
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 13, 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.

In June 13, 2008, Cytokinetics, Incorporated issued a press release announcing results from three Phase I clinical trials evaluating CK-1827452, a novel cardiac myosin activator, in healthy subjects. One study was designed to investigate the potential for drug-drug interactions with CK-1827452, the second, to evaluate the safety, tolerability and dose proportionality of single and multiple doses of two different strengths of an oral formulation of CK-1827452, and the third, to study the relative bioavailabilities of multiple modified release forms of orally administered CK-1827452.

A copy of the press release is being 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 of this Current Report on Form 8-K:

Exhibit No. Description

99.1 Press release, dated June 13, 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.

June 13, 2008

Cytokinetics, Incorporated

By: *Sharon Barbari*

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

Exhibit Index

<u>Exhibit No.</u>	<u>Description</u>
99.1	Press release, dated June 13, 2008

Contacts:

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Director, Corporate Development
(650) 624-3000

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

**CYTOKINETICS PROVIDES UPDATES FROM
PHASE I CLINICAL TRIALS OF CK-1827452**

***Results from Three Trials
Support Ongoing Clinical Development Program***

South San Francisco, CA, June 13, 2008 – Cytokinetics, Incorporated (Nasdaq: CYTK) announced results from three Phase I clinical trials evaluating CK-1827452, a novel cardiac myosin activator, in healthy subjects. One study was designed to investigate the potential for drug-drug interactions with CK-1827452, the second, to evaluate the safety, tolerability and dose proportionality of single and multiple doses of two different strengths of an oral formulation of CK-1827452, and the third, to study the relative bioavailabilities of multiple modified release forms of orally administered CK-1827452.

The first clinical trial is a single-center, open-label, sequential, parallel group study in healthy male subjects to evaluate the potential for certain drug-drug interactions. The primary objective of this study is to evaluate the effect of *ketoconazole* (a potent inhibitor of the drug-metabolizing enzyme, cytochrome P450 (CYP3A4) at steady-state on the pharmacokinetics of a single oral dose of CK-1827452 in subjects who are either extensive metabolizers (EM) or poor metabolizers (PM) with respect to their defined genotype for CYP2D6, another drug-metabolizing enzyme. The secondary objectives are to evaluate the pharmacokinetic parameters of CK-1827452 administered alone in subjects with PM genotype for CYP2D6 as compared to subjects with EM genotype for CYP2D6 and to evaluate the effect of *diltiazem* at steady-state in subjects with the EM genotype for CYP450 if there is evidence of any significant pharmacokinetic interaction between *ketoconazole* and CK-1827452. In EM subjects, *ketoconazole* caused a modest reduction in the clearance of CK-1827452 and consequently, a modest but statistically significant increase in the elimination half-life of CK-1827452, from 22 to 27 hours ($p < 0.01$). This modest increase in the half-life of CK-1827452 with *ketoconazole* resulted in an approximate 50% increase in the area under the CK-1827452 plasma concentration versus time curve (AUC), which reflects the overall exposure to the study drug, and which was also statistically significant ($p < 0.01$). Importantly, however, the maximum CK-1827452 plasma concentration (C_{max}) was unaffected by *ketoconazole* (65 versus 67 ng/mL). *Diltiazem* had no effect on either the C_{max} or AUC of CK-1827452 when the two were co-administered to EM subjects, although the half-life increased slightly, from 18 to 20 hours ($p < 0.01$).

“The results with *ketoconazole* are encouraging, as they suggest minimal potential for significant drug-drug interactions with CK-1827452,” said Andrew A. Wolff, M.D., F.A.C.C., Cytokinetics’ Senior Vice President of Clinical Research and Development and Chief Medical Officer. “First, we saw no effect of *ketoconazole* on the C_{max} of CK-1827452, and we believe it is the C_{max}, rather than the AUC, which determines the potential for intolerable effects of CK-1827452. Second, the approximately 50% increase in the CK-1827452 AUC due to *ketoconazole* observed in this study is modest by comparison to the increase in AUC observed with many other drugs as a result of interaction with inhibitors of *ketoconazole*; for example, some drugs’ AUCs increase by as much as 25- to 30-fold with *ketoconazole*. Importantly, there was no detectable interaction between CK-1827452 and *diltiazem*, a moderate CYP3A4 inhibitor that reflects the potential of many commonly-used drugs to inhibit CYP3A4 and cause drug-drug interactions.”

