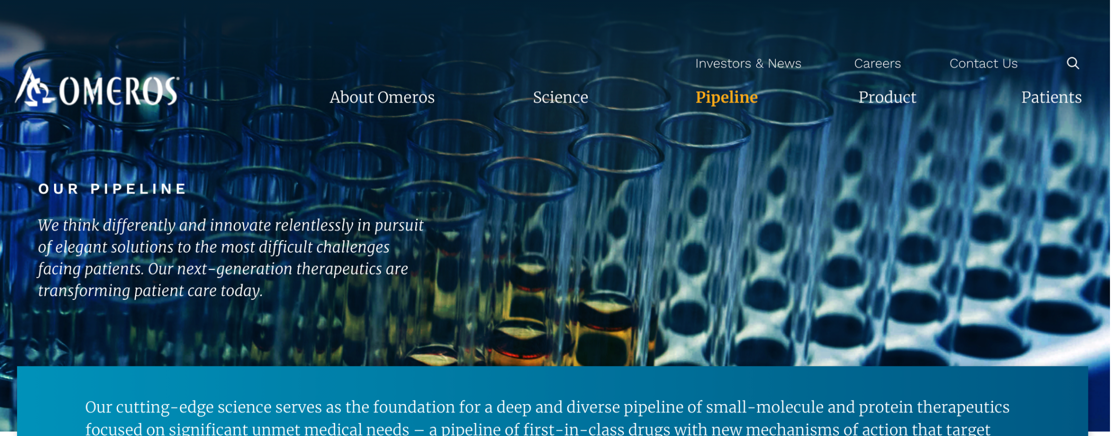

OUR PIPELINE

We think differently and innovate relentlessly in pursuit of elegant solutions to the most difficult challenges facing patients. Our next-generation therapeutics are transforming patient care today.

Our cutting-edge science serves as the foundation for a deep and diverse pipeline of small-molecule and protein therapeutics focused on significant unmet medical needs – a pipeline of first-in-class drugs with new mechanisms of action that target previously untapped receptors and enzymes.

Omeros has retained exclusive control of worldwide commercial rights for all products, product candidates, and programs.

Therapeutic Area
no filters

Phase
no filters

🟦 Antibody
🟧 Small Molecule

Download full PDF
Print

OMIDRIA®
(phenylephrine and ketorolac intraocular solution) 1%/0.3%, for addition to ocular irrigating solution

Marketed

Cataract surgery or intraocular lens replacement

OMIDRIA® is FDA-approved for use during cataract surgery or intraocular lens replacement to maintain pupil size by preventing intraoperative miosis (pupil constriction) and to reduce postoperative ocular pain.

LEARN MORE →

INDICATIONS AND USAGE
OMIDRIA is added to ophthalmic irrigating solution used during cataract surgery or intraocular lens replacement and is indicated for maintaining pupil size by preventing intraoperative miosis and reducing postoperative ocular pain.

IMPORTANT SAFETY INFORMATION
OMIDRIA must be added to irrigating solution prior to intraocular use.

OMIDRIA is contraindicated in patients with a known hypersensitivity to any of its ingredients.

Systemic exposure of phenylephrine may cause elevations in blood pressure.

Use OMIDRIA with caution in individuals who have previously exhibited sensitivities to acetylsalicylic acid, phenylacetic acid derivatives, and other nonsteroidal anti-inflammatory drugs (NSAIDs), or have a past medical history of asthma.

The most commonly reported adverse reactions at ≥ 2% are eye irritation, posterior capsule opacification, increased intraocular pressure, and anterior chamber inflammation.

Please see the **Full Prescribing Information** for OMIDRIA.

You are encouraged to report Suspected Adverse Reactions to the FDA. Visit **www.fda.gov/medwatch**, or call 1-800-FDA-1088.

Narsoplimab
(MASP-2 inhibitor, lectin pathway)

Phase 3

Hematopoietic Stem Cell Transplant-Associated TMA (HSCT-TMA)

Narsoplimab: Our first-in-class monoclonal antibody complement inhibitor

Narsoplimab, also known as OMS721, is a fully human monoclonal antibody targeting mannan-binding lectin-associated serine protease-2 (MASP-2), the effector enzyme of the lectin pathway of the complement system. The lectin pathway is activated primarily by tissue damage or microbial infection. Importantly, unlike other complement-targeting drugs on the market or in development, inhibition of MASP-2 by narsoplimab does not interfere with the classical complement pathway, a critical component of the acquired immune response to infection. Narsoplimab is designed to prevent complement-mediated inflammation and endothelial damage without affecting other pathways of innate immunity.

In a late-stage clinical trial compared to an historical control, narsoplimab improved the estimated median overall survival in high-risk HSCT-TMA patients by an order of magnitude and improved 100-day survival by approximately six-fold.

