



NEWS RELEASE

Omeros Corporation Announces Additional Positive Data for OMS721 in IgA Nephropathy

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-- Magnitude of Proteinuria Reduction Consistent With Previous Cohort --

SEATTLE--(BUSINESS WIRE)--Oct. 1, 2018-- Omeros Corporation (NASDAQ: OMER) today announced positive data from the company's ongoing Phase 2 clinical trial of OMS721 for the treatment of renal diseases. The data are from study patients with immunoglobulin A (IgA) nephropathy, a progressively worsening kidney disease and the most common glomerulonephritis worldwide, responsible for 10 percent of all people on dialysis globally. Patients in this cohort had IgA nephropathy with high risk of progression but none received corticosteroid treatment immediately prior to or during the study – different than the cohort reported last year in which all patients initiated the study while on steroid therapy. Reductions in protein levels in urine collected over a 24-hour duration (the “gold-standard” in assessing proteinuria) in this steroid-free OMS721-treated group were of similarly large magnitude to those seen in the earlier cohort. In IgA nephropathy, proteinuria is the most reliable prognostic factor for loss of kidney function – the greater the proteinuria reduction, the better the prognosis. OMS721 is Omeros' lead human monoclonal antibody targeting mannan-binding lectin-associated serine protease-2 (MASP-2), the effector enzyme of the complement system's lectin pathway. In addition to an ongoing Phase 3 clinical trial in IgA nephropathy, OMS721 is in Phase 3 clinical programs for hematopoietic stem cell-associated thrombotic microangiopathy (HSCT-TMA) and for atypical hemolytic uremic syndrome. OMS721 has been granted breakthrough therapy designations by FDA for both IgA nephropathy and HSCT-TMA.

“We now have results of treatment with OMS721 in IgA nephropathy patients both with and without concomitant steroid therapy,” stated Jonathan Barratt, Professor of Renal Medicine at the University of Leicester and Honorary Consultant Nephrologist at Leicester General Hospital. “In contrast to the previously reported cohort who were enrolled while on steroid therapy, the patients in this study were steroid-free, and yet the magnitude of proteinuria reduction across both groups is consistent and likely to indicate a significant renoprotective effect of OMS721 in IgAN, with a legacy effect again seen for months beyond cessation of OMS721 treatment. Notably, the current study’s patients are at very high risk of progressive renal disease and generally have long-standing, established disease with multiple comorbidities, representing a difficult-to-treat population. Despite that, almost all of the OMS721-treated patients responded to the first 12-week course of treatment. Collectively, these data further confirm the rationale for lectin pathway inhibition as a promising future option for the treatment of patients with IgAN.”

The current cohort study was designed to evaluate the safety and tolerability of once-weekly intravenous (IV) dosing of OMS721 and to describe the effect of the drug on 24-hour proteinuria in patients with IgA nephropathy. Twelve patients were enrolled. Per protocol, patients are required (i) to not receive corticosteroids during the study duration or for at least three months prior to screening and (ii) to be stable on an optimized dose of ACE inhibitors/ARBs (i.e., renin-angiotensin system [RAS] blockade) before enrollment. In a double-blind design, study patients were randomized 1:1 to receive either OMS721 or placebo for 12 weeks, and then both groups could receive extended OMS721 dosing at investigator discretion. Three patients were excluded from the analysis: two for unstable RAS blockade with at least a 50 percent change in dosing of RAS-blockade medication during the study, and one for untreated gastrointestinal disease that persisted throughout a substantial majority of the study duration. These conditions are known to affect proteinuria levels markedly, and results from these patients were determined by IgA nephropathy expert review to be unevaluable.

For the nine evaluable patients, median reductions in proteinuria following the initial 12-week course of treatment were 18.4 percent and 18.0 percent for the OMS721 and placebo groups, respectively. The one OMS721-treated patient whose proteinuria level remained elevated after the initial treatment course subsequently reached a 67.8-percent reduction from baseline following an additional course of OMS721 dosing.

