Bioheart Reports Promising Results From SDF-1 Modified MyoCell(R) Therapy in Heart Failure Study

Pre-Clinical Data Shows Increased Muscle and Blood Vessel Formation in Animals' Hearts

SUNRISE, Fla., July 24, 2008 /PRNewswire-FirstCall via COMTEX News Network/ -- Results of a preclinical study involving the injection of myoblasts modified to express stromal derived factor-1(alpha) protein (SDF-1) into the scar tissue of the myocardium of rats, which suggest improved heart function, were recently presented at two scientific meetings -- the International Society for Magnetic Resonance in Medicine (ISMRM) in Toronto, and the American Society of Gene Therapy Meeting (ASGT) in Boston.

The data, presented by Bijoy Thattaliyath, MD, Post-doctoral Associate, Department of Pediatric Cardiology, University of Florida Health Science Center, showed a decrease of 0.02ml in end systolic volume (0.13ml to 0.11ml) and a 38 percent increase in stroke volume (0.31 ml to 0.43 ml) from three weeks post-myocardial infarction (MI, heart attack) to eight weeks post-cell transplantation, as presented in one of the SDF-1 modified myoblast (SDF-MB) transplanted animals. These results are indicative of 'positive remodeling', or return to normal heart function in the same period. Similar results were found in other study animals. Histopathology confirmed that myoblasts expressing SDF-1 resulted in increased muscle and blood vessel formation in the damaged areas of the hearts, which was not observed in the control.

Additionally, there was an 11.7 percent plus or minus 2.9 percent (p=0.02) absolute improvement in ejection fraction between three weeks post-MI and eight weeks post-cell transplantation in the SDF-MB treated animals. By comparison, and as anticipated, the control arm worsened in the same period. Ejection fraction is the percentage of blood pumped out of the heart's left ventricle with every heartbeat into the body's vasculature. A higher percent ejection fraction means more efficient heart function.

"The study data suggest that SDF-1 modified myoblasts contribute to greater improvement in cardiac function after transplantation compared to non-modified myoblasts," said Dr. Thattaliyath. "This is a significant and encouraging finding, which adds to the growing body of investigational evidence of using autologous myoblasts in the treatment of congestive heart failure and opens the possibility of further study of MyoCell(R) Clinical Therapy."

SDF-1 is a protein with multiple perceived benefits, including angiogenesis (formation of new blood vessels which can provide nutrients to newly engrafted cells) and stem cell homing (attraction of other stem cells to transplantation site for the purpose of helping with the healing process).

The addition of the SDF-1 protein to MyoCell(R) autologous cell therapy results in a genetic modification that may further assist in future treatment for patients suffering from congestive heart failure. Bioheart obtained a worldwide exclusive license from The Cleveland Clinic to patent applications covering certain methods involving SDF-1 filed in the United States and certain foreign countries.

The study, conducted at the University of Florida and partially funded by Bioheart, focused on two primary hypotheses:

-- An increased expression of SDF-1 in the damaged myocardium should provide a strong homing signal for endogenous stem cells involved in myocardial repair.

-- SDF-1 modified myoblasts (SDF-MB) could undergo differentiation in the cardiac environment and assist myocardial performance, and provide a continuous source of SDF-1.

Study objectives included assessment of cardiac function following onset of MI, assessment of cardiac function in the presence and absence of SDF-1 and an assessment of the distribution and phenotype of the transplanted skeletal myoblasts in a total of seven differentiated study groups of approximately five subjects each. MRI assessments were conducted at one, four and eight weeks after the myoblast transplantation.

MyoCell(R) Clinical Therapy is currently being investigated in MARVEL(1), a randomized, double-blind, placebo-controlled, multi-center Phase II/III Trial involving 330 patients. The largest trial of its kind to date, MARVEL is currently enrolling NYHA Class II-IV congestive heart failure patients in approximately 25 of the most important cell therapy centers in the U.S. In the MARVEL Trial, MyoCell(R) is being delivered via a MyoStar(TM) injection catheter(2), in combination with the NOGA(R) XP Cardiac Navigation System. The Principal Investigator for the MARVEL Trial is Warren Sherman, MD, Director, Cardiac Cell-based Endovascular Therapies, Columbia University Medical Center, New York.

"We are very proud of our continued progress with our MyoCell(R) Clinical Therapy and our pre-clinical work with SDF-1," said Howard J. Leonhardt, Bioheart CEO and Chief Technology Officer. "We are currently working with the FDA on obtaining an IND approval for the start of our human trial involving SDF-1."