Breakthrough Therapy and Orphan status

In the US, the FDA has granted narsoplimab the following designations: (1) Breakthrough Therapy designation in patients who have persistent TMA despite modification of immunosuppressive therapy, (2) Orphan Drug designation for the prevention (inhibition) of complement-mediated TMAs and (3) Orphan Drug designation for the treatment of HSCT-TMA.

The European Commission also granted narsoplimab a designation as an Orphan Medicinal Product for treatment in hematopoietic stem cell transplantation.

LEARN MORE ABOUT NARSOPLIMAB →

FIND CLINICAL TRIALS FOR NARSOPLIMAB IN HSCT-TMA HERE →

EXPLORE OUR RESEARCH PROGRAMS IN COMPLEMENT-MEDIATED DISEASE →

Phase 3

IgA Nephropathy (IgAN)

Narsoplimab: Our first-in-class monoclonal antibody complement inhibitor

Narsoplimab, also known as OMS721, is a fully human monoclonal antibody targeting mannan-binding lectin-associated serine protease-2 (MASP-2), the effector enzyme of the lectin pathway of the complement system. The lectin pathway is activated primarily by tissue damage or microbial infection. Importantly, unlike other complement-targeting drugs on the market or in development, inhibition of MASP-2 does not interfere with the classical complement pathway, a critical component of the acquired immune response to infection. Narsoplimab is designed to prevent complement-mediated inflammation and endothelial damage without affecting other pathways of innate immunity.

In a Phase 2 clinical trial, narsoplimab reduced proteinuria in IgA nephropathy patients by 50%-90% and stabilized or increased glomerular filtration rates – effects unmatched by any other drug in development for IgA nephropathy.

Breakthrough Therapy and Orphan status

In the US, the FDA has granted narsoplimab the following designations: (1) Breakthrough Therapy designation for the treatment of IgA nephropathy and (2) Orphan Drug designation in IgA nephropathy.

In Europe, narsoplimab has received from the European Commission designation as an Orphan Medicinal Product for the treatment of primary IgA nephropathy.

LEARN MORE ABOUT NARSOPLIMAB →

FIND CLINICAL TRIALS FOR NARSOPLIMAB IN IGA NEPHROPATHY HERE →

EXPLORE OUR RESEARCH PROGRAMS IN COMPLEMENT-MEDIATED DISEASE →

Phase 3

Atypical Hemolytic Uremic Syndrome (aHUS)

Narsoplimab: Our first-in-class monoclonal antibody complement inhibitor

Narsoplimab, also known as OMS721, is a fully human monoclonal antibody targeting mannan-binding lectin-associated serine protease-2 (MASP-2), the effector enzyme of the lectin pathway of the complement system. The lectin pathway is activated primarily by tissue damage or microbial infection. Importantly, unlike other complement-targeting drugs on the market or in development, inhibition of MASP-2 does not interfere with the classical complement pathway, a critical component of the acquired immune response to infection. Narsoplimab is designed to prevent complement-mediated inflammation and endothelial damage without affecting other pathways of innate immunity.

Phase 2 clinical results are positive and a Phase 3 clinical program is underway.

Fast Track and Orphan status

In the US, the FDA has granted narsoplimab the following designations: (1) Fast Track designation for the treatment of patients with aHUS and (2) Orphan Drug designation for the prevention (inhibition) of complement-mediated TMAs, which includes aHUS.

LEARN MORE ABOUT NARSOPLIMAB →

FIND CLINICAL TRIALS FOR NARSOPLIMAB IN AHUS HERE →

EXPLORE OUR RESEARCH PROGRAMS IN COMPLEMENT-MEDIATED DISEASE →

Phase 2

Lupus Nephritis & Other Renal Diseases

Narsoplimab: Our first-in-class monoclonal antibody complement inhibitor

Narsoplimab, also known as OMS721, is a fully human monoclonal antibody targeting mannan-binding lectin-associated serine protease-2 (MASP-2), the effector enzyme of the lectin pathway of the complement system. The lectin pathway is activated primarily by tissue damage or microbial infection. Importantly, unlike other complement-targeting drugs on the market or in development, inhibition of MASP-2 does not interfere with the classical complement pathway, a critical component of the acquired immune response to infection. Narsoplimab is designed to prevent complement-mediated inflammation and endothelial damage without affecting other pathways of innate immunity.

In a Phase 2 clincial trial, narsoplimab reduced proteinuria by an unprecedented 70% in patients with lupus nephritis. We plan to initiate a Phase 3 program for narsoplimab in lupus nephritis.

LEARN MORE ABOUT NARSOPLIMAB →

EXPLORE OUR RESEARCH PROGRAMS IN COMPLEMENT-MEDIATED DISEASE →

OMS405

(PPARγ)

Phase 2

Opioid and Nicotine Addiction

OMS405: Small-molecule peroxisome proliferator-activated receptor gamma (PPARγ) agonist

In our peroxisome proliferator-activated receptor gamma program, we are advancing PPARγ agonists for the treatment and prevention of addiction to substances of abuse, which may include opioids, nicotine and alcohol. Omeros is the first to demonstrate a link between PPARγ and addiction disorders and has a broad intellectual property estate directed to this target. Data from clinical studies and from animal models of addiction demonstrate that PPARγ agonists could be efficacious in the treatment of a wide range of addictions.

