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UNITED STATES SECURITIES AND EXCHANGE COMMISSION
Washington, DC 20549

FORM 8-K

CURRENT REPORT
Pursuant to Section 13 or 15(d) of
The Securities Exchange Act of 1934

Date of Report (Date of earliest event reported): September 13, 2006

CYTOKINETICS, INCORPORATED

(Exact name of registrant as specified in its charter)

DELAWARE	**000-50633**	**94-3291317**
(State or other jurisdiction of incorporation)	(Commission File Number)	(IRS Employer Identification No.)

280 East Grand Avenue
South San Francisco, California 94080
(Address of principal executive offices, including zip code)

650-624-3000
(Registrant's telephone number, including area code)

(Former name or former address, if changed since last report)

Check the appropriate box below if the Form 8-K filing is intended to simultaneously satisfy the filing obligation of the registrant under any of the following provisions (see General Instruction A.2. below):

☐ Written communications pursuant to Rule 425 under the Securities Act (17 CFR 230.425)

☐ Soliciting material pursuant to Rule 14a-12 under the Exchange Act (17 CFR 240.14a-12)

☐ Pre-commencement communications pursuant to Rule 14d-2(b) under the Exchange Act (17 CFR 240.14d-2(b))

☐ Pre-commencement communications pursuant to Rule 13e-4(c) under the Exchange Act (17 CFR 240.13e-4(c))

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ITEM 8.01. OTHER EVENTS.

Cytokinetics, Incorporated issued a press release announcing data from a first-in-humans Phase I clinical trial evaluating CK-1827452, a novel cardiac myosin activator, administered intravenously. Data from this double-blind, randomized, placebo-controlled, dose-escalation Phase I clinical trial of CK-1827452 were presented at a session entitled "Recent and Late Breaking Trials" at the 10th Annual Meeting of the Heart Failure Society of America in Seattle, Washington. This clinical trial was conducted to investigate the safety, tolerability, pharmacokinetics, and pharmacodynamic profile of a six-hour infusion of CK-1827452 in healthy volunteers. A copy of the press release is being filed with this Current Report on Form 8-K as Exhibit 99.1, and is hereby incorporated by reference under this Item 8.01.

ITEM 9.01. FINANCIAL STATEMENTS AND EXHIBITS.

(c) Exhibits.

The following Exhibit is filed as part of this Current Report on Form 8-K:

Exhibit No.	Description
99.1	Presentation of Phase I Clinical Trial Data Press Release, dated September 13, 2006.

SIGNATURES

Pursuant to the requirements of the Securities Exchange Act of 1934, the registrant has duly caused this report to be signed on its behalf by the undersigned hereunto duly authorized.

CYTOKINETICS, INCORPORATED

By: /s/ Sharon Surrey Barbari
Sharon Surrey-Barbari
Senior Vice President, Finance and Chief Financial Officer

Date: September 13, 2006

EXHIBIT INDEX

Exhibit No.	Description
99.1	Presentation of Phase I Clinical Trial Data Press Release, dated September 13, 2006.

Exhibit 99.1

TO BUSINESS AND MEDICAL EDITORS:

Cytokinetics Announces Presentation of Phase I Clinical Trial Data
for CK-1827452 During Recent and Late Breaking Trials Session
at the 2006 Heart Failure Society of America Annual Meeting

Novel Cardiac Myosin Activator Demonstrates Dose-Dependent Increases in Indices of Cardiac Function

Investor Luncheon and Teleconference Today to Discuss Clinical Results

SOUTH SAN FRANCISCO, Calif., Sept. 13 /PRNewswire-FirstCall/ — Cytokinetics, Incorporated (Nasdaq: CYTK) announced positive data today from a first-in-humans Phase I clinical trial evaluating CK-1827452, a novel cardiac myosin activator, administered intravenously. Data from this double-blind, randomized, placebo-controlled, dose-escalation Phase I clinical trial of CK-1827452 were presented at a session entitled "Recent and Late Breaking Trials" at the 10th Annual Meeting of the Heart Failure Society of America in Seattle, Washington. The presentation was made by John R. Teerlink, M.D., F.A.C.C., F.A.H.A., F.E.S.C., Associate Professor of Medicine at the University of California, San Francisco, and Director of the Heart Failure Clinic, Veterans Affairs Medical Center, San Francisco. Dr. Teerlink was a Co-Principal Investigator and responsible for echocardiographic analysis for the Phase I clinical trial. This clinical trial was conducted to investigate the safety, tolerability, pharmacokinetics and pharmacodynamic profile of a six-hour infusion of CK-1827452 in healthy volunteers.