This clinical trial continues to enroll PM subjects in order to examine the pharmacokinetics of CK-1827452 in this group. Additional data from this trial comparing the pharmacokinetics of CK-1827452 in subjects with the EM versus the PM genotype for CYP2D6 are expected to be available later in 2008.

The second Phase I clinical trial was a single-center study designed to evaluate the safety, tolerability, and pharmacokinetics of an oral formulation of CK-1827452 administered both as a single oral dose and as multiple oral doses of 10 mg and 30 mg strength capsules of CK-1827452. The primary objective of this study was to evaluate the safety and tolerability of CK-1827452 after a single oral dose and after multiple oral doses to steady-state in healthy men and women. The secondary objective of this study was to evaluate the pharmacokinetics of CK-1827452 after a single oral dose and after multiple oral doses to steady-state and to compare the pharmacokinetic parameters between healthy men and women. CK-1827452 was well-tolerated in the trial, with no drug-related serious adverse events. Dose-proportionality between the 10 mg and 30 mg dose levels was observed in both men and women, both after a single dose and after multiple doses to steady-state, with no differences observed between men and women.

The third Phase I clinical trial was a single-center, two-part, open-label study to evaluate modified release forms of CK-1827452 in healthy subjects. Since an immediate release formulation of CK-1827452 was found to be rapidly absorbed in a previous study in healthy subjects, the purpose of developing these modified release forms is to reduce the rate of drug absorption without significantly affecting the overall bioavailability. The primary objective of this study was to assess the pharmacokinetics and relative bioavailability of three different oral modified release prototypes of CK-1827452 as compared to the immediate release formulation in up to twelve healthy male subjects. The secondary objective of the trial was to determine whether there is an effect of food on the pharmacokinetics of one of these oral modified release prototypes of CK-1827452. The single dose pharmacokinetics of one formulation in both the fasted and fed states demonstrated that it reduced C_{max} as compared to the immediate release formulation without a substantial effect on overall bioavailability. This prototype modified release oral formulation of CK-1827452 has been selected to proceed forward into further clinical testing.

“We are pleased with these data, which support our ongoing Phase II clinical trials program evaluating CK-1827452 for the potential treatment of heart failure,” stated Andrew A. Wolff, M.D., F.A.C.C., Cytokinetics’ Senior Vice President of Clinical Research and Development and Chief Medical Officer. “Our program continues to characterize the pharmacokinetics and pharmacodynamic effects of both oral and intravenous formulations of CK-1827452 in order to enable further study of this novel drug candidate in both the inpatient and outpatient settings.”

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 CK-1827452 in a diversity of patients with heart failure. These ongoing and planned trials are designed to evaluate 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.

In March 2008, Cytokinetics announced positive results from an interim analysis of its first and ongoing Phase IIa clinical trial of CK-1827452 in patients with stable heart failure. The safety data from this interim analysis suggest that the drug was well-tolerated with no serious adverse events reported in heart failure patients exposed to the intended range of doses and plasma concentrations. In addition, data from the first two cohorts demonstrated that, when compared to placebo, CK-1827452 produced statistically significant and clinically relevant increases in Doppler-derived stroke volume and fractional shortening in association with statistically significant prolongations of systolic ejection time. Statistically significant correlations were observed between the increases in each of these three indices of cardiac ventricular function and increases in the plasma concentration of CK-1827452. Left ventricular ejection

fraction, a measurement with high variability in patients with ventricular disease, also increased with increasing plasma concentrations; however, this increase in left ventricular systolic function did not reach statistical significance in these initial cohorts. Heart rate decreased slightly at higher concentrations and blood pressure remained unchanged in the first two cohorts of the Phase IIa trial. Across the doses and associated plasma concentration levels evaluated, the pharmacokinetics of CK-1827452 were generally linear with respect to dose and similar to those observed in the healthy subjects in the first-time-in-humans Phase I trial of CK-1827452. Data from this ongoing clinical trial of CK-1827452 are planned to be presented at the Heart Failure Congress, the annual meeting of the Heart Failure Association of the European Society of Cardiology, to be held June 14-17, 2008 in Milan, Italy.