After the initial 12-week treatment with OMS721 or placebo, all patients rolled into a dosing-extension period during which time a patient could receive, at investigator discretion, treatment with OMS721 if his/her respective 24-hour proteinuria level remained above 1 gram or the proteinuria response was less than a 50-percent reduction from baseline. Eight patients received OMS721 in this extension period, after either first receiving 12 weeks of OMS721 or placebo. Comparison with baseline of the proteinuria data after at least 18 weeks in the study (i.e., after the first course of OMS721 or placebo treatment and six-week follow-up) shows a median proteinuria reduction for this group of 55.7 percent. Four patients in this OMS721 dosing-extension period have reached between 9 and 12

months beyond baseline, and show reductions in proteinuria of 53.9 percent, 57.4 percent, 65.3 percent, and 67.8 percent. At most recent assessment, two of these four patients have continued to demonstrate sustained reductions in proteinuria for 2.5 and 5 months, respectively, after cessation of treatment with OMS721; the other two patients just recently completed treatment courses.

For the evaluable patients in the OMS721-treated and placebo groups, respectively, median ages at study start were 50 vs 33 years old; median time since diagnosis was 19 vs 9 years; and median baseline proteinuria was 2.9 vs 2.5 g/day. Forty-four percent of these patients had nephrotic-range proteinuria at baseline, ranging from approximately 4.5 to 12 g/day, and most patients had multiple comorbidities.

Consistent with all other studies conducted with OMS721, the drug was well tolerated and no safety concerns were seen in this cohort of patients. Across all 12 patients, no treatment-related serious adverse events were observed, and most reported adverse events were mild or moderate.

The OMS721 treatment-extension period in this cohort study remains ongoing. Omeros plans to report additional data from this Phase 2 cohort at a future medical congress.

"We're pleased with the strength of the data across a relatively small cohort of predominantly high-proteinuria patients and with the magnitude of the effect on proteinuria in the absence of steroid exposure," stated Gregory A. Demopoulos, M.D., chairman and chief executive officer of Omeros. "Given these results, we are exploring ways to streamline our active Phase 3 development program in IgA nephropathy. In addition, while most IgA nephropathy patients treated with OMS721 have responded rapidly to a single course of dosing, this study has demonstrated the potential benefit provided by limited multi-course dosing of the drug. Our ongoing Phase 3 ARTEMIS-IGAN trial incorporates repeat-dosing regimens to take advantage of this opportunity."

No treatments are approved for IgA nephropathy. With an annual incidence of approximately 1 per 100,000, it is estimated that 1 in 1,400 persons in the U.S. will develop IgA nephropathy in his or her lifetime. As many as 40 percent of them will develop end-stage renal disease.

About Omeros' MASP Programs

Omeros controls the worldwide rights to MASP-2 and all therapeutics targeting MASP-2, a novel pro-inflammatory protein target involved in activation of the complement system, which is an important component of the immune system. The complement system plays a role in the inflammatory response and becomes activated as a result of tissue damage or microbial infection. MASP-2 is the effector enzyme of the lectin pathway, one of the principal complement activation pathways. Importantly, inhibition of MASP-2 does not appear to interfere with the antibody-dependent classical complement activation pathway, which is a critical component of the acquired immune

response to infection, and its abnormal function is associated with a wide range of autoimmune disorders. MASP-2 is generated by the liver and is then released into circulation. Adult humans who are genetically deficient in one of the proteins that activate MASP-2 do not appear to be detrimentally affected by the deficiency. OMS721 is Omeros' lead human MASP-2 antibody.

