



October 6, 2015

Arbutus Presents Preclinical Data at the 2015 International Meeting on Molecular Biology of Hepatitis B Viruses

Preclinical Results Include Initial Combination Data

VANCOUVER, British Columbia and DOYLESTOWN, Pa., Oct. 6, 2015 (GLOBE NEWSWIRE) -- Arbutus Biopharma Corporation (Nasdaq:ABUS), an industry-leading therapeutic solutions company focused on developing a cure for chronic hepatitis B virus infection (HBV), today announced three presentations at the 2015 International Meeting on Molecular Biology of Hepatitis B Viruses being held on October 4 - 8, 2015, at Dolce Bad Nauheim, Germany.

"We are excited to present these data supporting our HBV research and development efforts, in particular, related to TKM-HBV, our lead HBV RNAi clinical candidate, as well as our novel cccDNA formation inhibitors," said Dr. Michael J. Sofia, Arbutus's Chief Scientific Officer. "Our preclinical results validate that the unique three RNAi trigger design of TKM-HBV leads to reductions in hepatitis B surface antigen (HBsAg) and the other viral antigens but also results in a reduction of cccDNA levels, as well as unique synergistic effects of our novel cccDNA inhibitors in combination with nucleot(s)ide analogs."

Presentation Information and Abstract Summaries:

1. "Profiling the Effects of TKM-HBV on cccDNA in Humanized Chimeric Mouse Model of HBV"

Summary: Data utilizing quantitative real-time PCR (qPCR) combined with differential tissue lysis to enable specific detection of cccDNA showed that four weekly doses of TKM-HBV alone at 0.3 mg/kg was able to reduce cccDNA levels by 42% when compared to untreated animals. This confirms the unique three-trigger RNAi product designed to reduce all viral antigens also results in reduction of cccDNA levels.

Date and Time: October 6, 2015, from 7.00am - 9.00am (PT) /10.00 am - 12.00pm (ET)

2. "TKM-HBV, a Novel RNA Interference Treatment for Chronic Hepatitis B, Mediates Global Viral Antigen Reductions through a Well-Defined Mechanism of Action"

Summary: Through a well-characterized mechanism of action, TKM-HBV mediated cleavage of viral RNA transcripts leads to global reduction of all viral antigens from intrahepatic and peripheral compartments within days after a single treatment. In addition to creating a permissive environment for immune response activation by effective suppression of HBsAg, repression of viral proteins such as HBcAg and HBx may also be beneficial through inhibiting cccDNA replication, stability or transcriptional activity. This confirms the unique three-trigger product targets all the HBV mRNA transcripts and leads to reduction of all viral antigens.

Date and Time: October 7, 2015, from 8.00am - 10.00am (PT)/ 11.00am - 1.00pm (PT)

3. "Novel Inhibitors of HBV cccDNA Formation Exhibit Synergistic Effects with Nucleoside and Nucleotide Analogs"

Summary: Multi-dose combinations of ARB-199 and ARB-596 with nucs were examined for cccDNA expression and found to 1) have no antagonistic effects between the two types of compounds, and 2) result in measurable synergy at suboptimal doses of both nucs and cccDNA formation inhibitor compounds. These results suggest that equivalent clinical combinations could potentially result in faster declines of cccDNA levels than is currently obtainable with 'nuc' therapeutics.

Date and Time: October 7, 2015, from 8.00am - 10.00am (PT)/ 11.00am - 1.00pm (PT)

About TKM-HBV

The goal of TKM-HBV is to facilitate HBsAg loss in patients with chronic hepatitis B. The continued presence of HBsAg in chronic HBV is believed to be responsible for disease pathogenesis and impairing the body's ability to clear the virus. Blocking HBsAg may lead to a functional cure by promoting immune-mediated clearance and control of HBV, potentially through HBsAg seroconversion. TKM-HBV is a novel lipid nanoparticle (LNP) formulated RNAi therapy that uniquely targets three highly conserved regions of the HBV viral genome. Targeting multiple sites on the HBV genome allows for potent reduction of multiple viral antigens, knockdown across a broad range of HBV genotypes, and a decrease in the probability of developing antiviral resistance. Preclinical studies with TKM-HBV have shown reductions of HBsAg and other important viral markers across the most prevalent HBV genotypes, demonstrating that TKM-HBV has the potential to treat patients with chronic HBV.

About Arbutus

Arbutus Biopharma Corporation is a biopharmaceutical company dedicated to discovering, developing and commercializing a cure for patients suffering from chronic hepatitis B infection (HBV). Our strategy is to target the three pillars necessary to develop a curative regimen for HBV: suppressing HBV replication within liver cells, stimulating and reactivating the body's immune system so that it can mount an effective defense against the virus and, eliminating the reservoir of viral genomic material known as covalently closed circular DNA, or cccDNA that is the source of HBV persistence. Our portfolio of assets includes a broad pipeline of drug candidates for use in combination to develop a cure for HBV. To support continuous discovery of potential novel drug candidates and technologies, Arbutus has a research collaboration agreement with the Baruch S. Blumberg Institute that provides exclusive rights to in-license any intellectual property generated through the relationship. The Baruch S. Blumberg Institute was established in 2003 by the Hepatitis B Foundation.

Arbutus is headquartered in Vancouver, BC, Canada with offices in Doylestown, PA, USA. For more information, visit www.arbutusbio.com.

CONTACT: Investors

Adam Cutler

Senior Vice President, Corporate Affairs

Phone: 604.419.3200

Email: acutler@arbutusbio.com

Helia Baradarani

Manager, Investor Relations

Phone: 604.419.3200

Email: hbaradarani@arbutusbio.com

Media

Please direct all media inquiries to: media@arbutusbio.com