In this Phase I clinical trial, the maximum tolerated dose (MTD) was determined to be 0.5 mg/kg/hr for the six-hour infusion in healthy volunteers. At this dose, the six-hour infusion of CK-1827452 produced a mean increase in left ventricular ejection fraction of 6.8 absolute percentage points as compared to placebo ($p<0.0001$). At the same dose, CK-1827452 also produced a mean increase in fractional shortening of 9.2 absolute percentage points versus placebo ($p<0.0001$). These increases in indices of left ventricular function were associated with an 84 milliseconds mean prolongation of systolic ejection time ($p<.0.0001$). These mean changes in ejection fraction, fractional shortening and ejection time were dose-proportional across the range of doses evaluated in this clinical trial. In addition, CK-1827452 exhibited linear, dose-proportional pharmacokinetics across the range of doses studied.

At the MTD of 0.5 mg/kg/hr for 6 hours and below, CK-1827452 was well-tolerated when compared to placebo. In addition, CK-1827452 was clinically well tolerated at 0.625 mg/kg/hr for six hours but too few volunteers received this dose to define it as the MTD. At the end of the six-hour infusion at the MTD, mean standing systolic blood pressure fell 13.0 mmHg ($p<0.0001$) and mean supine systolic blood pressure fell 7.4 mmHg ($p<0.05$) versus placebo. At doses up to and

through the MTD, there were no dose-related changes versus placebo in the electrocardiographic PR, QT or corrected QT intervals. Up to and including the MTD, there was no dose-related increase in the overall incidence of adverse events.

At doses above the MTD that were not tolerated, the CK-1827452 infusions were terminated early due to symptoms of chest tightness, light-headedness, palpitations and feeling hot in both subjects treated at 1.0 mg/kg/hr and in one of two subjects treated at 0.75 mg/kg/hr. In these subjects, signs of intolerability included tachycardia (~150 bpm) and electrocardiographic changes. In one volunteer dosed at 1.0 mg/kg/hr, slight, transient increases in the cardiac-specific proteins troponin I & T were observed, but the cardiac-specific fraction of the enzyme creatine kinase remained normal. Subsequent electrocardiograms and echocardiograms returned to normal in this subject, and cardiac magnetic resonance imaging enhanced by gadolinium, a sensitive test for myocardial injury, detected no cardiac abnormality. These effects are believed to be related to an excess of the intended pharmacologic effect, resulting in excessive prolongation of the systolic ejection time, and resolved promptly with discontinuation of the infusions of CK-1827452.

“I was very pleased to share these data today with the heart failure treatment community. I believe these data are encouraging for heart failure patients currently underserved by available treatment options,” stated Dr. John Teerlink. “These Phase I data support the further evaluation of CK-1827452 as a potential novel approach to address limitations of existing treatments for heart failure.”

The Phase I clinical trial activity of CK-1827452 presented today is consistent with results from preclinical models that evaluated CK-1827452 in both normal dogs and dogs with heart failure. In these models, underlying the increases in cardiac function (as determined by dose-dependent increases in fractional shortening) were dose-related increases in the systolic ejection time, which have now also been observed in humans. In a preclinical study evaluating the effects of CK-1827452 in both normal dogs and dogs with heart failure (also presented at this meeting), CK-1827452, at the same dose, produced statistically significantly larger percent increases in stroke volume and cardiac output in dogs with heart failure compared to normal dogs (11% vs. 28% and 2% vs. 13%, respectively, $p < 0.05$). Furthermore, when administered to dogs with heart failure as a 0.25 mg/kg bolus followed by a 72-hour infusion at 0.25 mg/kg/hr, CK-1827452 increased fractional shortening (42%), stroke volume (45%) and cardiac output (32%) without increasing myocardial oxygen consumption.

