

Conference Title: OMEROS CORPORATION Annual Meeting

Date: Friday, June 27, 2025

Operator: Hello, and welcome to the Annual Meeting of Shareholders of Omeros Corporation. Please note that today's meeting is being recorded. During the meeting, we'll have a question-and-answer session. Shareholders who registered with their 16-digit control number can submit questions or comments at any time by using the Ask a Question feature at the bottom left corner of the screen. It is now my pleasure to turn today's meeting over to Dr. Gregory Demopoulos, Chairman and CEO of Omeros Corporation. Dr. Demopoulos, the floor is yours.

Gregory Demopoulos: Thank you, Operator, and welcome, everyone, to the 2025 Annual Meeting of Shareholders of Omeros Corporation. I'm Greg Demopoulos, Chairman and CEO of Omeros, and I will be presiding as chairman for this meeting. Peter Cancelmo, our Vice President and General Counsel, will act as secretary of the meeting. Also joining us today are members of our board of directors, Erica Fewel, a representative of Ernst & Young, our auditor is also on the line.

During the question-and-answer session at the end of today's event, Erica will be available to respond to appropriate questions. A representative of Broadridge Financial Services is also in attendance and has been appointed to act as Inspector of Elections. Peter, please take us through the rules of conduct and procedures.

Peter Cancelmo: Thanks, Greg. The link to the agenda for today's meeting is available by clicking on the Meeting Agenda link under Reading Materials on the right side of your screen. You will also find under the Meeting Materials heading a link to the Rules of Conduct for this meeting. To conduct an orderly meeting, we ask that participants abide by these rules.

Our agenda for today's event is divided into two parts. In the first part, we will address the formal business of the meeting. The second part will involve a brief management presentation followed by a question-and-answer session. Should you desire to ask a question during the meeting, please

use the Ask a Question feature at the bottom left corner of your screen and submit your question online. We will address your questions after the company presentation.

In order to get to as many questions as possible in the time allotted, each question should be succinct and limited to one topic, and questions on the same topic may be grouped, summarized, and answered together. Similar to our past shareholder meetings and as stated in the Rules of Conduct, we will not engage in questions or discussions that are irrelevant to our business or operations, or are substantially repetitious of questions or statements from other shareholders. Thank you in advance for your cooperation.

I'd like to remind you that the management, presentation and question and answer session will include statements that are forward-looking. These statements are based on management's beliefs and expectations as of today only, and are subject to change. All forward-looking statements involve risks and uncertainties that could cause the company's actual results to differ materially from expectations. Please refer to the Risk Factors section of the company's most recent Annual Report on Form 10-K for a discussion of these risks and uncertainties. We will now proceed to the formal business of the meeting.

Gregory Demopoulos: Thank you, Peter. The Secretary has delivered an affidavit of mailing establishing that notice for this meeting was duly given, all shareholders of record at the close of business on May 23rd, 2025, are entitled to vote at this meeting. Our first order of business is to determine whether a quorum for the purpose of transacting business before this meeting is present. Peter, do you have a report?

Peter Cancelmo: Yes. The shareholders' list shows that holders of 58,592,713 shares of common stock are entitled to vote at this meeting. We are informed by the Inspector of Elections that there are represented, in person or by proxy, at this meeting approximately 40,679,757 shares, or approximately 69.42% of all the shares entitled to vote at this meeting.

Gregory Demopolos: Thank you, Peter. With the quorum present, I declare this meeting to be duly convened for purposes of transacting such business as may properly come before us. The next order of business is a description of the matters to be voted on at today's meeting. The first proposal before the shareholders is the election of two class one directors, each to serve until the 2028 annual meeting of Shareholders, and until their respective successors are duly elected and qualified.

The nominees for election as class one directors are Arnold Hanish and Rajiv Shah. No other persons having been nominated in accordance with the company's bylaws. The nominations are now closed. The second proposal before the shareholders is the approval of the non-binding advisory resolution on the compensation paid to our executive officers, or say-on-pay. The third and final proposal before the shareholders today is the ratification of the appointment of Ernst & Young LLP as our independent registered public accounting firm for the fiscal year ending December 31st, 2025.

The polls are open for online voting. If you have not yet voted or wish to change your vote, you may do so now. Any shareholder who has already voted and who does not want to change their vote does not need to take any further action. We will now pause briefly for voting to take place. The online voting will now be closed. Peter, as secretary, will you please report the results of the voting?

