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): December 1, 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 December 1, 2006 Cytokinetics, Incorporated issued a press release announcing that the results from an oral bioavailability study evaluating CK-1827452, a novel cardiac myosin activator, in healthy volunteers support advancement of an oral formulation of CK-1827452 into Phase II clinical trials. The study was designed as an open-label, four way crossover study to investigate the absolute bioavailability, and the effects of food on bioavailability, of two oral formulations of CK-1827452. 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 December 1, 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

December 1, 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 December 1, 2006.

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CYTOKINETICS ANNOUNCES ORAL BIOAVAILABILITY RESULTS FOR CK-1827452

Study Provides Validation for Advancement of Oral Formulation into Phase II Clinical Trials

South San Francisco, CA, December 1, 2006 – Cytokinetics, Incorporated (Nasdaq: CYTK) announced today that the results from an oral bioavailability study evaluating CK-1827452, a novel cardiac myosin activator, in healthy volunteers support advancement of an oral formulation of CK-1827452 into Phase II clinical trials. The study was designed as an open-label, four way crossover study to investigate the absolute bioavailability, and the effects of food on the bioavailability, of two oral formulations of CK-1827452.

In this study, ten healthy volunteers were enrolled and received in random order CK-1827452 at 0.125 mg/kg administered: (i) as a reference intravenous infusion at a constant rate over one hour, (ii) as a liquid solution taken orally in a fasted state, (iii) as an immediate-release solid formulation taken orally in a fasted state, and (iv) as an immediate-release solid formulation taken orally following consumption of a standard, high-fat breakfast. Pharmacokinetic data from this study demonstrated absolute oral bioavailability of approximately 100% for each of the three conditions of oral administration.

These data suggest relatively little variability in oral absorption between patients and therefore predictable plasma levels with oral administration of CK-1827452, which may help to ensure the safety and tolerability of CK-1827452 during chronic oral administration. Oral absorption while fasting was rapid for the liquid solution and immediate-release solid formulation. The median time to maximum plasma concentration after dosing (T_{max}) was 0.5 hours for the liquid solution and 1 hour for the immediate-release formulation. Food delayed the rate of absorption (median T_{max} 3 hr), but did not diminish the extent of absorption. All four conditions of administration were well tolerated, and there were no serious adverse events.

The oral bioavailability data from this study suggests that absorption of CK-1827452 may have a low susceptibility for pharmacokinetic interactions with drugs metabolized by the cytochrome P450 enzymes in the liver and small intestine. These data also indicate that CK-1827452 is unlikely to interact pharmacokinetically with drugs that inhibit intestinal p-glycoprotein, a protein which acts to limit the absorption of some molecules by pumping them back into the intestinal lumen following their absorption into the epithelial cells of the small intestine. The rapid oral absorption of the immediate-release solid formulation under fasted conditions suggests that a modified-release formulation may be desirable for later-stage development and commercialization. Finally, analysis of the combined pharmacokinetic data from this oral bioavailability study and from the first-in-humans study (in which healthy volunteers received intravenous CK-1827452) supports dosing CK-1827452 both intravenously and orally without requiring adjustment for patient weight.

“We are pleased that this study demonstrated such high levels of bioavailability in humans for CK-1827452 when administered orally,” stated David J. Morgans, Jr., Ph.D, Cytokinetics’ Senior Vice President of Preclinical Research and Development. “These data are consistent with what we had observed in preclinical models and will inform further formulation development and manufacturing activities in 2007.”

“We believe these data further support our Phase II clinical trials program for CK-1827452 that is planned to evaluate both oral and intravenous formulations and is designed to evaluate the pharmacokinetics and pharmacodynamic effects of this novel drug candidate in heart failure patients in the inpatient and outpatient treatment settings,” stated Andrew A. Wolff, M.D., F.A.C.C., Cytokinetics’ Senior Vice President of Clinical Research and Development and Chief Medical Officer.

Development Status of CK-1827452

Data from the first-in-humans Phase I clinical trial of CK-1827452 administered intravenously were previously announced at the Heart Failure Society of America meeting in Seattle in September, 2006 and the American Heart Association Scientific Session in November, 2006. This clinical trial demonstrated 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 dose levels exceeding the MTD in humans appeared 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. Pharmacokinetic data from this completed Phase I clinical trial suggested that the half-life of CK-1827452 was sufficient to support development of an oral dosing formulation.

Based on updated development timelines and plans, Cytokinetics expects that CK-1827452 will be entering an international Phase II clinical trials program in patients with heart failure in late 2006 or early 2007. 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% 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 are conducting research and development activities focused towards the potential treatment of cancer and other indications. Cytokinetics and GSK are continuing collaborative research focused to translational research directed to the mitotic kinesin, centromere-associated protein E (CENP-E). GSK-923295, a CENP-E inhibitor, is being developed under the strategic alliance by GSK; GSK expects to begin clinical trials with GSK-923295 in 2007. Cytokinetics is responsible for the development of *ispinesib* and SB-743921, each a novel inhibitor of the mitotic kinesin, kinesin spindle protein (KSP). *Ispinesib* has been the subject of a broad clinical trials program comprising nine Phase II clinical trials as well as six Phase I or Ib clinical trials. Cytokinetics plans to conduct additional clinical trials with *ispinesib* and 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, and is advancing CK-1827452 in both intravenous and oral formulations for the treatment of heart failure. 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 regarding expected initiation, timing and scope and targeted indications of clinical trials within Cytokinetics' and its partners' clinical development and research programs, including Cytokinetics' clinical research and development activities with respect to CK-1827452, the size and growth of expected markets for CK-1827452, the potential benefits of Cytokinetics' drug candidates and potential drug candidates, enabling capabilities of Cytokinetics' biological focus and the potential 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 decisions by GSK to discontinue its research or development efforts for CENP-E under Cytokinetics' collaboration with GSK, difficulties or delays in patient enrollment for clinical trials, unexpected adverse side effects or inadequate therapeutic efficacy of Cytokinetics' drug candidates, including CK-1827452, and other potential difficulties or delays in development, testing, regulatory approval, production and marketing of Cytokinetics' drug candidates that could slow or prevent clinical development, product approval or market acceptance (including the risks relating to uncertainty of patent protection or trade secret for Cytokinetics' intellectual property, Cytokinetics' ability to obtain additional financing if necessary and unanticipated research and development and other costs), changing standards of care and the introduction by others of products 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.

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