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 15, 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)	(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 15, 2006 Cytokinetics, Incorporated issued a press release announcing the presentation of Phase I clinical trial data for CK-1827452, and non-clinical data from Cytokinetics' cardiovascular program which is directed towards cardiac myosin activators, at the 2006 Annual American Heart Association Scientific Sessions held from November 12-15, 2006 in Chicago, Illinois. A copy of this press release is being filed with this Current Report on Form 8-K, as Exhibit 99.1, and is hereby incorporated by reference into 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 Press Release, dated November 15, 2006.

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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 15, 2006

By: James H. Sabry

Name: James H. Sabry
Title: Chief Executive Officer

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Exhibit Index

Exhibit No.	Description
99.1	Press Release, dated November 15, 2006

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CYTOKINETICS ANNOUNCES PRESENTATIONS OF CLINICAL TRIAL DATA ON CK-1827452 AND NON-CLINICAL DATA FROM ITS CARDIAC MYOSIN ACTIVATOR PROGRAM PRESENTED AT THE 2006 ANNUAL AMERICAN HEART ASSOCIATION SCIENTIFIC SESSIONS MEETING

South San Francisco, CA, November 15, 2006 – Cytokinetics, Incorporated (Nasdaq: CYTK) announced today that Phase I clinical trial data for CK-1827452, and non-clinical data from Cytokinetics' cardiovascular program which is directed towards cardiac myosin activators, were presented at the 2006 Annual American Heart Association (AHA) Scientific Sessions held from November 12-15, 2006 in Chicago, Illinois.

An oral presentation entitled, "The Selective Cardiac Myosin Activator, CK-1827452, a Calcium-Independent Inotrope, Increases Left Ventricular Systolic Function by Increasing Ejection Time Rather than the Velocity of Contraction," 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. The oral presentation highlighted Phase I clinical trial data for CK-1827452 administered in an intravenous formulation. 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.

As previously announced at the Heart Failure Society of America in Seattle in September, 2006, the data from this Phase I clinical trial indicated that the maximum tolerated dose (MTD) was 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 a mean prolongation of systolic ejection time of 84 milliseconds ($p<0.0001$). 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. The adverse effects at the higher dose levels exceeding the MTD in humans appear similar to the adverse findings observed in the preclinical safety studies which occurred at similar plasma concentrations. 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.

In addition, two oral presentations relating to non-clinical data from Cytokinetics' cardiovascular program directed towards cardiac myosin activators were made at the 2006 Annual American Heart Association Scientific Sessions. One oral presentation entitled, "Activating Cardiac Myosin, a Novel Inotropic Mechanism to Improve Cardiac Function in Conscious Dogs with Congestive Heart Failure," provided supporting data relating to the non-clinical profile of CK-1827452. This presentation contained data demonstrating that CK-1827452, consistent with its mechanism of action, increases cardiac output and stroke volume significantly more in conscious dogs with heart failure than in normal dogs.

In addition, a second oral presentation entitled, "Cardiac Myosin Activator, CK-1316719, Increases Myofibril ATPase Activity and Myocyte Contractility in a Rat Model of Heart Failure," provided data further validating the mechanism of another of Cytokinetics' cardiac myosin activators. This presentation concluded that cardiac myocytes isolated from rats with heart failure do not become desensitized to cardiac myosin activators and that cardiac myofibrils isolated from rats with heart failure respond similarly to a myosin activator in comparison to cardiac myofibrils isolated from experimental controls. These observations are supportive of the use of cardiac myosin activators as a potential therapeutic strategy in the treatment of heart failure.

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"We are pleased to have the opportunity to present these data relating to our cardiac myosin program and our drug candidate, CK-1827452, to the broader cardiology community. These data provide further 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 further exploring CK-1827452 as a potential therapy for the treatment of patients with heart failure."

Development Status of CK-1827452

In August 2006, Cytokinetics initiated a Phase I clinical trial evaluating the pharmacokinetic profile of CK-1827452 when administered orally to healthy volunteers. Pharmacokinetic data from the recently completed Phase I clinical trial of the intravenous formulation of CK-1827452 in healthy volunteers suggests that the half-life of CK-1827452 may be sufficient to support development of an oral dosing formulation. Data from this Phase I clinical trial are anticipated by the end of 2006.

Cytokinetics expects that CK-1827452 will be entering an international Phase II clinical trials program in patients with heart failure before the end of 2006. This program is planned to evaluate the safety and efficacy of CK-1827452 in a diversity of patients including those with stable heart failure, ischemic heart disease, tachycardias, impaired renal function, acutely decompensated heart failure, and patients with chronic heart failure at increased risk for heart failure death and hospital admission. 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 percent 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) and SB-743921 are being developed under the strategic alliance with GSK. GSK is conducting Phase II and Ib clinical trials for *ispinesib* 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, anticipated dates of release of data from clinical trials, the size and growth of expected markets for CK-1827452, the potential benefits of CK-1827452 and Cytokinetics' other drug candidates and potential drug candidates and the benefits of data obtained from completed clinical trials and non-clinical studies. 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. 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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