Peter Cancelmo: Yes. Based on preliminary review of the votes cast, Arnold Hanish and Rajiv Shah have each been elected as class one directors. The proposed say-on-pay resolution has been approved, and the selection of Ernst & Young as our independent registered public accounting firm for fiscal year ending December 31st, 2025 has been ratified.

Gregory Demopolos: Thank you, Peter, and thank you all for attending today. This concludes the formal part of our meeting. And the annual meeting of shareholders for 2025 is now adjourned. I will now turn to the management presentation, after which we'll have a brief question-and-answer period.

All right. Let's move on to the presentation. Here you see a chart of the programs at Omeros. This is the full pipeline. All programs are based on novel targets that we have discovered, or targets for which we were the first to identify their respective functions. As a result the IP, the intellectual property, around each of these programs is broad. We'll go through a number of these programs today, but we'll go through this information in more depth as we walk through the presentation. As you can see, though, we have multiple meaningful value inflection points coming up over the next 24 months across our MASP-2 or in our narsoplimab program. With anticipated approvals in both the U.S. and Europe, our MASP-3 or Zaltenibart program, as well as in our addiction and oncology platforms.

Let's first look at our narsoplimab our proprietary MASP-2 inhibitor. The initial indication for narsoplimab is stem cell transplant-associated thrombotic microangiopathy TA-TMA. The BLA was accepted in early May, with a PDUFA date assigned of September 25th. As announced earlier today, the MAA was submitted to the European Medicines Agency, or EMA, with an expected decision mid-next year. The protocol and statistical analysis plan for this BLA were agreed with FDA, and the data are strong, consistently showing a threefold improvement in survival.

The drug has shown no safety signal of concern. So the benefit-risk balance here is heavily weighted to the benefits. We expect that narsoplimab will become the first and only drug approved for TA-TMA, which represents a substantial market with the opportunity for significant label expansion.

The three pathways in the complement system are shown here: the adaptive or classical pathway, the lectin or innate pathway, and the alternative pathway, which functions as an amplification loop

on the other two pathways. Narsoplimab targets MASP-2, the effector enzyme and only targetable enzyme in the lectin pathway. The lectin pathway is activated by patterns of carbohydrates on microbes and on damaged cells. Like the damage endothelial cells in TA-TMA. Endothelial damage is what triggers TA-TMA an often life-threatening complication of stem cell transplant.

An important thing to note here is that MASP-2 inhibition, unlike inhibition of C3, C5 and other complement targets, leaves the infection-fighting lytic arm of the classical pathway entirely intact, protecting against infection.

This slide depicts the role of endothelial damage in TA-TMA and associated life-threatening organ dysfunction. TA-TMA occurs in nearly 40% of allogeneic transplants, and one-year survival as you see in the lower right-hand corner, in high risk, TA-TMA is well, less than 20%. OMS 721 TA-TMA 001 was our pivotal trial of narsoplimab and TA-TMA. The primary efficacy endpoint, a rigorous composite, was developed arm in arm with FDA and required improvement in both laboratory values and organ function. Survival was the secondary endpoint.

Again, all patients had high risk to TA-TMA, with an expected response rate of about 15% and survival expected to be well less than 20%. Here are the resultant data. 61% showed a complete response, and survival was far better than expected in high risk TA-TMA patients.

Narsoplimab received Breakthrough Therapy designation from FDA in 2020. Concurrent with the initial submission of our BLA, an FDA reorganization occurred and our division moved for review of the BLA from the Oncology Center of Excellence to OCNHEN or the Office of Cardiology, Non-malignant hematology, endocrinology and nephrology. Despite earlier agreements with FDA, we received a CRL or complete response letter. We did not resubmit our BLA until we had agreement on our protocol and our SAP, or statistical analysis plan. And the primary endpoint is survival with an external control. All analyses were performed independently by Berry Consultants.

The external control for this resubmission is a registry, which is considered the highest level of real-world data. Here you see the results. Our pivotal trial patients versus the control demonstrated a hazard ratio of 0.32 with a P value of, I believe 11 zeros, followed by something like a two or a three. We also compared the 136 patients in our expanded access program to that same external control. Yielding a similar hazard ratio of 0.3 to 0.46 across the numerous analyses performed, again with similar p values.

