21,643	21,643	21,643	21,643
Deferred revenue	-	-	-	-	-	-	-	-	-	-	-
Current portion of notes payable	69	193	190	198	198	-	-	-	-	-	-
Current portion of deferred rent	-	-	-	-	-	-	288	288	288	288	288
Current portion of lease financing obligations	-	-	-	-	-	228	250	250	250	250	250
Total current liabilities	17,521	16,065	16,057	16,071	16,071	16,973	24,753	24,753	24,753	24,753	24,753
Deferred rent	9,220	9,205	9,189	9,142	9,142	9,041	8,940	8,940	8,940	8,940	8,940
Notes payable, net	49,973	69,833	70,289	79,512	79,512	80,503	81,511	81,511	81,511	81,511	81,511
Commitments and contingencies	-	-	-	-	-	-	-	-	-	-	-
Total Liabilities	76,714	95,103	95,535	104,725	104,725	106,517	115,204	115,204	115,204	115,204	115,204
Shareholder's equity											
Common stock	391	393	429	438	438	439	444	447	447	447	447
Additional paid-in capital	382,548	386,905	427,054	432,002	432,002	436,424	444,041	507,908	507,908	507,908	507,907
Accumulated other comprehensive income	-	-	-	-	-	-	-	-	-	-	-
Deficit accumulated	(423,681)	(436,293)	(450,255)	(469,887)	(469,887)	(484,976)	(499,335)	(511,143)	(522,330)	(522,330)	(552,266)
Total shareholder's equity	(40,742)	(48,995)	(22,772)	(37,447)	(37,447)	(48,113)	(54,850)	(2,788)	(13,976)	(13,976)	(43,911)
Total liability and shareholder's equity	35,972	46,108	72,763	67,278	67,278	58,404	60,354	112,416	101,228	101,228	71,293

Source: Company reports and H.C. Wainwright & Co. estimates.

Table 8: Omeros Corporation (OMER)—Historical Statements of Cash Flows, Financial Projections

FY end December 31
$ in thousands, except per share data

	2016A					2017E					
	1QA	2QA	3QA	4QA	2016A	1QA	2QA	3QE	4QE	2017E	2018E
Cash flows from operating activities											
Net loss	(20,539)	(12,612)	(13,962)	(19,632)	(66,745)	(15,089)	(14,359)	(15,108)	(14,588)	(59,143)	(45,936)
Adjustments for:											
Stock-based compensation	4,422	2,609	2,535	4,016	13,582	3,276	3,132	3,300	3,400	13,108	16,000
Depreciation & amortization	53	69	89	89	300	115	119	119	119	472	476
Loss on early extinguishment of debt	-	-	-	5,595	5,595	-	-	-	-	-	-
Conversion of accrued interest to notes payable	-	-	-	516	516	-	-	-	-	-	-
Other non-cash expense	213	471	511	266	1,461	995	1,017			2,012	-
Change in operating assets & liabilities											
Accounts receivable	(129)	(2,160)	(1,651)	(1,580)	(5,520)	(1,444)	(5,594)	-	-	(7,038)	-
Inventories	(1,500)	192	384	268	(656)	168	55	-	-	223	-
Other current assets	(420)	178	927	(338)	347	(1,494)	(186)	-	-	(1,680)	-
Accounts payable	1,272	(1,540)	(35)	(16)	(319)	771	7,657	-	-	8,428	-
Deferred rent	-	(2)	(16)	(47)	(65)	-	-	-	-	-	-
Total change in operating assets & liabilities	(777)	(3,332)	(391)	(1,713)	(6,213)	(1,999)	1,932	-	-	(67)	-
Cash flows from operating activities	**(16,628)**	**(12,795)**	**(11,218)**	**(10,863)**	**(51,504)**	**(12,702)**	**(8,159)**	**(11,689)**	**(11,069)**	**(43,618)**	**(29,460)**
Cash flows from investing activities											
Investment in PPE	-	(34)	(44)	(48)	(126)	(72)	(244)	(50)	(50)	(416)	(200)
Purchases of marketable securities	(18)	(20,607)	(37,496)	(15,845)	(73,966)	(1,042)	(60)	-	-	(1,102)	-
Sales and maturities of marketable securities	14,650	16,225	17,000	9,882	57,757	11,778	6,150	-	-	17,928	-
Cash flows from investing activities	**14,632**	**(4,416)**	**(20,540)**	**(6,011)**	**(16,335)**	**10,664**	**5,846**	**(50)**	**(50)**	**16,410**	**(200)**
Cash flows from financing activities											
Proceeds from long-term debt	-	19,864	-	80,136	100,000	-	-	-	-	-	-
Payments on notes payable	-	(34)	(28)	(70,075)	(70,137)	-	-	-	-	-	-
Payments for debt prepayment and extinguishment costs	-	-	-	(5,700)	(5,700)	-	-	-	-	-	-
Payments for debt issuance costs	-	-	-	(1,501)	(1,501)	-	-	-	-	-	-
Payments on lease financing obligations	-	-	-	-	-	(51)	(73)	-	-	(124)	-
Increase in restricted cash and investments	-	-	(156)	5,000	4,844	-	-	-	-	-	-
Proceeds from issuance of common stock and warrants	-	724	37,279	36	38,039	-	-	-	-	-	-
Proceeds upon exercise of stock options and warrants	1,609	268	371	905	3,153	1,147	4,490	63,870	-	69,507	-
Cash flows from financing activities	**1,609**	**20,822**	**37,466**	**8,801**	**68,698**	**1,096**	**4,417**	**63,870**	**-**	**69,383**	**-**
Net increase/ decrease in cash and cash equivalents	**(387)**	**3,611**	**5,708**	**(8,073)**	**859**	**(942)**	**2,104**	**52,131**	**(11,119)**	**42,174**	**(29,660)**
Effect of exchange rate	-	-	-	-	-	-	-	-	-	-	-
Cash and cash equivalents, beginning of period	1,365	978	4,589	10,297	1,365	2,224	1,282	3,386	55,517	2,224	44,398
Cash and cash equivalents, end of period	978	4,589	10,297	2,224	2,224	1,282	3,386	55,517	44,398	44,398	14,739