Phase 3 clinical programs are in progress for OMS721 in atypical hemolytic uremic syndrome (aHUS), in immunoglobulin A (IgA) nephropathy and in hematopoietic stem cell transplant-associated thrombotic microangiopathy (HSCT-TMA). Also, two Phase 2 trials are ongoing. One is continuing to evaluate IgA nephropathy patients and has generated positive data in two cohorts of patients with IgA nephropathy (with and without concurrent corticosteroid therapy, respectively) and with lupus nephritis; the other is enrolling and has reported positive data in patients with HSCT-TMA and in patients with aHUS. OMS721 can be administered both intravenously and subcutaneously, and Omeros expects to commercialize each formulation of OMS721 for different therapeutic indications. In parallel, Omeros is developing small-molecule inhibitors of MASP-2. Based on requests from treating physicians, Omeros has established a compassionate-use program for OMS721, which is active in both the U.S. and Europe. The FDA has granted OMS721 breakthrough therapy designation for IgA nephropathy and for high-risk HSCT-TMA, orphan drug status for the prevention (inhibition) of complement-mediated thrombotic microangiopathies and for the treatment of IgA nephropathy, and fast track designation for the treatment of patients with aHUS.

Omeros also has identified MASP-3 as responsible for the conversion of pro-factor D to factor D and as a critical activator of the human complement system's alternative pathway. The alternative pathway is linked to a wide range of immune-related disorders. In addition to its lectin pathway inhibitors, the company is advancing its development of antibodies and small-molecule inhibitors against MASP-3 to block activation of the alternative pathway. Omeros plans for its MASP-3 program to enter the clinic late next year.

About Omeros Corporation

Omeros is a commercial-stage biopharmaceutical company committed to discovering, developing and commercializing small-molecule and protein therapeutics for large-market as well as orphan indications targeting inflammation, complement-mediated diseases and disorders of the central nervous system. The company's drug product OMIDRIA® (phenylephrine and ketorolac intraocular solution) 1% / 0.3% is marketed for use during cataract surgery or intraocular lens (IOL) replacement to maintain pupil size by preventing intraoperative miosis (pupil constriction) and to reduce postoperative ocular pain. In the European Union, the European Commission has approved OMIDRIA for use in cataract surgery and other IOL replacement procedures to maintain mydriasis (pupil dilation), prevent miosis (pupil constriction), and to reduce postoperative eye pain. Omeros has multiple Phase 3 and Phase 2 clinical-stage development programs focused on: complement-associated thrombotic

microangiopathies; complement-mediated glomerulonephropathies; cognitive impairment; and addictive and compulsive disorders. In addition, Omeros has a diverse group of preclinical programs and a proprietary G protein-coupled receptor (GPCR) platform through which it controls 54 new GPCR drug targets and corresponding compounds, a number of which are in preclinical development. The company also exclusively possesses a novel antibody-generating platform.

Forward-Looking Statements

This press release contains forward-looking statements within the meaning of Section 27A of the Securities Act of 1933 and Section 21E of the Securities Exchange Act of 1934, which are subject to the “safe harbor” created by those sections for such statements. All statements other than statements of historical fact are forward-looking statements, which are often indicated by terms such as “anticipate,” “believe,” “could,” “estimate,” “expect,” “exploring,” “goal,” “intend,” “likely,” “look forward to,” “may,” “plan,” “potential,” “predict,” “promising,” “project,” “prospects,” “should,” “will,” “would” and similar expressions and variations thereof. Forward-looking statements are based on management’s beliefs and assumptions and on information available to management only as of the date of this press release. Omeros’ actual results could differ materially from those anticipated in these forward-looking statements for many reasons, including, without limitation, risks associated with product commercialization and commercial operations, unproven preclinical and clinical development activities, regulatory oversight, intellectual property claims, competitive developments, litigation, and the risks, uncertainties and other factors described under the heading “Risk Factors” in the company’s Quarterly Report on Form 10-Q filed with the Securities and Exchange Commission on August 9, 2018. Given these risks, uncertainties and other factors, you should not place undue reliance on these forward-looking statements, and the company assumes no obligation to update these forward-looking statements, even if new information becomes available in the future.

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