ABOUT CONGESTIVE HEART FAILURE

Congestive heart failure (CHF), or heart failure, is a condition in which the heart cannot pump enough oxygenated blood to the body's vital organs. People with heart failure find that they cannot exert themselves as they become tired and short of breath. Current therapeutic options include palliative medical therapy (symptom-treating medicine), cardiac assist devices or cardiac transplantation. Heart failure is a leading cause of hospitalizations in people over age 65.

ABOUT MYOCELL(R) CLINICAL CELL THERAPY

MyoCell(R) clinical cell therapy, developed by Bioheart, Inc., is currently being studied as an investigational product in Europe and the U.S. MyoCell(R) clinical cell therapy is intended to be used to improve cardiac function months or even years after a patient has suffered severe heart damage due to a heart attack. The procedure involves a physician removing a small amount of muscle obtained from the patient's thigh. From this muscle specimen, autologous myoblasts (muscle stem cells) are then isolated, grown using Bioheart's proprietary cell-culturing process, and injected directly into the scar tissue of the patient's heart. The myoblast cells are delivered via an endoventricular needle-injection catheter during a minimally invasive procedure performed by an interventional cardiologist or vascular surgeon. The myoblast-based muscle formation in the newly populated regions of scar tissue are intended to improve cardiac function by helping the heart muscle beat more efficiently.

ABOUT BIOHEART, INC.

Bioheart, Inc. (Nasdaq: BHRT) is committed to delivering intelligent devices and biologics that help monitor, diagnose and treat heart failure and cardiovascular diseases. Its goals are to improve a patient's quality of life and reduce health care costs and hospitalizations. Specific to biotechnology, Bioheart is focused on the discovery, development and, subject to regulatory approval, commercialization of autologous cell therapies for the treatment of chronic and acute heart damage. Its lead product candidate, MyoCell(R), is an innovative clinical muscle-derived stem cell therapy designed to populate regions of scar tissue within a patient's heart with new living cells for the purpose of improving cardiac function in chronic heart failure patients. The Company's pipeline includes multiple product candidates for the treatment of heart damage, including Bioheart Acute Cell Therapy, an autologous, adipose tissue-derived stem cell treatment for acute heart damage, and MyoCell(R) SDF-1, a therapy utilizing autologous cells that are genetically modified to express additional potentially therapeutic growth proteins. For more information on Bioheart, visit www.bioheartinc.com.

Footnotes:

(1) MARVEL: A Phase II/III, Double-Blind, Randomized, Placebo-Controlled Multi-center study to Assess the Safety and Cardiovascular Effects of MyoCell Implantation by a Catheter Delivery System in Congestive Heart Failure Patients Post-Myocardial Infarction(s)

(2) The MYOSTAR(TM) Injection Catheter is not available for sale in the U.S. It is in use in IND investigations

Forward Looking Statements:

Except for historical matters contained herein, statements made in this press release are forward-looking and are made pursuant to the safe harbor provisions of the Private Securities Litigation Reform Act of 1995. Without limiting the generality of the foregoing, words such as "may", "will", "to", "plan", "expect", "believe", "anticipate", "intend", "could", "would", "estimate", or "continue" or the negative other variations thereof or comparable terminology are intended to identify forward-looking statements.

Investors and others are cautioned that a variety of factors, including certain risks, may affect our business and cause actual results to differ materially from those set forth in the forward-looking statements. These risk factors include, without limitation, (i) our ability to secure additional financing; (ii) the timely success and completion of our clinical trials; (iii) the occurrence of any unacceptable side effects during or after preclinical and clinical testing of our product candidates; (iv) regulatory approval of our product candidates; (v) our dependence on the success of our lead product candidate; (vi) our inability to predict the extent of our future losses or if or when we will become profitable; (vii) our ability to protect our intellectual property rights; (viii) our inability to predict the extent of our future losses or if or when we will become profitable; and (viii) intense competition . The

company is also subject to the risks and uncertainties described in its filings with the Securities and Exchange Commission, including the section titled "Risk Factors" in its Annual Report on Form 10-K for the year ended December 31, 2007, as amended by Amendment No. 1 on Form 10-K/A and its quarterly report on Form 10-Q for the quarter ended March 31, 2008.

SOURCE Bioheart, Inc.

http://www.bioheartinc.com