“We are pleased with the results of this clinical trial. These data track closely with data from our preclinical testing of CK-1827452 and provide useful validation for the underlying therapeutic hypothesis for this novel form of inotropic therapy,” stated Andrew

A. Wolff, M.D., F.A.C.C., Cytokinetics' Senior Vice President of Clinical Research and Development and Chief Medical Officer. "We look forward to initiating a series of Phase II clinical trials to further evaluate the effects of CK-1827452 in various subpopulations of heart failure patients to inform potential registration studies."

In addition, Cytokinetics presented three posters relating to non-clinical data arising from its cardiovascular program. Two posters entitled, "In Vitro and In Vivo Characterization of CK-1827452, a Selective Cardiac Myosin Activator," presented by Kathleen A. Elias, Ph.D., Cytokinetics and "Activating Cardiac Myosin, a Novel Inotropic Mechanism to Improve Cardiac Function in Conscious Dogs with Congestive Heart Failure," presented by You-Tang Shen, M.D., Department of Cell Biology and Molecular Medicine and Cardiovascular Research Institute, New Jersey Medical School, University of Medicine and Dentistry of New Jersey, Newark, NJ, provide supporting data on the preclinical profile of CK-1827452. The first poster contained data demonstrating that CK-1827452, consistent with its mechanism of action, increases contractility in myocytes without increasing calcium and significantly increases cardiac fractional shortening in normal rats, normal dogs and rats with heart failure. The second poster demonstrated that CK-1827452 increased stroke volume and left ventricular fractional shortening in normal dogs and increased cardiac output, stroke volume and left ventricular fractional shortening in dogs with heart failure. In association with the improvement of left ventricular systolic performance, left ventricular filling pressures, heart rate and total peripheral resistance decreased in the dogs with heart failure. In addition, a third poster entitled, "Cardiac Myosin Activator, CK-1316719, Increases Myofibril ATPase Activity and Myocyte Contractility in a Rat Model of Heart Failure," presented by Robert L. Anderson, Cytokinetics, provided data further validating the mechanism of another one of Cytokinetics' cardiac myosin activators.

"We are excited about the totality of both clinical and preclinical data presented this week at the Heart Failure Society of America," added James Sabry, M.D., Ph.D., Cytokinetics' CEO. "Our company is pleased to share these promising results with the scientific and medical communities further underscoring the value of our commitment to novel cytoskeletal drug discovery and development with potential benefit to patients in need."

Investor Luncheon and Presentation Today

Cytokinetics senior management will host an investor luncheon from 12:00 p.m. to 2:00 p.m. PT today in the Menzies Room at the Grand Hyatt Seattle, 721 Pine Street, Seattle, Washington to discuss the results of the Phase I clinical trial of CK-1827452. At the luncheon, a panel of speakers will discuss preclinical and Phase I clinical trial data with CK-1827452 and provide commentary on CK-1827452 and trends in the treatment of heart failure. The panel will include Fady Malik, M.D., Ph.D., F.A.C.C., Director of Cardiovascular Programs at Cytokinetics, Barry Massie, M.D., F.A.C.C., Professor of

Medicine at the University of California, San Francisco and Chief of Cardiology at the Veterans Affairs Medical Center in San Francisco, John R. Teerlink, M.D., F.A.C.C., F.A.H.A., F.E.S.C., Associate Professor of Medicine at the University of California, San Francisco and the Director of the Heart Failure Clinic at the Veterans Affairs Medical Center, San Francisco, and Andrew A. Wolff, M.D., F.A.C.C., Senior Vice President of Clinical Research and Development and the Chief Medical Officer at Cytokinetics. The panel presentation and discussion will be simultaneously webcast beginning at 12:30 p.m. PT and can be accessed in the Investor Relations section of Cytokinetics' website at www.cytokinetics.com . The live audio of the forum is also accessible via telephone to investors, members of the news media and the general public by dialing either (866) 578-5771 (United States and Canada) or (617) 213-8055 (International) and typing in the passcode 82325202.

