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): November 10, 2008

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 November 10, 2008, Cytokinetics, Incorporated issued a press release announcing that interim analyses of data from a Phase IIa clinical trial of CK-1827452 in stable heart failure patients were presented during a Special Program at the 2008 Scientific Sessions of the American Heart Association, held November 8-12, 2008 in New Orleans, LA. CK-1827452 is a novel cardiac myosin activator being developed for the potential treatment of patients with either acutely decompensated or chronic heart failure. CK-1827452 is the subject of a Collaboration and Option Agreement between Cytokinetics and Amgen, Inc. 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 this Current Report on Form 8-K:

Exhibit No. Description

99.1 Press Release, dated November 10, 2008.

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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

November 10, 2008

By: /s/ Sharon Barbari

Name: Sharon Barbari
Title: Senior Vice President, Finance and Chief Financial Officer

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Exhibit Index

Exhibit No.	Description
99.1	Press release, dated November 10, 2008

Contact:
Christopher S. Keenan
Director, Investor Relations
(650) 624-3000

**CYTOKINETICS ANNOUNCES DATA
FROM A CLINICAL TRIAL OF CK-1827452 IN STABLE HEART FAILURE PATIENTS
PRESENTED AT THE 2008 SCIENTIFIC SESSIONS OF THE AMERICAN HEART ASSOCIATION**

*Novel Drug Candidate Demonstrates Statistically Significant Increases in
Systolic Ejection Time, Stroke Volume, Fractional Shortening, Cardiac Output and Ejection Fraction*

South San Francisco, CA, November 10, 2008 – Cytokinetics, Incorporated (Nasdaq: CYTK) announced today that interim analyses of data from a Phase IIa clinical trial of CK-1827452 in stable heart failure patients were presented during a Special Program at the 2008 Scientific Sessions of the American Heart Association, held November 8-12, 2008 at the Ernest N. Morial Convention Center in New Orleans, LA. CK-1827452 is a novel cardiac myosin activator being developed for the potential treatment of patients with either acutely decompensated or chronic heart failure. CK-1827452 is the subject of a Collaboration and Option Agreement between Cytokinetics and Amgen Inc.

Oral Presentation at the 2008 Scientific Sessions of the American Heart Association

The oral presentation entitled, "The Selective Cardiac Myosin Activator, CK-1827452, Increases Systolic Function in a Concentration-Dependent Manner in Patients with Stable Heart Failure" was presented in a Cardiovascular Seminar (#117) entitled "Translational Trials and Strategies: First in Man" on Sunday, November 9, 2008, by John Cleland, MD, FACC, FRCP, FESC, Professor of Cardiology, Castle Hill Hospital, University of Hull, United Kingdom. The presentation included data from 28 patients (eight patients from each of Cohorts 1, 2 and 3 and four patients from Cohort 4) in this ongoing clinical trial.

These interim analyses demonstrated statistically significant increases in systolic ejection time ($p < 0.0001$) and fractional shortening ($p < 0.05$) at CK-1827452 plasma concentrations greater than 100 ng/mL, and statistically significant increases in stroke volume ($p < 0.01$) at CK-1827452 plasma concentrations greater than 200 ng/mL. In addition, there were statistically significant correlations between increasing CK-1827452 plasma concentration and increases in systolic ejection time, stroke volume, fractional shortening (all $p < 0.0001$), and cardiac output ($p < 0.01$). There were also statistically significant correlations between increasing CK-1827452 concentration and decreases in supine and standing heart rate (both $p < 0.0001$) and left ventricular end-systolic volume ($p < 0.05$).

Ejection fraction was calculated by two different methods. The first relies exclusively on two-dimensional (2D) echocardiographic imaging to measure left ventricular end-systolic and end-diastolic volumes. In the second, "hybrid" method, stroke volume (measured using Doppler technology) is divided by the left ventricular end-diastolic volume (assessed by 2D echocardiography). There were statistically significant correlations between increasing CK-1827452 plasma concentration and increases in ejection fraction by both methods ($p < 0.05$ for the 2D method; $p < 0.0001$ for the hybrid method); furthermore, increases in ejection fraction by the hybrid method achieved statistical significance at CK-1827452 plasma concentrations greater than 300 ng/mL.

"I am pleased to have the opportunity to present these data at the Scientific Sessions of the American Heart Association. In particular, additional analysis of the ejection fraction using Doppler-derived stroke volume now demonstrates that this cardiac myosin activator appears to improve all major indices of cardiac systolic function in stable heart failure patients," stated Dr. Cleland. "This novel drug candidate offers a potentially exciting new advance in the treatment of heart failure, which is a debilitating disease affecting millions of people worldwide."

"We continue to be encouraged by the progress of the clinical trials program and by the growing body of human clinical data for CK-1827452," stated Andrew A. Wolff, MD, FACC, Cytokinetics' Senior Vice President of Clinical Research and Development and Chief Medical Officer. "CK-1827452 offers a potential therapeutic option for patients suffering from heart failure, a disease with high mortality and morbidity that costs the health care system billions of dollars in re-hospitalizations and lost productivity."