We also looked at narsoplimab in our expanded access program as salvage therapy in patients who had failed or stopped one or more regimens of eculizumab, ravulizumab, pegcetacoplan, and or defibrotide. Historically, those patients who fail C5 inhibition have a very dismal outcome. Well, less than 20%. Narsoplimab delivered an impressive 50% survival across both adults and children.

Okay. So here are the results of the primary analysis and associated sensitivity analysis. In this case, a picture really is worth a thousand words. FDA also asked us to assess E values, that's what you see in the far right column. E values tell you what the likelihood is that unknown or unseen confounders could affect your results. E values greater than three to four tell you that it is extremely remote, that confounders could undo your results. Which is the case here.

Here you again see a forest plot, this one assessing the pivotal trial plus the EAP versus the control and all associated sensitivity analysis. Again, the results are similarly striking. This slide summarizes the data that we just went through. In short high unmet need with no approved therapy. The efficacy data are stellar, and across all studies with narsoplimab we have seen no safety signal of concern. So the distillate here is that the benefit-risk profile is heavily weighted to the benefit. Nadia, would you take us through the commercial slides?

Nadia Dac: Absolutely. Well, in addition to addressing a significant unmet need, the TA-TMA market represents an attractive commercial opportunity. With an established transplant patient population, a consolidated prescriber base that can be targeted with focused resources. Since transplants are

tracked and reported, we know where the highest volume takes place. The top 40 U.S. centers are responsible for approximately 60% of the annual allogeneic transplant volume, and in Europe, the top six countries treat about 60% of the allogeneic volume.

Our pre-launch efforts, through a small field team, have concentrated on educating the transplant care teams in these centers on the signs, symptoms, and urgency to diagnose TA-TMA while also understanding their processes and needs when it comes to treating TA-TMA. Once approved, the field team will expand in order to target all registered transplant centers.

What we've learned through these interactions is that HCPs are concerned about transplant-related complications, yet they don't feel they have adequate treatments to address TA-TMA. More specifically, until very recently, HCPs have not had consistent diagnostic criteria for TMA. And since no treatments are currently approved to treat it, they have limited efficacy data and no clear dosing data to guide off-label treatments. Finally, they have concerns about safety data with these off-label treatments. More specifically, they're concerned about the infection risk with C5 inhibitors.

Transplant experts have reported very favorable perceptions of narsoplimab when exposed to its value proposition, and those that have gained direct experience with narsoplimab through our expanded access program. Narsoplimab demonstrated impressive survival results in both the clinical trial and the real-world setting. It demonstrated a threefold greater survival versus an untreated external control. In fact, when other off-label treatments, including the C5 inhibitor eculizumab failed in these patients. As we've heard, more than 50% of the patients that transitioned to narsoplimab treatment achieved more than one year survival from time of TMA diagnosis.

And given its well-tolerated profile with no safety signals of concern, narsoplimab lends itself well to being dosed in either the in or outpatient setting, with a defined dosing duration that provides patient flexibility, offering more cost effectiveness versus the current off-label approaches.

As the first and only approved treatment for TA-TMA, our SOP will create reimbursement hurdles for current off-label approaches. Since unique ICD ten codes are essential for billing in the treatment of TA-TMA, were established back in 2021. Our ongoing outreach to payers has focused on both disease education and the fact that narsoplimab should be the only treatment reimbursed against the dedicated codes. Our team continues to receive requests by payers for product information exchanges, which we anticipate will increase as we get closer to our PDUFA date.

As mentioned earlier, our ongoing pre-launch efforts have focused on two main goals: educating the transplant team on the signs, symptoms and urgency to diagnose TA-TMA, as well as account profiling to understand each center's unique processes and needs when it comes to treating patients with an approved treatment. Through these efforts, we have identified Fast Start accounts among the top 40 centers where HCPs actively monitor for TA-TMA, with an HCP championing the urgency to treat these patients, ideally with an approved treatment. This champion would lead the formulary exception process approval for narsoplimab once approved, and we have a clear understanding of the continuation of care for narsoplimab patients as they transition from the end to outpatient setting.

The combination of a significant unmet need of patients diagnosed with TA-TMA, the impressive value proposition of narsoplimab as the first and only approved treatment once approved, and the targeted and sustained pre-launch efforts to date enable a solid launch success platform. Greg.