Source: Company reports and H.C. Wainwright & Co. estimates

Public companies mentioned in this report:

Abbott Laboratories (ABT; not rated)
AbbVie (ABBV; not rated)
Akari Therapeutics (AKTX; not rated)
Alexion Pharmaceuticals (ALXN; not rated)
Allergan plc (AGN; not rated)
Alnylam Pharmaceuticals (ALNY; not rated)
Amgen (AMGN; not rated)
Amicus Therapeutics (FOLD; not rated)
AstraZeneca plc (AZN; not rated)
AveXis (AVXS; not rated)
bluebird bio (BLUE; not rated)
Blueprint Medicines (BPMC; not rated)
Eli Lilly & Co. (LLY; not rated)
GlaxoSmithKline plc (GSK; not rated)
Imprimis Pharmaceuticals (IMMY; not rated)
Innoviva (INVA; not rated)
Johnson & Johnson (JNJ; not rated)
Loxo Oncology (LOXO; not rated)
Merck & Co. (MRK; not rated)
Novartis AG (NVS; not rated)
Ophthotech (OPHT; not rated)
Pfizer, Inc. (PFE; not rated)
Roche Holding (RHHBY; not rated)
Sanofi S.A. (SNY; not rated)
Takeda Pharmaceutical Co. Ltd. (TKPHF; not rated)
Teva Pharmaceutical Industries (TEVA; not rated)
Theravance Biopharma (TBPH; not rated)
Ultragenyx (RARE; Under Review)

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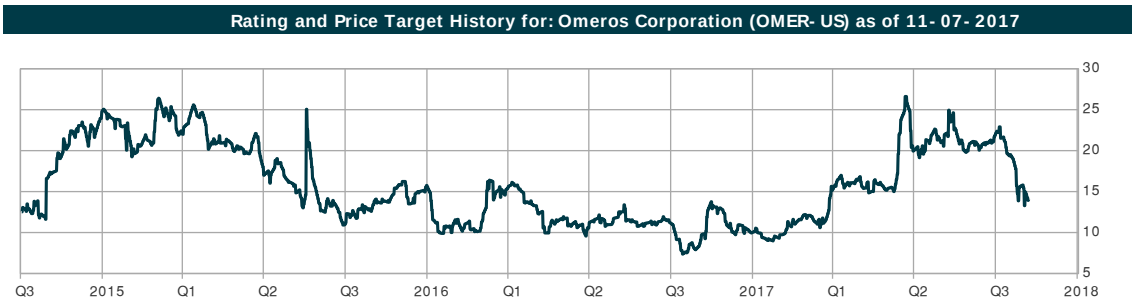
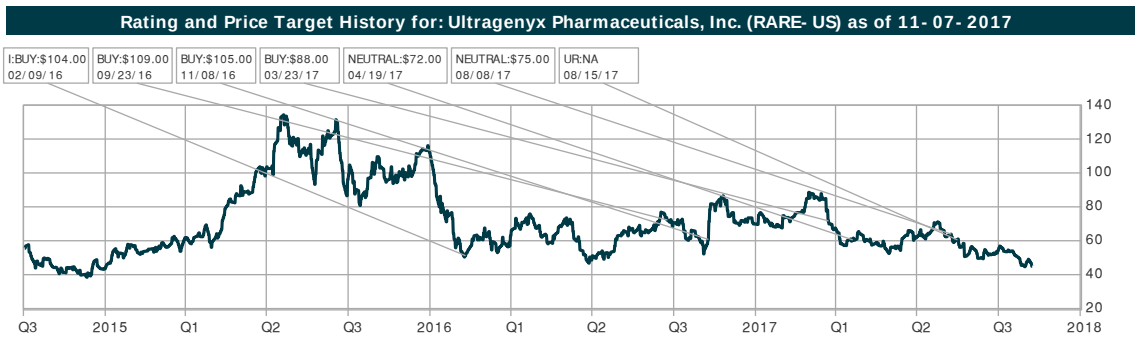
RETURN ASSESSMENT

Market Outperform (Buy): The common stock of the company is expected to outperform a passive index comprised of all the common stock of companies within the same sector.

Market Perform (Neutral): The common stock of the company is expected to mimic the performance of a passive index comprised of all the common stock of companies within the same sector.

Market Underperform (Sell): The common stock of the company is expected to underperform a passive index comprised of all the common stock of companies within the same sector.





Related Companies Mentioned in this Report as of Nov/7/2017

Company	Ticker	H.C. Wainwright Rating	12 Month Price Target	Price	Market Cap
Ultragenyx Pharmaceuticals, Inc.	RARE	Under Review	$NA	$45.48	

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Distribution of Ratings Table as of November 7, 2017				
			IB Service/Past 12 Months	
Ratings	Count	Percent	Count	Percent
Buy	228	90.84%	80	35.09%
Neutral	11	4.38%	0	0.00%
Sell	0	0.00%	0	0.00%
Under Review	12	4.78%	1	8.33%
Total	251	100%	81	32.27%

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