



NEWS RELEASE

Omeros' MASP-2 Antibody Preserves Brain Tissue and Function in Stroke Models

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-- Data Published in Journal of Neuroinflammation --

SEATTLE--(BUSINESS WIRE)--Sep. 8, 2016-- Omeros Corporation (NASDAQ: OMER) today announced results from its OMS721 complement program. OMS721 is Omeros' lead antibody targeting mannan-binding lectin-associated serine protease-2 (MASP-2). In well-established animal models of ischemic brain injury or stroke, MASP-2 deficiency or inhibition significantly limited brain infarct size and protected against functional loss. The results of the studies were recently published in the Journal of Neuroinflammation (Orsini et al., Mannan binding lectin-associated serine protease-2 (MASP-2) critically contributes to post-ischemic brain injury independent of MASP-1, J.

Neuroinflammation 2016, 13:213) and can be accessed online at <http://link.springer.com/article/10.1186/s12974-016-0684-6>.

The studies were conducted collaboratively in the laboratories of Prof. Wilhelm Schwaeble at the University of Leicester and of Dr. Maria-Grazia De Simoni, head of the Laboratory of Inflammation and Nervous System Diseases, IRCCS-Istituto di Ricerche Farmacologiche Mario Negri. Focal brain ischemia was induced in wild-type (WT) mice and in mice deficient for MASP-2 by transient middle cerebral artery occlusion (tMCAO). Forty-eight hours after ischemia, histopathologic damage to brain tissue and functional neurological deficits were assessed. All surgical procedures and assessments were performed by investigators blinded to the experimental conditions. MASP-2-deficient mice, compared to WT mice, had significantly reduced infarct volumes (19 percent reduction, $p < 0.05$) and

significantly lower neurological deficits (44 percent [$p < 0.05$] and 39 percent [$p < 0.01$] reductions for general and focal functional deficits, respectively). Consistent results were seen with a derivative of Omeros' lead human MASP-2 antibody OMS721 modified for improved activity in mice and administered in three doses, two prior to and the third following ischemia. Compared to an isotype control antibody, the OMS721 derivative decreased infarct volume by 20 percent ($p < 0.05$) and reduced general (46 percent, $p < 0.01$) and focal (45 percent, $p < 0.01$) neurological deficits. In an additional model of stroke in which brain ischemia is induced by three-vessel occlusion, MASP-2-deficient mice were again protected against ischemic brain damage as evidenced by a 31 percent reduction ($p < 0.01$) in infarct volume. Immunohistochemical and morphologic analyses further demonstrated the protective effects of MASP-2 inhibition on brain tissue during stroke. Additional studies are planned to define further the therapeutic window for OMS721 in stroke.

"The data clearly identify MASP-2, the effector enzyme of the lectin pathway, as a primary driver in ischemic brain tissue injury," said Prof. Dr. Eberhard Weihe, director of the Institute of Anatomy and Cell Biology and head of the department of molecular neuroscience at Philipps-University, Marburg. "The strength and consistency of the data in these studies across infarct size, functional outcomes and other histopathological assessments suggest an important role for OMS721, a highly selective MASP-2 inhibitor, in protecting the brain, heart, kidney and other organs from the damage of ischemic injury. Certainly the ability to preserve both cerebral tissue and associated neurological function would represent a major advance in the acute management of stroke patients."

Omeros currently is conducting a Phase 3 clinical program evaluating OMS721 in atypical hemolytic uremic syndrome as well as Phase 2 programs assessing the drug in hematopoietic stem cell transplant-associated thrombotic microangiopathy, in thrombotic thrombocytopenic purpura and in IgA nephropathy and other complement-related renal diseases.

About Omeros' MASP Program

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 appears to be unique to, and required for the function of, one of the principal complement activation pathways, known as the lectin pathway. 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 is associated with a wide range of autoimmune disorders. Adults who are genetically deficient in one of the proteins that activate MASP-2 do not appear to be detrimentally affected by the deficiency. Omeros has received both orphan drug status and fast track designation from the U.S. FDA for its lead human MASP-2 antibody OMS721. Omeros has an ongoing Phase 3 clinical program

for OMS721 in atypical hemolytic uremic syndrome, an ongoing Phase 2 program targeting hematopoietic stem cell transplant-associated thrombotic microangiopathy as well as thrombotic thrombocytopenic purpura and another ongoing Phase 2 program targeting complement-related renal diseases. An investigator-requested compassionate use program for OMS721 is also underway. OMS721 has demonstrated no safety concerns in human trials or chronic toxicity studies. In addition to potential intravenous administration, Omeros plans to commercialize OMS721 initially for one or more therapeutic indications as a subcutaneous injection. In addition to its antibodies, Omeros is developing small molecules targeting MASP-2.

Omeros also has identified MASP-3 as the protein essential to the activation of the alternative pathway of complement (APC). APC inhibitors are thought to have preventive or therapeutic effects across a broad range of diseases including hemolytic uremic syndrome (HUS), atypical HUS, paroxysmal nocturnal hemoglobinuria, traumatic brain injury, arthritis, wet age-related macular degeneration, ischemia-reperfusion injury, transplant-related complications and other immune-related disorders. In its OMS906 program, Omeros is developing both antibody and small molecules to block MASP-3. Through its growing intellectual property position, Omeros exclusively controls inhibitors of the protein activator of the alternative pathway (MASP-3) and, with its OMS721 program, inhibitors of the effector enzyme of the lectin pathway (MASP-2), allowing the company to target with unprecedented precision diseases caused by dysregulation of one or both of these pathways.

About Omeros Corporation

Omeros is a biopharmaceutical company committed to discovering, developing and commercializing both small-molecule and protein therapeutics for large-market as well as orphan indications targeting inflammation, coagulopathies and disorders of the central nervous system. Part of its proprietary PharmacoSurgery® platform, the company's first drug product, OMIDRIA® (phenylephrine and ketorolac injection) 1%/0.3%, was broadly launched in the U.S. in April 2015 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 lens replacement procedures to maintain mydriasis (pupil dilation), prevent miosis (pupil constriction), and to reduce postoperative eye pain. Omeros has clinical-stage development programs focused on: complement-related thrombotic microangiopathies; complement-mediated glomerulopathies; Huntington's disease and cognitive impairment; addictive and compulsive disorders; and preventing problems associated with urologic surgical procedures. In addition, Omeros has a proprietary G protein-coupled receptor (GPCR) platform, which is making available an unprecedented number of new GPCR drug targets and corresponding compounds to the pharmaceutical industry for drug development, and a platform used to generate antibodies.

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,” “goal,” “intend,” “look forward to,” “may,” “plan,” “potential,” “predict,” “project,” “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, Omeros’ 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, 2016. 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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Cook Williams Communications, Inc.
Jennifer Cook Williams, 360-668-3701
Investor and Media Relations
jennifer@cwcomm.org