UNITED STATES
SECURITIES AND EXCHANGE COMMISSION
WASHINGTON, D.C. 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): June 1, 2009

# 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) | (I.R.S. Employer Identification No.) |
| 280 East Grand Avenue, South San Francisco, California | | 94080 |
| (Address of principal executive offices) | | (Zip Code) |

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

Not Applicable

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:

[ ] 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.**

On June 1, 2009, Cytokinetics, Incorporated issued a press release announcing that data from two Phase IIa clinical trials evaluating CK-1827452 were presented in the Late Breaking Trials Session and in two poster presentations at the 2009 Heart Failure Congress of the European Society of Cardiology, held May 30-June 2, 2009 in Nice, France. The company believes these results support further clinical development of CK-1827452.

A copy of the press release is filed as Exhibit 99.1 to this Current Report on Form 8-K, and is incorporated herein by reference.

**Item 9.01 Financial Statements and Exhibits.**

(d) Exhibits

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

Exhibit No. Description
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99.1 Press Release, dated June 1, 2009.

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**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

*June 1, 2009*

*By: /s/ Michael S. Rabson*

*Name: Michael S. Rabson*
*Title: Senior Vice President, Business Development and Legal, General Counsel*

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Exhibit Index

| Exhibit No. | Description |
| --- | --- |
| 99.1 | Press release, dated June 1, 2009 |

Contact:

*Christopher S. Keenan*
*Director, Investor & Media Relations*
*(650) 624-3000*

**CYTOKINETICS ANNOUNCES PRESENTATIONS OF DATA FROM PHASE IIA CLINICAL TRIALS OF CK-1827452 AT THE 2009 HEART FAILURE CONGRESS OF THE EUROPEAN SOCIETY OF CARDIOLOGY**

**Results from Two Completed Studies Support Further Clinical Development**

***South San Francisco, CA, June 1, 2009*** – Cytokinetics, Incorporated (Nasdaq: CYTK) announced today that data from two Phase IIa clinical trials evaluating CK-1827452 were presented in the Late Breaking Trials Session and in two poster presentations at the 2009 Heart Failure Congress of the European Society of Cardiology, held May 30-June 2, 2009 in Nice, France. The company believes these results support further clinical development of CK-1827452. Amgen Inc. has exercised its option and has obtained an exclusive, world-wide (except Japan) license to CK-1827452, subject to specified development and commercialization participation rights retained by Cytokinetics.

"I am pleased to have the opportunity to present these data from the first Phase IIa clinical trial of this novel mechanism compound in a Late Breaking Session at the 2009 Heart Failure Congress of the European Society of Cardiology. This rigorously conducted trial of CK-1827452 has generated interesting results consistent with the novel mechanism of action of this drug and support its further development as a possible treatment for patients with heart failure," stated Dr. John McMurray MD, FACC, FRCP, FESC, Professor of Medical Cardiology at the University of Glasgow in Glasgow, Scotland, United Kingdom.

"Data from these key Phase IIa clinical trials evaluating CK-1827452 support our therapeutic hypothesis for this potentially important new drug candidate in this complex disease population," stated Andrew A. Wolff, MD, FACC, Cytokinetics' Senior Vice President of Clinical Research and Development and Chief Medical Officer. "We believe these two clinical trials, together with others we have conducted with CK-1827452, have established a solid basis for further clinical development."

**Late Breaking Trials Session**

A Late Breaking Trials presentation titled "The Selective Cardiac Myosin Activator, CK-1827452, Increases Systolic Function in Heart Failure" was presented on Monday, June 1, by John McMurray, MD, FACC, FRCP, FESC, Professor of Medical Cardiology at the University of Glasgow in Glasgow, Scotland, United Kingdom. The clinical trial was multi-center, double-blind, randomized, and placebo-controlled. Its primary objective was to evaluate the safety and tolerability of CK-1827452 administered as an intravenous infusion to stable heart failure patients. Its secondary objectives were to establish a relationship between plasma concentration and pharmacodynamic effect of CK-1827452 and to determine the pharmacokinetics of CK-1827452 in this population. Overall, a total of 151 treatment periods were initiated in 45 patients.