An archived replay of the webcast will be available via Cytokinetics' website until September 27, 2006. The replay will also be available via telephone from September 13, 2006 at 2:30 p.m. PT until September 27, 2006 by dialing (888) 286-8010 (United States and Canada) or (617) 801-6888 (International) and typing in the passcode 56551325.

Development Status of CK-1827452

A Phase I, first-in-humans clinical trial designed to evaluate CK-1827452, a novel, small-molecule, direct activator of cardiac myosin, was completed with an intravenous formulation in healthy volunteers. In this Phase I clinical trial, the maximum tolerated dose (MTD) was determined to be 0.5 mg/kg/hr for the six-hour infusion in healthy volunteers. At this dose, the six-hour infusion of CK-1827452 produced a statistically significant and clinically relevant increase in ejection fraction and fractional shortening, as measured from baseline to the end of the infusion, in comparison to placebo; these clinically relevant increases in cardiac function were associated with a statistically significant prolongation of systolic ejection time. At the MTD, CK-1827452 was well-tolerated when compared to placebo. Across the dosing levels evaluated in this clinical trial, infusions of CK-1827452 were characterized by linear, dose-proportional pharmacokinetics.

The clinical activity of CK-1827452 in the Phase I clinical trial was consistent with results from preclinical models which evaluated this drug candidate in both normal dogs and dogs with heart failure. In these preclinical models, underlying the increase in ejection fraction and fractional shortening was a dose-related increase in the systolic ejection time, which has now also been observed in humans. Data from a dog model of heart failure demonstrated that CK-1827452 increased cardiac contractility and cardiac output without increasing myocardial oxygen consumption. Preclinical studies have also demonstrated more pronounced effects of CK-1827452 on indices of cardiac function in dogs with heart failure compared to effects achieved in normal dogs.

Cytokinetics initiated a Phase I clinical trial to evaluate the pharmacokinetic profile of CK-1827452, when administered orally, in August of 2006 and expects that CK-1827452 will be entering an international Phase II clinical trials program in patients with heart failure in the second half of 2006. This program is planned to evaluate CK-1827452 in a diversity of patients including those with stable heart failure, inducible ischemia, impaired renal function and acute heart failure. This program is designed to test the safety and efficacy of CK-1827452, in both intravenous and oral formulations, for the potential treatment of heart failure across the continuum of care, both in the hospital and the outpatient settings.

Background on the Heart Failure Market

Heart failure is a widespread and debilitating syndrome affecting approximately five million people in the United States alone. The high and rapidly growing prevalence of heart failure translates into significant hospitalization rates and associated societal costs. The number of hospital discharges in the United States identified with a primary diagnosis of heart failure rose from 550,000 in 1989 to over 1 million in 2003. Heart failure is one of the most common primary discharge diagnoses identified in hospitalized patients over the age of 65 in the United States. The annual costs of heart failure in the United States are estimated to be $29.6 billion, including $19.3 billion for inpatient care. According to industry reports, the U.S. market for heart failure drugs was approximately $1.33 billion in 2004. Despite currently available therapies, readmission rates for patients over the age of 65 remain high at 30 to 40 percent within six months of hospital discharge and mortality rates exceed 50% over the five year period following a diagnosis of acute heart failure. The limited effectiveness of current therapies points to the need for next-generation therapeutics that may offer improved efficacy without increased adverse events.

Background on Cardiac Myosin Activators and Cardiac Contractility

Cardiac myosin is the cytoskeletal motor protein in the cardiac muscle cell that is directly responsible for converting chemical energy into the mechanical force resulting in cardiac contraction. Cardiac contractility is driven by the cardiac sarcomere, a highly ordered cytoskeletal structure composed of cardiac myosin, actin and a set of regulatory proteins, and is the fundamental unit of muscle contraction in the heart. The sarcomere represents one of the most thoroughly characterized protein machines in human biology. Cytokinetics' cardiovascular program is focused towards the discovery and development of small molecule cardiac myosin activators in order to create next-generation treatments to manage acute and chronic heart failure. Cytokinetics' program is based on the hypothesis that activators of cardiac myosin may address certain mechanistic liabilities of existing positive inotropic agents by increasing cardiac contractility without increasing intracellular calcium, which may be associated with adverse clinical effects in heart failure patients. Current inotropic agents, such as beta-adrenergic receptor