Gregory Demopoulos: Thanks, Nadia. You see here that some examples of the broad range of potential indications for our MASP-2 inhibitors, specifically in narsoplimab and our long-acting MASP-2 inhibitor, OMS 1029. The list is, of course, substantially longer than this, but just wanted to provide some sense of the breadth of MASP-2 involvement in diseases. Next slide, please. So, Steve, would you take us through Zaltenibart?

Steven Whitaker: Absolutely. Thanks, Greg. We'll briefly discuss Zaltenibart, its target MASP-3 differences from other complement inhibitors and data from our PNH or Paroxysmal Nocturnal Hemoglobin area program. Zaltenibart is a human monoclonal antibody that inhibits mannan-binding lectin associated serine protease three or MASP-3. Zaltenibart favorable pharmacokinetic properties and can be dosed as infrequently as once every 8 to 12 weeks. MASP-3 is the most proximal target in the alternative pathway of complement, which we'll show in the next slide.

The alternative pathway has been validated clinically. Three drugs inhibiting different alternative pathway targets have been approved in several indications. Notably, MASP-3 has important differences from other complement targets, and Zaltenibart has important differences from other alternative pathway drugs. These differences provide the potential for clinically meaningful safety, efficacy, and convenience differentiation from other complement inhibitors.

Zaltenibart is a late-phase asset in a Phase 3 program in PNH, which is an orphan hematological disease and a Phase 2 program in C3 glomerulopathy, which is an orphan kidney disease. Patent protection for Zaltenibart has a long patent runway. This is a diagram of the complement pathway. The alternative pathway is shown in orange. Each of these proteins, when activated sequentially, activates the next protein in the cascade. As you can see, MASP-3 is the most upstream protein in the alternative pathway. Inhibiting MASP-3 inhibits activation of factor D, which then inhibits activation of factor B, which inhibits activation of C3.

Inhibitors of each of these alternative pathway targets have been approved in a variety of diseases. We have shown clinically that MASP-3 inhibition not only blocks factor deactivation, but also blocks activation of the entire alternative pathway. This is important because, as we said, blocking the alternative pathway has been demonstrated to successfully treat a variety of diseases.

Another significant point is that injury in PNH is caused by both the membrane attack complex, or MAC in this diagram, and C3B. As you can see, drugs that inhibit C5, such as Solaris and ultramarathons, can only stop C5-mediated cell injury and not the C3b-mediated injury.

Now, let's talk about the innate characteristics of MASP-3 in Zaltenibart that provide potential advantages over other clinically validated targets and treatments. This slide connects these characteristics to potential advantages. First, MASP-3 has a low plasma concentration and slow turnover that makes it easier to inhibit than all other complement targets. If targets have high concentration, more drug is required for inhibition, and if targets have rapid turnover, drugs must be given more frequently to maintain inhibition. Less frequent dosing can provide better compliance and fewer missed doses. Also, available data indicates that MASP-3 is not an acute-phase reactant. Acute phase reactants or proteins whose concentration increases during inflammatory events such as influenza, bacterial infection, and surgery, or trauma. If these events occur, the blood concentration of most other complement targets increase, and there may not be enough drug available to inhibit the target mass.

MASP-3's unique combination of these factors may allow benefits not available with other targets. MASP-3 inhibition also does not affect the classical or terminal complement pathways. As Greg noted, these pathways are important for fighting infections and are maintained with MASP-3 inhibition. The combination of MASP-3 properties with Zaltenibart's pharmacokinetic and pharmacodynamic properties allow dosing as infrequently as once every 8 to 12 weeks. This can enhance convenience and adherence compared to other alternative pathway inhibitors that are dosed orally twice daily, orally three times daily, when also given with a C5 inhibitor or subcutaneously twice weekly.

A differentiator in PNH that is not included on this general slide is the location of MASP-3 and C5 in the complement pathway. MASP-3's position in the alternative pathway allows inhibition of both intravascular and extravascular hemolysis, while C5 inhibition only blocks intravascular hemolysis.

These potential benefits could provide marked positive and meaningful differentiation to other complement inhibitors.

