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Phase 2 Clinical Trial Data Show Significant Improvements in Outcomes and Symptoms in Advanced Heart Failure Patients Treated with Celladon's Genetically Targeted Enzyme Replacement Therapy MYDICAR®

CUPID Trial Data Presented Today as Late-Breaker at Heart Failure 2010, the Annual Meeting of the Heart Failure Association of the European Society of Cardiology

BERLIN, May 30, 2010 – Celladon Corp., a biopharmaceutical company focused on the discovery and development of innovative treatments for cardiovascular diseases, today announced that six-month data from its Phase 2 clinical trial of MYDICAR® show improvements in clinical outcomes and disease markers in advanced heart failure patients treated with the genetically targeted enzyme replacement therapy.

The study met its primary safety and efficacy endpoints for high dose MYDICAR® versus placebo. The primary efficacy endpoint is a composite endpoint that encompasses the simultaneous assessment of patients' clinical outcomes, exercise tolerance, heart failure symptoms, biomarkers, and cardiac function. Barry Greenberg, M.D., Professor of Medicine at the University of California, San Diego, presented the data in advanced heart failure patients as a late-breaking trial presentation at Heart

Failure Congress 2010, the annual meeting of the Heart Failure Association of the European Society of Cardiology.

“Cardiologists have limited treatment options available for patients with advanced heart failure,” said Dr. Greenberg. “Our data show that patients who received MYDICAR® showed evidence of clinical improvement and had less worsening of heart failure and cardiovascular events requiring hospitalizations. What was most impressive to me was that the effects of MYDICAR® were consistent across a wide range of domains and clinical endpoints that are commonly used to assess the status of heart failure patients. I am very encouraged by these results and optimistic that larger studies will confirm these data. Confirmation of these findings in larger trials would provide us with a much needed new approach for treating patients with advanced heart failure.”

High dose MYDICAR® treated patients had a statistically significant reduction in cardiovascular events as defined by death, the need for left ventricular assist device (LVAD) or cardiac transplant, worsening of heart failure or heart failure related hospitalizations, which translated into a of 50 percent risk-reduction in favor of high dose MYDICAR® (hazard ratio 50 percent versus placebo; $P=0.040$). In addition, the mean duration of hospitalization in the MYDICAR® high dose group during the six-month period was 0.2 days/patient, a substantially shorter period of time than the 2.1 days/patient of the placebo treated group.

Additionally, patients treated with high dose MYDICAR® improved significantly in their heart failure symptoms, their exercise tolerance, serum biomarkers and cardiac function. Specifically, the quality of life worsened by +3.4 points for placebo treated patients but improved by -10.3 points for MYDICAR® treated patients, as measured by the Minnesota Living with Heart Failure® Questionnaire; exercise tolerance, measured with the six-minute walk test, decreased (worsened) in the placebo group by 87 meters but increased by one meter in the MYDICAR® group. A serum biomarker of heart failure, NT-ProBNP, worsened by +5540 pg/mL in placebo treated patients, and improved by -13.5 pg/mL in MYDICAR® treated patients, cardiac function worsened

(heart further enlarged) by 18.2 mL (left ventricular end-systolic volume) in the placebo group, but improved by -9.6 mL in the MYDICAR® group, indicating a reverse remodeling of the damaged heart in the MYDICAR® treated patients.

Krisztina Zsebo, Ph.D., president and CEO of Celladon, said, “The consistency of these improvements across multiple outcomes measures is very encouraging. The positive results of the study suggest that MYDICAR® might have a significant impact on the progression of advanced heart failure, either slowing it down or possibly reversing the course of the disease, which is tremendous progress for more than 350,000 advanced heart failure patients in the United States that are estimated to fall under the CUPID population.”

The CUPID Trial

The CUPID trial (Calcium Up-regulation by Percutaneous administration of gene therapy In cardiac Disease) is a randomized, double-blind, placebo-controlled study to assess the efficacy and safety of MYDICAR®, a genetically targeted enzyme replacement therapy for advanced heart failure. Enrolled patients had severe forms of heart failure defined by New York Heart Association Class III or IV heart failure, significantly impaired pumping function of their hearts (ejection fraction ≤ 35 percent), and less than half the normal ability to transport and utilize oxygen during exercise testing ($VO_2\text{max} \leq 20$ mL/kg/min). The CUPID trial ClinicalTrials.gov Identifier is NCT00454818.

Primary outcome measures included safety, worsening of heart failure leading to hospitalization, frequency of and time to cardiac transplantation or LVAD implantation, changes in patients’ ability to exercise, echocardiographic assessments, a blood test for NT-proBNP, and symptoms of heart failure.

About MYDICAR®

MYDICAR® is a genetically targeted enzyme replacement therapy intended to restore levels of SERCA2a, a regulator of calcium cycling and contractility. SERCA2a levels

decline in all forms of late-stage heart failure resulting in deficient heart function. With MYDICAR®, the SERCA2a gene is delivered using recombinant adeno-associated viral vector (AAV), a naturally occurring virus that is not associated with any disease in humans. MYDICAR® is delivered in a single dose directly to the heart muscle during a short outpatient procedure that is performed in a cardiac catheterization laboratory. MYDICAR® is synergistic and additive across current heart failure treatments such as ACE inhibitors, β -blockers, spironolactone/diuretics, and biventricular pacing devices. No treatment substitution decision is therefore required by the treating physician.

About Heart Failure

Chronic heart failure is an increasingly important health problem. It is the leading medical cause of hospitalization and is expected to result in an estimated direct and indirect cost to the U.S. healthcare system in 2010 of \$39.2 billion. About 5 million people in the U.S. have heart failure, and another 550,000 new cases are diagnosed each year. Heart failure contributes to or causes about 280,000 deaths annually. The most common symptoms of heart failure are shortness of breath, feeling tired and swelling in the ankles, feet, legs and sometimes the abdomen. There is no cure for heart failure.

About Celladon

Celladon Corp., based in La Jolla, Calif., was launched in October 2004 as a privately held biotechnology company with the goal of becoming the leader in developing molecular therapies for the treatment of heart failure. The company's products target calcium cycling and contractility deficit in heart muscle cells. In addition to MYDICAR®, Celladon is developing traditional small molecule activators of SERCA2a for the treatment of heart failure. To learn more about Celladon, visit Celladon's Web site at www.celladon.net.