The authors concluded that CK-1827452 significantly increases systolic ejection time, stroke volume, and ejection fraction calculated by a hybrid method employing Doppler-derived stroke volume in a concentration dependent manner. Statistically significant increases were demonstrated in systolic ejection time at plasma concentrations greater than 100 ng/mL, in stroke volume at plasma concentrations greater than 200 ng/mL, and in hybrid ejection fraction at plasma concentrations greater than 300 ng/mL. At plasma concentrations greater than 400 ng/mL, increases in stroke volume appeared to plateau in association with a concentration-dependent decline in heart rate. In addition, the data demonstrated statistically significant correlations between increasing CK-1827452 plasma concentration and decreases in left ventricular end-systolic volume and left ventricular end-diastolic volume. For the patients that were tolerant of all study drug infusions, no consistent pattern of adverse events with either dose or duration of infusion emerged.

This presentation included the first public disclosure of analyses showing that patients with reduced stroke volumes (< 50 mL) at baseline had pharmacodynamic responses to CK-1827452 that were generally greater than those in patients with greater stroke volumes at baseline, demonstrating robust pharmacodynamic activity in this more severely affected sub-population of patients from the study. The authors concluded that these findings support further study and translation of this novel mechanism into patients with heart failure.

**Poster Presentations**

A poster titled "Echocardiographic Detection of Increases in Ejection Fraction in Patients with Heart Failure Receiving the Selective Cardiac Myosin Activator, CK-1827452" was displayed on Sunday, May 31, 2009 and was presented by Jonathan H. Goldman, MD, FACC, Chief Medical Officer, ICON Medical Imaging, Warrington, PA. This poster included the first public disclosure of analyses comparing the effect of CK-1827452 on ejection fraction calculated from two ventricular volumes assessed by the biplane Method of Discs and two hybrid methods that use a measurement of stroke volume based on Doppler interrogation of the left ventricular outflow tract and a single assessment of ventricular volume by the Method of Discs. All three measurements of ejection fraction increased with the plasma concentration of CK-1827452; however, increases of greater magnitude were observed with the hybrid methods. Ejection fraction assessed by the hybrid methods correlated better with systolic ejection time than did ejection fraction assessed by the Method of Discs. Ejection fraction by the hybrid method based on left ventricular end-systolic volume was slightly better-correlated with systolic ejection time than the hybrid ejection fraction based on left ventricular end-diastolic volume.

A poster titled "A Phase II Safety Study Evaluating the Novel Cardiac Myosin Activator, CK-1827452, in Patients with Ischemic Cardiomyopathy and Angina" was displayed on Sunday, May 31, and was presented by Barry H. Greenberg, MD, Chair of the Safety Review Committee for this clinical trial and Director, Advanced Heart Failure Treatment Program, University of California, San Diego Medical Center. This poster was the first public presentation of data from a double-blind, randomized, placebo-controlled Phase IIa clinical trial evaluating the effect of CK-1827452 on symptom-limited exercise tolerance in heart failure patients with ischemic cardiomyopathy and angina, and included the first detailed public disclosure of specific adverse events in the trial. The study was the subject of a Cytokinetics press release in December 2008, in which the safety data were summarized.

The primary safety endpoint of this clinical trial was the proportion of patients who stopped their exercise treadmill test due to angina at a stage earlier than the shorter of two baseline exercise treadmill tests during double-blind therapy with CK-1827452 or placebo. The authors concluded that in heart failure patients with ischemic cardiomyopathy and angina, who theoretically could be most vulnerable to the possible deleterious consequences of systolic ejection time prolongation, treatment with CK-1827452 at concentrations that increase cardiac function did not deleteriously affect a broad range of safety assessments in the setting of exercise. Cytokinetics believes that the results of this trial, together with previous studies evaluating the pharmacodynamic effects of CK-1827452 in healthy volunteers and stable heart failure patients, support further clinical assessments of CK-1827452 in patients with heart failure.

**Development Status of CK-1827452**

CK-1827452 has been the subject of a clinical trials program comprised of multiple Phase I and Phase IIa trials. This program was designed to evaluate the safety, tolerability, pharmacodynamics and pharmacokinetic profile of both intravenous and oral formulations of CK-1827452 for the potential treatment of heart failure across the continuum of care, in both hospital and outpatient settings. Two Phase IIa clinical trials of CK-1827452 from this program have been completed, and two Phase IIa clinical trials of CK-1827452 are ongoing.