Now let's turn to PNH specifically. PNH is a life-threatening disease that is caused by complement dysregulation. The complement dysregulation causes hemolytic anemia that occurs both intravascular through the MAC and extravascular through C3b, as mentioned earlier. Untreated patients suffer anemia, fatigue, shortness of breath, and thrombosis that can be fatal. C5 inhibitors such as eculizumab and ravulizumab are the standard of care and inhibit intravascular hemolysis only. Alternative pathway inhibitors control both intravascular and extravascular hemolysis, an important differentiator to C5 inhibitors.

PNH is a large market that is expected to grow to approximately \$10 billion in 2032, and Zaltenibart has the potential to capture significant market share due to the unique properties of the drug and the target MASP-3.

Let's review clinical data supporting Zaltenibart treatment in PNH. We'll show one of the Zaltenibart studies in patients with PNH. This study was conducted in patients who would not receive previous complement inhibitor treatment. The design was typical of studies in this field. After a screening period, all patients received Zaltenibart, which was previously known as OMS 906. It was administered at a low dose of 5mg/kg subcutaneously once every four weeks.

I'd like to note that our Phase 3 program will use the dosing regimen of 8 milligram/kg intravenously, once every eight weeks. That provides higher exposure and longer duration of effect. At study entry, patients had anemia with evidence of hemolysis. Nine of 11 patients had classical PNH, while two patients also had myelodysplastic syndrome or MDS. This is a form of blood cancer that is associated with PNH and bone marrow failure. The primary endpoint of this study was safety because this was an early study. The efficacy measures are relevant to our Phase 3 primary endpoints. The primary endpoint of both Phase 3 studies is a composite of the proportion of

patients who achieve both an increase in their hemoglobin level of at least two grams per deciliter from baseline and an absolute hemoglobin of at least 12g per deciliter.

The left panel on this slide shows the mean hemoglobin of patients having classical PNH, and the patients with MDS in different lines. The classical PNH population is the upper black line. This is also our population for Phase 3. As you can see, both populations showed marked improvement. In fact, all patients achieved an increase of at least two grams per deciliter, regardless of the presence or absence of MDS. The mean hemoglobin increase in the classical PNH patients was 5.7g per deciliter, and the mean increase in patients with MDS was 3.4g per deciliter. Notably, all classical PNH patients also achieved an absolute hemoglobin of 12g per deciliter. In other words, all of the classical PNH patients achieved both components of the Phase 3 primary endpoint.

The right panel of this slide demonstrates the change in lactate dehydrogenase, or LDH, during treatment. LDH is a measure of intravascular hemolysis. The LDH falls dramatically after treatment initiation and reaches a mean between the upper limit of normal and 1.5 times the upper limit of normal. A treatment target in this indication. The LDH also falls substantially in the patients with MDS, but does not reach the level of the classical PNH patients due to the underlying nature of MDS.

As I mentioned, the data we just reviewed was from evaluation of the 5 milligram/kg subcutaneous every four-week dosing regimen. That dosing regimen provides lower Zaltenibart exposure than our Phase 3 dosing regimen of 8 milligram/kg IV every eight weeks. This slide provides preliminary data from evaluation of our Phase 3 dosing regimen in the Zaltenibart treatment-naive population.

The table includes data on the two components of our Phase 3 primary endpoint. As you can see, all of Zaltenibart-treated patients achieve both components of a two-gram-per-deciliter hemoglobin increase and an absolute hemoglobin of at least 12g per deciliter. Data from a Phase 3 trial lptacopan, which is an approved alternative pathway factor B inhibitor in their treatment-naive

population, are also provided in the table. It's important to note that these data were not obtained from a head-to-head trial, and differences in designs, patient characteristics, and geographical regions may affect results.

As you can see, Iptacopan-treated patients achieved in 93% in the two-gram-per-deciliter hemoglobin increase increased measure and a 63% response in the 12-gram-per-deciliter absolute hemoglobin response. While we can't make direct comparisons between the trials, I think you can see what we're very excited about the potential of Zaltenibart and PNH. In view of the efficacy we've observed the potential safety and efficacy, differentiation to other complement inhibitors, and the convenient treatment regimen allowed by the characteristics of Zaltenibart and its target MASP-3. With that, I'll turn this over to Nadia for a commercial perspective.

Nadia Dac: Thanks, Steve. Zaltenibart targeting of MASP-3, which is the key activator of the alternative pathway, has several potential advantages over the PNH competitive landscape, including preserving complement's lytic function to allow appropriate response to infection, which is very important. Through market research and advisory boards, HCPs have noted that when our Phase 2 results are replicated in our planned head-to-head Phase 3 trials, Zaltenibart compelling efficacy in combination with safety advantages over other complement inhibitors and the low treatment burden could become standard of care in a significant share of PNH patients.