Our collaborators at The New York State Psychiatric Institute have completed two Phase 2 clinical trials related to our PPARγ program. These studies evaluated a PPARγ agonist, alone or in combination with other agents, for treatment of addiction to heroin and to nicotine. Recently published results of the heroin study demonstrated that, although not altering the reinforcing or positive subjective effects of heroin, the PPARγ agonist significantly reduced heroin craving and overall anxiety. The National Institute on Drug Abuse provided substantially all of the funding for these clinical trials and solely oversaw the conduct of these trials. We can use these data for future FDA submissions and retain all rights to the PPARγ program. We have also reported positive results from a Phase 2 clinical trial conducted by an independent investigator evaluating the effects of a PPARγ agonist in patients with cocaine use disorder, which showed decreased cravings and protection of brain white matter.

OMS527

(PDE7 inhibitor)

Phase 1

−

Addictions and Compulsive Disorders; Movement Disorders

OMS527: Small-molecule phosphodiesterase 7 (PDE7) inhibitor

OMS527 is a small-molecule PDE7 inhibitor that modulates dopamine levels in the areas of the brain responsible for addiction. OMS527 reduces craving and relapse across multiple drugs of abuse, including nicotine, opioids, and alcohol, and also blocks binge eating in animal models. Omeros' PDE7 inhibitors do not appear to be addictive or to depress pleasure from normal activities (e.g., social interactions, sex, sports, etc.), both of which are shortcomings of currently marketed addiction therapies.

We reported positive results from our Phase 1 single-ascending- and multiple-ascending-dose clinical trial designed to assess safety, tolerability and pharmacokinetics of our lead compound in healthy subjects. The double-blind, randomized, placebo-controlled trial evaluated six cohorts of eight volunteers in the single-ascending-dose portion of the trial, including a cohort to assess whether pharmacokinetics is affected by food, and three cohorts of 12 volunteers in the multiple-ascending-dose portion. The compound was generally well tolerated with no significant adverse events being reported. The pharmacokinetic data support once-daily dosing, with or without food. Our focus is nicotine addiction and we are planning our Phase 2 development.

EXPLORE OUR RESEARCH PROGRAMS IN CENTRAL NERVOUS SYSTEM DISORDERS →

OMS906

(MASP-3 inhibitor, alternative pathway)

Phase 1

−

PNH and a Wide Range of Other Alternative Pathway Disorders

OMS906: Monoclonal antibody complement inhibitor

OMS906 is a humanized monoclonal antibody targeting mannan-binding lectin-associated serine protease-3 (MASP-3), the key activator of the alternative pathway of complement. The alternative pathway is involved in a wide range of diseases and disorders, including those targeted by marketed alternative pathway inhibitors or those in development. Unlike these drugs, MASP-3 inhibition does not interfere with the classical complement pathway, a critical component of the acquired immune response to infection. A Phase 1 clinical trial is underway.

MASP-2, MASP-3, MASP-2/3

Preclinical

−

Disorders of the Lectin and Alternative Pathways of Complement

Small-molecule inhibitors of MASP-2, MASP-3, MASP 2/3

As part of lifecycle planning for narsoplimab we are developing a long-acting second-generation antibody against MASP-2 designed for subcutaneous administration which is targeted for clinical entry early in 2022, followed by our orally available small-molecule inhibitor of MASP-2. We are also developing small-molecule inhibitors of MASP-3 and bispecific inhibitors of MASP-2/-3.

GPR174

Preclinical

−

Cancer

GPR174: Small-molecule inhibitor of GPR174

GPR174 appears to be a unique target for cancer immunotherapy. Inhibition of GPR174 in human immune cells upregulates T-helper-1 (TH-1) cytokine levels (e.g., IL-2, interferon-γ), reduces expression of tumor-promoting immune checkpoint molecules (e.g., PDL-1, CTLA-4, LAG-3) and other tumor promoters (e.g., amphiregulin), and suppresses tumor-protecting regulatory T cells (Tregs). Based on our data, we believe that GPR174 controls a major pathway in cancer and that modulation of the receptor could provide a seminal advance in immuno-oncologic treatments for a wide range of solid and hematologic tumors. We continue to focus on GPR174 and several other of our GPCR targets with the objective of moving them as rapidly as possible into human trials.

GPR161

Preclinical

−

Cancer

GPR161

GPR161 appears to be involved in the development of a number of different types of cancer.

Related Pages

RESEARCH AREAS →

DISCOVERY TECHNOLOGIES →

©2020 Omeros Corporation, All rights reserved.