agonists or inhibitors of phosphodiesterase activity, increase cardiac cell contractility by increasing the concentration of intracellular calcium, which indirectly activates cardiac myosin; this effect on calcium levels, however, also has been linked to potentially life-threatening side effects. The inotropic mechanism of current drugs also increases the velocity of cardiac contractility and shortens systolic ejection time. In contrast, cardiac myosin activators have been shown to work in the absence of changes in intracellular calcium by a novel mechanism that directly stimulates the activity of the cardiac myosin motor protein. Cardiac myosin activators accelerate the rate-limiting step of the myosin enzymatic cycle and shift the enzymatic cycle in favor of the force producing state. This calcium-independent inotropic mechanism results not in an increase in the velocity of cardiac contraction, but instead, in a lengthening of the systolic ejection time, which results in increased cardiac contractility and cardiac output in a potentially more oxygen-efficient manner.

About Cytokinetics

Cytokinetics is a biopharmaceutical company focused on the discovery, development and commercialization of novel small molecule drugs that specifically target the cytoskeleton. The cytoskeleton is a complex biological infrastructure that plays a fundamental role within every human cell. Cytokinetics' focus on the cytoskeleton enables it to develop novel and potentially safer and more effective classes of drugs directed at treatments for cancer, cardiovascular disease and other diseases. Under a strategic alliance established in 2001, Cytokinetics and GlaxoSmithKline (GSK) are collaborating to develop and commercialize small molecule therapeutics targeting human mitotic kinesins for applications in the treatment of cancer and other diseases. Ispinesib (SB-715992), SB-743921 and GSK-923295 are being developed under the strategic alliance with GSK. GSK is conducting Phase II and Ib clinical trials for ispinesib and a Phase I clinical trial for SB-743921, and Cytokinetics is conducting a Phase I/II trial of SB-743921 in non-Hodgkin's lymphoma. Cytokinetics' unpartnered cardiovascular disease program is the second program to leverage the company's expertise in cytoskeletal pharmacology. Cytokinetics recently completed a Phase I clinical trial with CK-1827452, a novel small molecule cardiac myosin activator, for the intravenous treatment of heart failure and also is advancing CK-1827452 as a potential drug candidate for the treatment of chronic heart failure via oral administration. Additional information about Cytokinetics can be obtained at http://www.cytokinetics.com .

This press release contains forward-looking statements for purposes of the Private Securities Litigation Reform Act of 1995 (the "Act"). Cytokinetics disclaims any intent or obligation to update these forward-looking statements, and claims the protection of the Safe Harbor for forward-looking statements contained in the Act. Examples of such statements include, but are not limited to, statements about the timing, scope and focus of Cytokinetics'

clinical research and development activities with respect to CK-1827452, including potential future clinical trials, the size and growth of expected markets for CK-1827452, the potential benefits of CK-1827452 or Cytokinetics' other drug candidates and potential drug candidates, and the benefits of data obtained from completed clinical trials. Such statements are based on management's current expectations, but actual results may differ materially due to various factors. Such statements involve risks and uncertainties, including, but not limited to, those risks and uncertainties relating to difficulties or delays in patient enrollment for clinical trials, unexpected adverse side effects or inadequate therapeutic efficacy of CK-1827452 or Cytokinetics' other drug candidates and other potential difficulties or delays in development, testing, regulatory approval, production and marketing of CK-1827452 or Cytokinetics' other drug candidates that could slow or prevent clinical development, product approval or market acceptance (including the risks relating to uncertainty of patent protection for Cytokinetics' intellectual property or trade secrets, Cytokinetics' ability to obtain additional financing if necessary and unanticipated research and development and other costs), changing standards of care and the introduction of products by competitors or alternative therapies for the treatment of indications currently or potentially targeted by CK-1827452 and the implementation and maintenance of procedures, policies, resources and infrastructure relating to compliance with new or changing laws, regulations and practices. For further information regarding these and other risks related to Cytokinetics' business, investors should consult Cytokinetics' filings with the Securities and Exchange Commission.

SOURCE Cytokinetics, Incorporated

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