Physicians mentioned a high degree of concern about patient compliance with the orals that are dosed two to three times per day, and the potential for breakthrough hemolysis when patients forget to take their pills. With the expected efficacy and safety profile of Zaltenibart dosed only four to six times per year, both physicians and patients reported confidence that the most powerful treatment would be protecting them, without having to think about their disease on a daily basis as they take those multiple pills. The bottom line is that Zaltenibart represents patient-friendly power in the HCPs hands providing control without that anxiety about compliance. Back to you, Greg.

Gregory Demopoulos: Thanks, Nadia. So our MASP-3 inhibitors, like our inhibitors of MASP-2, have broad applicability. Many of these indications have been validated by other alternative pathway inhibitors, including Iptacopan and others. A partial list of potential indications is seen here for the reasons that Steve and Nadia discussed. We believe that MASP-3 is the premier alternative pathway target, and that Zaltenibart is the premier alternative pathway inhibitor.

Turning now to our PDE7 inhibitor program. OMS527 in addiction. The National Institute on Drug Abuse, or NIDA, approached us requesting that we pursue OMS527 in the treatment of cocaine use disorder. There currently is no approved treatment for cocaine use disorder or CUD. NIDA fully funded primate drug-drug interaction studies, which showed that OMS527 does not enhance the detrimental effects of cocaine. And then earlier this year, NIDA committed full funding for an inpatient clinical trial comparing the safety and efficacy of on OMS527 to placebo in the treatment of adults with CUD. A data readout is expected late this year or early next year.

So we'll wrap up today's presentation with a quick review of our OncotoX program, which is one part and only one part of Omeros is oncology platform. So the OncotoX program is, as I stated our entry to oncology. It involves a proprietary large molecule which is designed to target and kill only dividing cancer cells. The first indication for OncotoX that we've identified will be acute myeloid leukemia, or AML. In extensive in vitro and in vivo studies, our drug has shown really superior activity to current AML standard of care treatment. I'll share some data with you along these lines in the next few slides.

Equally important, though, the OncotoX is agnostic to underlying AML mutations. So, whatever the mutation, it appears that the OncotoX works. We expect them to be able to address the majority of AML cases. Importantly, in preliminary preclinical tolerability studies, the OncotoX is well tolerated, which bodes well for its safety profile. We're currently building a strong intellectual property position for the program and have initiated IND-enabling studies. We expect to be in the

clinic in 18 to 24 months. For this, we have the support and guidance of a really distinguished clinical steering committee with AML leaders from premier cancer centers across the nation.

We recently unveiled our program to prospective partners at the American Association of Cancer Research in Chicago and received quite strong interest. Throughout the next slides, I'll share with you some more information about the structure and mechanism of action of OncotoX, as well as results from animal studies.

Next slide, please. So this slide gives you a sense of the structure of OncotoX. It's composed of three units each, which serves a very distinct function. To the right in blue is the targeting moiety or targeting element that delivers the OncotoX to the specific cells of interest, so it targets surface proteins highly expressed in this case in AML cells. It's reminiscent of antibody drug conjugates or ADCs, but it's not an antibody. Really, nor does it involve chemical conjugation. It's a three-component hybrid protein.

To the left in yellow is the OncotoX payload for this. We use an enzyme that causes double-strand DNA breaks. So when you think about this, it has the effect of radiotherapy, but without the use of tissue-damaging radio isoforms. The payload, as I mentioned earlier, only kills dividing cells, which means that it can eliminate AML, blast, and leukemia stem cells, both of which are refractory to chemotherapy and really represent a major challenge in the treatment of AML today.

Because it targets dividing cells, only the payload spares the regular hematopoietic stem cells, reducing potential toxicity concerns. Now, the middle piece that you see there in orange is highly proprietary. It's the component that provides for the delivery and the expression of the OncotoX in the right place at the right time. I'll explain this a bit more on the next slide.

It's important to note that the overall size of the OncotoX is approximately half the size of an antibody. Why is that important? Well, that allows the molecule to reach areas that an antibody or

an antibody drug conjugate and ADC really can't reach. As mentioned previously, the first indication is AML. And while we're focused on AML, there are a substantial number of other hematologic malignancies that share the same highly expressed cell surface proteins. And that would make these other indications similarly potential target indications.