Development Status of CK-1827452

CK-1827452 is currently the subject of a clinical trials program comprised of multiple Phase I and Phase IIa trials. This program is 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. CK-1827452 is the subject of three Phase IIa clinical trials. The first clinical trial is the above-referenced ongoing Phase IIa clinical trial of CK-1827452 in patients with stable heart failure. The second clinical trial is a Phase IIa trial that is designed to evaluate an intravenous formulation together with an oral formulation of CK-1827452 in patients with ischemic cardiomyopathy and angina. The third clinical trial is an open-label Phase IIa clinical trial that is designed to evaluate an intravenous formulation of CK-1827452 in patients with stable heart failure undergoing clinically indicated coronary angiography in the cardiac catheterization laboratory.

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. 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. 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 output in a potentially more oxygen-efficient manner.

Background on the Heart Failure Market

Heart failure is a debilitating syndrome affecting millions of people in the United States. The growing prevalence of heart failure translates into high hospitalization rates and associated societal costs. In 2004, over 5 million patients carried a diagnosis of chronic heart failure in the United States alone. Many of these patients with chronic heart failure suffer episodic deterioration. The number of diagnosed events of acute heart failure was over 4 million in 2004. These numbers are increasing due to the aging population and an increased likelihood of survival after acute myocardial infarction. The costs to society and the individual attributable to the prevalence of heart failure are high. The annual direct and indirect costs of heart failure on the nation's health care system are estimated to be $35 billion in 2008. A portion of that cost comes from heart failure drugs used to treat both chronic and acute heart failure. Sales of drugs to treat heart failure reached over $1.6 billion in 2004, including $1.3 billion for chronic heart failure and $0.3 billion for acute heart failure. Despite currently available therapies, readmission rates for patients remain as high as 42% within one year of hospital discharge and mortality rates are approximately 60% over the five-year period following a diagnosis of chronic 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.

About Cytokinetics

Cytokinetics is a biopharmaceutical company focused on the discovery, development and commercialization of novel small molecule drugs that may address areas of significant unmet clinical needs. Cytokinetics' cardiovascular disease program is focused to cardiac myosin, a motor protein essential to cardiac muscle contraction. Cytokinetics' lead compound from this program, CK-1827452, a novel small molecule cardiac myosin activator, entered Phase II clinical trials for the treatment of heart failure in 2007. Under a strategic alliance established in 2006, Cytokinetics and Amgen Inc. are performing joint research focused on identifying and characterizing activators of cardiac myosin as back-up and follow-on potential drug candidates to CK-1827452. Amgen has obtained an option for an exclusive license to develop and commercialize CK-1827452, subject to Cytokinetics' development and commercial participation rights. Cytokinetics' cancer program is focused on mitotic kinesins, a family of motor proteins essential to cell division. Under a strategic alliance established in 2001, Cytokinetics and GlaxoSmithKline (GSK) are conducting research and development activities focused on the potential treatment of cancer. Cytokinetics is developing two novel drug candidates that have arisen from this program, *ispinesib* and SB-743921, each a novel inhibitor of kinesin spindle protein (KSP), a mitotic kinesin. Cytokinetics is conducting the Phase I portion of a Phase I/II clinical trial of *ispinesib* as monotherapy as a first-line treatment in chemotherapy-naïve patients with locally advanced or metastatic breast cancer. In addition, Cytokinetics is conducting the Phase I portion of a Phase I/II trial of SB-743921 in patients with non-Hodgkin or Hodgkin lymphoma. GSK has an option for the joint development and commercialization of *ispinesib* and SB-743921. GSK-923295, an inhibitor of centromere-associated protein E (CENP-E) inhibitor, is being developed under the strategic alliance by GSK; GSK began a Phase I clinical trial with GSK-923295 in 2007. In April 2008, Cytokinetics announced the selection of a potential drug candidate directed towards skeletal muscle contractility which may be developed as a potential treatment for skeletal muscle weakness associated with neuromuscular diseases or other conditions. 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' research and development programs, including the design, conduct and results of its clinical trials for CK-1827452 and the potential significance of such results; the size and growth of potential markets for drug candidates arising out of Cytokinetics' heart failure program, including for CK-1827452; and the properties and potential clinical benefits of CK-1827452 and Cytokinetics' other drug candidates and potential drug candidates, including CK-1827452's potential effects on cardiac systolic function. 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 approval 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, but not limited to, difficulties or delays 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; 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 and others may introduce products or alternative therapies for the treatment of indications CK-1827452 or Cytokinetics' other drug candidates and potential drug candidates may target; and risks and uncertainties relating to Amgen's and GSK's decisions as to whether to exercise their respective options and the timing and receipt of payments, including option fees, milestones and royalties on future potential product sales under Cytokinetics' respective agreements with Amgen and GSK. 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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