



For Immediate Release

CHIMERIX'S BROAD-SPECTRUM ANTIVIRAL CMX001 SHOWN IN ANIMAL MODEL TO BE A POTENT INHIBITOR OF HERPES SIMPLEX VIRUSES

Preclinical Data Demonstrating CMX001 Penetration of Blood-Brain Barrier and Superiority to Acyclovir Against HSV Infection Published in The Journal of Infectious Diseases

RESEARCH TRIANGLE PARK, NC, NOVEMBER 22, 2010 - Chimerix, Inc., a pharmaceutical company developing orally-available antiviral therapeutics, today announced that data demonstrating preclinical efficacy of Chimerix's lead antiviral compound CMX001 against herpes simplex virus (HSV) as well as its ability to penetrate the blood-brain barrier was published in the November 15, 2010 issue of *The Journal of Infectious Diseases*¹.

The paper, "*Efficacy of CMX001 Against Herpes Simplex Virus in Mice and Correlations with Drug Distribution Studies*," details the results of experiments comparing the efficacy of CMX001 and acyclovir, an approved antiviral, in highly predictive preclinical models of HSV and HSV infection. HSV may lead to central nervous system (CNS) infections among infants and herpes encephalitis among children and adults, resulting in significant mortality and permanent neurological damage.

Treatment with CMX001 reduced mortality rates and effectively inhibited HSV replication in a mouse model of disseminated infections and central nervous system infections. Notably, orally-administered CMX001 was shown in this model to penetrate the blood-brain barrier and eradicate signs of virus in brain tissue. In contrast, acyclovir-treated animals continued to show detectable levels of the virus in brain tissue during and after treatment. Researchers concluded that CMX001, because of its unique cellular uptake and biodistribution, holds promise for improved outcomes in the treatment of herpesviruses.

"Herpes simplex virus infections that reach the central nervous system are a significant cause of mortality – particularly among newborns. While use of acyclovir has improved outcomes, the number of fatalities and cases of permanent neurologic impairment is unacceptably high," said Debra C. Quenelle, Ph.D., Associate Professor, The University of Alabama at Birmingham School of Medicine. "CMX001 has demonstrated improved efficacy to acyclovir in reducing viral load and reducing viral replication, particularly in the CNS, in studies of HSV-infected mice which have been highly predictive of outcome in humans. Based on these promising results, CMX001 may have potential for use -- either alone or in combination -- in the treatment of herpes encephalitis, neonatal herpes, or other severe HSV infections in humans."

"These data extend the growing body of evidence for the potential use of CMX001 as a broad-spectrum agent against infections caused by double-stranded DNA viruses," said George

Painter, Ph.D., Chairman and Chief Scientific Officer of Chimerix. “Preclinical and early clinical safety and efficacy data suggest that CMX001 has significant potential for the prevention and treatment of many serious, persistent and potentially fatal diseases.”

Detailed Study Results

Researchers compared the efficacy of CMX001 to acyclovir in BALB/c mice inoculated intranasally with HSV types 1 or 2. CMX001 was effective in reducing mortality against both HSV-1 and HSV-2 infections using doses of 5mg/kg to 1.25mg/kg administered orally once daily, even when treatments were delayed 48–72 hours post viral inoculation. CMX001 administered at 5mg/kg dose and acyclovir at 100mg/kg reduced viral replication in target organs, including lung, liver, kidney and spleen.

CMX001 was more efficacious than acyclovir in reducing viral replication in the CNS in mice infected with either HSV-1 or HSV-2. Specifically, CMX001 reduced viral replication below the limits of detection in all areas of the brain by day three post-infection through ten days post viral infection, which was also three days after cessation of treatment. In contrast, samples obtained from mice treated with acyclovir showed detectable levels of the virus in areas of the brain during treatment. Samples taken from acyclovir-treated mice ten days post viral infection had virus quantities in the brain that were equal to or exceeded the control group, suggesting a rebound of viral replication in these tissues.

Drug distribution studies in uninfected mice showed that CMX001 at 5mg/kg distribution was widespread within four hours of administration and continued through 24 hours. High levels of drug were found early in the lung, liver and spleen, but not in the kidney, without apparent toxicity. CNS distribution was undetectable at low doses, but detectable levels were observed in mice given 10 mg/kg of CMX001. Researchers hypothesize that CMX001’s ability to eradicate virus from brain tissue in spite of its low distribution to the CNS to may be attributed to its potency, and to possibly increased blood-brain barrier penetration associated with infection.

About HSV Infections

HSV infections of the central nervous system (CNS) remain a significant cause of morbidity and mortality in humans, in spite of therapy with acyclovir. HSV, a double-stranded DNA virus, infects approximately 3,200 neonates annually², some of whom will develop CNS disease. HSV encephalitis occurs in children greater than six months of age and in one of 250,000 adults annually in the United States. Acyclovir has been the only FDA-approved drug and is the standard of care for treatment of disseminated infections and CNS HSV infections in neonates and herpes encephalitis in older children and adults. However, mortality still exceeds 25 percent and greater than 50 percent of survivors have significant neurologic impairment.

About Chimerix and CMX001

Chimerix is developing novel antiviral therapeutics with the potential to transform patient care in multiple settings, including transplant, oncology, acute care and global health.

The company’s lead candidate, CMX001, is in Phase 2 clinical studies in immunocompromised transplant and cancer patients for the treatment of life-threatening viruses, including cytomegalovirus and adenovirus. Over 300 people have received CMX001 to date. CMX001 has been well tolerated in all studies, with a growing body of evidence of the compound’s antiviral activity in humans. In Chimerix’s ongoing placebo-controlled studies, CMX001 has been administered to more than 200 patients and healthy volunteers. In addition, at the request

of leading physicians at over 45 medical centers throughout the United States, Canada, Europe and Israel, CMX001 has been administered to more than 120 patients under investigator-held Emergency Investigational New Drug applications (EINDs) for the treatment of a wide range of infections caused by dsDNA viruses for which there are either no approved treatments or where patients have failed the available treatment. To date, CMX001 has been used to treat patients with 12 different dsDNA viral infections across all five families of dsDNA viruses that affect humans. CMX001 is also being developed as a medical countermeasure in the event of a smallpox release. Chimerix has received significant funding from the National Institutes of Allergy and Infectious Disease to develop CMX001 for smallpox.

Chimerix's second clinical-stage antiviral compound, CMX157, has completed Phase 1 clinical studies. CMX157 is in development as a potent nucleoside analogue against multi-drug resistant HIV infections.

Led by a world-class antiviral drug development team, Chimerix is also leveraging the company's extensive chemical library to pursue new treatments for hepatitis C virus, malaria and other global public health needs. For additional information on Chimerix, please visit <http://www.chimerix.com>.

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¹*The Journal of Infectious Diseases* 2010;202:1492–1499 © 2010 by the Infectious Diseases Society of America.

²Kimberlin, DW. *Management of HSV encephalitis in adults and neonates: diagnosis, prognosis and treatment. Herpes* 2007 Jun;14(1):11-6.