Okay. This slide shows a cartoon a schema of a OncotoX's predicted mechanism of action. Through its targeting domain, it binds to a surface receptor which is targeted by OncotoX and is highly expressed on AML cells. It enters the cells through receptor-mediated endocytosis. Once in the endosome, which is that yellow circle that you see on the slide the low pH in the endosome allows the delivery domain of OncotoX to undergo a dramatic conformational change. And that allows the OncotoX to insert itself into the membrane and create a pore. Part of the OncotoX, then threads through the pore and into the cytosol, releasing the payload in the cytosol while avoiding any degradation in the endosome.

The payload then carries a nuclear localization signal, and that signal allows it to translocate to the nucleus, where like radiotherapy, but without the detrimental side effects of radiotherapy, it breaks the double-stranded DNA, and that leads obviously to cell cycle arrest and cell death of the cancerous dividing cells.

So here are some of the data that have been generated, and these are representative. And obviously, there we have generated substantially more data than you're seeing here. But MOLM 13 is a human AML cell line, as I mentioned before, appears to be mutation agnostic. The mutation in MOLM 13 is flit three. Important because it is extremely aggressive. The cell line carries luciferase, which allows the tracking of tumor burden in the animals. So luciferase can be both visualized. And I'll show you an image of that in a moment, and quantified so we can see it, and we can count the signal.

We infuse immunodeficient mice with the MOLM 13 cell line. And a day later, because of the aggressive nature of MOLM 13, we start treatment for five consecutive days and then we stop. So we treat for five days, and that's it. Tumor burden and survival are monitored. A standard of care in AML consists of another class and is a cytokine. It's a combination therapy. When mice are treated with that combination of venetoclax and associated, there is a really only marginal benefit in controlling tumor burden in this model. And you can see that in the upper left figure, resulting. Again, this is now in the upper right figure there you see only a median survival benefit of about one and a half days.

Now, this is really in sharp contrast to OncotoX, and that's demonstrated in the lower two panels. When we treat with OncotoX, the tumor burden is completely flatlined, as you see in the lower left. And the survival benefit now is well past 80 days. In vivo and in vitro OncotoX greatly outperforms the current AML standard of care. And again, what you're seeing here is following five days of treatment only and then cessation of treatment.

On the next slide. So this is why the luciferase marker is important. We can actually identify and then quantify the signal. And here you see really the visual manifestation of the study that I just described. You can see that at day 16, after tumor cells are infused into the mice, the vehicle-treated mice are riddled with tumor throughout the body. In contrast, there is no detectable tumor burden in mice treated with OncotoX.

Again, I'm going to underscore that this is following only daily five days of treatment with OncotoX with no additional treatment. And yet we're seeing survival in these animals out well beyond 80 days. So this is really in quite stark contrast to the one and a half day benefit seen with the current standard of care. We expect to be sharing more information on this program as well as the other programs we've gone through today, and in the not-too-distant future.

I hope that the presentation provided useful information on some of the programs and the reasons for our excitement around them. So let's open the meeting to questions. As I mentioned earlier, those shareholders who registered to attend the meeting with the proxy control number may submit questions online by using the Ask a Question feature on the bottom left corner of your screen. We'll read the questions out loud. Though we may paraphrase here, longer questions, or combine multiple questions on the same topic, to get to as many questions as we can. I'm joined here today by other members of the management team. So as far as relevant questions arise, they'll introduce themselves and will share their responses with you.

All right. First question. What is your plan to address your financial position in the near term? Well, as you have seen, we addressed the substantial debt overhang by exchanging approximately 71 million in principal of our convertible senior notes. Those were due in 2026. We've moved those out to 2029. Have also reached an agreement to convert another 10 million of principal of those convertible notes to equity. That's been ongoing.

Overall this reduced our total debt by 10 million, reduced our near-term repayment obligations, and left us with a relatively small stub of 17 million on the 26 notes. So I think we're in good shape with respect to the convertibles. Our focus has been on non-dilutive capital. I think it's clear we've spoken previously about the strong amount of inbound interest around our programs with respect to partnering. While we don't talk specifically or give details about our partnering discussions, I can tell you that those are ongoing. As you know, we have an ATM available for use as well.