In addition, Cytokinetics has conducted five Phase I clinical trials of CK-1827452 in healthy subjects: a first-time-in-humans study evaluating an intravenous formulation, an oral bioavailability study evaluating both intravenous and oral formulations, and three studies of oral formulations: a drug-drug interaction study, a dose proportionality study and a study evaluating modified-release formulations. Data from each of these trials have been reported previously.

**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. 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 further activates the cardiac sarcomere. 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 contraction 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 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 function in a potentially more oxygen-efficient manner.

**About Cytokinetics**

Cytokinetics is a clinical-stage biopharmaceutical company focused on the discovery and development of novel small molecule therapeutics that modulate muscle function for the potential treatment of serious diseases and medical conditions. Cytokinetics' cardiac muscle contractility program is focused on cardiac muscle myosin, a motor protein essential to cardiac muscle contraction. Cytokinetics' lead compound from this program, CK-1827452, a novel small molecule cardiac muscle myosin activator, is in Phase II clinical trials for the treatment of heart failure. In mid-2009, Cytokinetics plans to initiate a Phase I clinical trial of CK-2017357, a fast skeletal muscle troponin activator, in healthy volunteers in the United States. CK-2017357 is being developed as a potential treatment for diseases and medical conditions associated with aging, muscle wasting, and neuromuscular dysfunction. In January 2009, Cytokinetics announced the selection of a potential drug candidate directed towards smooth muscle contractility. Cytokinetics' smooth muscle myosin inhibitors have arisen from research focused towards potential treatments for diseases and conditions, such as systemic hypertension, pulmonary arterial hypertension or bronchoconstriction.

Cytokinetics' cancer development programs are focused on mitotic kinesins, a family of motor proteins essential to cell division. Cytokinetics is developing two drug candidates that have arisen from this program, ispinesib and SB-743921, each an inhibitor of kinesin spindle protein. In addition, Cytokinetics and GlaxoSmithKline are conducting research and development activities focused on GSK-923295, an inhibitor of centromere-associated protein E (CENP-E).

All of these drug candidates and potential drug candidates have arisen from Cytokinetics' research activities and are directed towards the cytoskeleton. The cytoskeleton is a complex biological infrastructure that plays a fundamental role within every human cell. Additional information about Cytokinetics can be obtained at 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 Act's safe harbor for forward-looking statements. Examples of such statements include, but are not limited to, statements relating to Cytokinetics' and its partners' research and development programs, including the initiation, design, conduct and results of clinical trials relating to CK-1827452 and Cytokinetics' other drug candidates and the significance of such results; and the properties and potential benefits of CK-1827452 and Cytokinetics' other drug candidates and potential drug candidates. Such statements are based on management's current expectations, but actual results may differ materially due to various risks and uncertainties, including, but not limited to, potential difficulties or delays in the development, testing, regulatory approvals for trial commencement, progression or product sale or manufacturing, or production of CK-1827452 or Cytokinetics' other drug candidates that could slow or prevent clinical development or product approval, including risks that current and past results of clinical trials or preclinical studies may not be indicative of future clinical trials results, patient enrollment for or conduct of clinical trials may be difficult or delayed, including without limitation, due to political instability in countries where clinical trials of CK-1827452 or Cytokinetics' other drug candidates are being conducted, CK-1827452 or Cytokinetics' other drug candidates may have adverse side effects or inadequate therapeutic efficacy, the U.S. Food and Drug Administration or foreign regulatory agencies may delay or limit Cytokinetics' or its partners' ability to conduct clinical trials, and Cytokinetics may be unable to obtain or maintain patent or trade secret protection for its intellectual property; Amgen's decisions with respect to the design, conduct, timing and continuation of development activities for CK-1827452; GSK's decisions with respect to the design, conduct, timing and continuation of development activities for GSK-923295; Cytokinetics may incur unanticipated research and development and other costs or be unable to obtain additional financing necessary to conduct development of its products; standards of care may change rendering CK-1827452 and Cytokinetics' other drug candidates obsolete; others may introduce products or alternative therapies for the treatment of indications Cytokinetics' drug candidates and potential drug candidates may target; and risks and uncertainties relating to the timing and receipt of payments from its partners, including option fees, milestones and royalties on future potential product sales under Cytokinetics' collaboration agreements with such partners. For further information regarding these and other risks related to Cytokinetics' business, investors should consult Cytokinetics' filings with the Securities and Exchange Commission.*

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