Our next question. Any update on your paused programs, particularly with the Phase 3 trials for 906? When will they restart? The steps we've taken to date to lower the near-term expenses were executed with the goal of ensuring that our highest priority clinical development programs --. That includes our Phase 3 trials for 906 and PNH, that those can be restarted as quickly and seamlessly as possible.

As I stated earlier, we're assessing partnering around a number of our programs, and we expect to continue being able to move those programs forward in the not-too-distant future. So while we have suspended the trials, we've also, of course, suspended the activities of the CROs. So with respect to restarting, we'll re-engage those CROs once the resources are in place, and then the trials tend to restart within a few weeks in the U.S. and slightly longer in ex-U.S. geographies.

Next question. What is the status of the BLA review? Have there been any communications or other signals from FDA? We have ¹[received and responded to FDA information requests and the review process appears to be proceeding well. We don't comment on the specifics of our interactions with FDA, including the specific content of information requests. We believe that we have responded thoroughly and well to all information requests to date. Cathy do you have anything to add to that?

Cathy Melfi: Agree with your comments, Greg. The review and the process have been proceeding well.

Gregory Demopoulos: Okay. Next question. Do you expect the timeline for reviewing the BLA will be affected by issues at the agency? Will FDA be able to complete its review by the PDUFA date? Look, we are aware of reports that FDA has not met its target goal date for some recent reviews, but our review seems to be proceeding as expected. I understand that Commissioner Makary has committed to FDA meeting its PDUFA dates and there are reports of FDA, in fact, delivering approval decisions earlier than the corresponding PDUFA dates. The team reviewing our BLA is the same group that aligned on our statistical analysis plan. So they are familiar with the analyses and a good part of the information submitted in the BLA and we'd expect] that familiarity with the product, with the application, will continue to help with the efficiency of the review.

¹ The bracketed text provides remarks that were intermittently inaudible during the webcast of the 2025 Annual Meeting of Shareholders of Omeros Corporation due to audio transmission errors. In the archived webcast, the bracketed remarks occur at approximately minute 55:39 and end at approximately 57:30.

In the past, you've stated that Omeros has partnered assets. Could you elaborate having them and monetizing them are two very different processes? Yes. fully recognized having them and monetizing them do not always equate to the same things. We have a number of partner assets. I think that's clear. When you look at narsoplimab, when you look at 906, when you look at other programs in our pipeline, I think it's clear that these are highly partner-able programs. I've already stated that the inbound interest on a number of our programs has been high. I think that the difference between monetizing and having may be narrowing here.

Next question. Again, around partnering, it seems. Partnering seems to be a viable option, and opportunity is the reason that progress has not been announced is that no one is interested in partnering or is Omeros trying to do everything on their own. I think the answer to both of those is a resounding no. Clearly, there is a strong interest in the programs. And no, Omeros is not trying to do everything on its own. We are, as I've said, quite intently focused on partnering one or more of our programs. And because we don't comment on ongoing processes does not mean that those processes are not underway or that there is some deficiency in interest. I think neither of those are accurate.

Another question on narsoplimab program is the resubmitted BLA. In the resubmitted BLA, you state that immortal time bias has been adjusted for. Could you confirm whether that adjustment was applied to patient by patient using left truncation for each control subject, or whether it was done as a single cohort-level time shift? And to date, has FDA signaled any concerns about the specific method you used to correct for immortal time bias? That is a pretty technical question. The immortal time bias was accounted for in our analyses using multiple imputations, and those were done by sampling, with replacement, from the 28 intervals observed across the OMS 721 TMA 001 clinical trial. So effectively, this is multiple imputations. And then running a thousand imputations. The result is something that is quite robust. The method was discussed with FDA. FDA effectively agreed with that. It was part of our statistical analysis plan, which FDA agreed. So I think that we are in good shape there.

Cathy Melfi: I'll just add that we also did multiple sensitivity analysis looking at alternatives for the immortal time bias. And those sensitivity analyses confirm the robustness of what we did and the results.

Gregory Demopoulos: So with that, Peter, are we seeing anything else?

Peter Cancelmo: No. No others.

Gregory Demopoulos: Okay. With that, we will conclude the 2025 annual Shareholder meeting. And thank you, everyone, for joining us today and for your continued support. Have a good rest of the day. Thank you.

Operator: This concludes the meeting. You may now disconnect.