In addition to these Phase I and Phase IIa trials of CK-1827452, Cytokinetics has initiated another Phase IIa clinical trial designed to evaluate the safety and tolerability of an intravenous and oral formulations of CK-1827452 in patients with ischemic cardiomyopathy. In addition, Cytokinetics has also opened enrollment in a third Phase IIa clinical trial designed to evaluate an intravenous form of CK-1827452 in stable heart failure patients undergoing cardiac catheterization.

Data from the first-time-in-humans Phase I clinical trial of CK-1827452 administered intravenously were previously announced at the Heart Failure Society of America annual meeting in September 2006 and the American Heart Association Scientific Sessions in November 2006. Data from this clinical trial demonstrated that a six-hour intravenous infusion of CK-1827452 produced statistically significant increases in Doppler-derived stroke volume, fractional shortening and left ventricular ejection fraction versus placebo in healthy subjects. Underlying these increases in indices of left ventricular function was a lengthening of the systolic ejection time. These mean changes in stroke volume, ejection fraction, fractional shortening and systolic ejection time were dose-proportional across the range of plasma concentrations evaluated. In addition, CK-1827452 exhibited generally linear, dose-proportional pharmacokinetics across the range of doses studied. At the maximum tolerated dose of 0.5 mg/kg/hr for six hours and below, CK-1827452 was well-tolerated compared to placebo. The adverse effects at intolerable doses in humans appeared similar to the adverse findings observed in the preclinical safety studies and 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 December 2006, Cytokinetics announced the results of a Phase I oral bioavailability study which were further described during a poster session at the 2007 Heart Failure Society of America Annual Meeting. Analyses of the combined pharmacokinetic data from this oral bioavailability study and from the first-time-in-human study (in which healthy subjects received intravenous CK-1827452) supports dosing CK-1827452 both intravenously and orally without requiring adjustment for patient weight.

Background on Amgen Collaboration

In January 2007, Cytokinetics and Amgen announced a strategic collaboration to discover, develop and commercialize novel small-molecule therapeutics that activate cardiac muscle contractility for potential applications in the treatment of heart failure. In addition, Amgen obtained an option to receive an exclusive license to develop and commercialize Cytokinetics' lead drug candidate from its cardiovascular disease program, CK-1827452, and other drug candidates arising from the collaboration, subject to Cytokinetics' development and commercial participation rights. The option is for worldwide license rights, excluding Japan. Under the agreement, Cytokinetics received approximately \$75 million, comprised of a non-refundable up-front license and technology access fee of \$42 million and equity investment of approximately \$33 million.

Research activities under the collaboration are focused on identifying and characterizing activators of cardiac myosin as back-up and follow-on potential drug candidates to CK-1827452. During the initial two-year research term, in addition to performing research at its own expense under the collaboration, Cytokinetics will continue to conduct all development activities for CK-1827452, at its own expense, subject to Amgen's option and according to an agreed development plan. Amgen's option is exercisable during a defined period, the ending of which is dependent upon the satisfaction of certain conditions, primarily the delivery of Phase I and Phase IIa clinical trials data for CK-1827452 in accordance with an agreed plan sufficient to support its progression into Phase IIb clinical development. To exercise its option, Amgen would pay a non-refundable exercise fee of \$50 million and thereafter would be responsible for development and commercialization of CK-1827452 and related compounds, subject to Cytokinetics' development and commercial participation rights. In addition, Cytokinetics may be eligible to receive pre-commercialization and commercialization milestone payments of up to \$600 million on CK-1827452 and other products arising from the research, as well as escalating royalties. Cytokinetics also has the opportunity to earn increased royalties by participating in Phase III development costs. In that case, Cytokinetics could co-promote products in North America and would be expected to play a significant role in the agreed commercial activities. If Amgen elects not to exercise its option on CK-1827452, Cytokinetics may then proceed to independently develop CK-1827452 and the research collaboration would terminate.

Background on the Heart Failure Market

Heart failure is a widespread and debilitating syndrome affecting millions of people in the United States. The high and rapidly growing prevalence of heart failure translates into significant hospitalization rates and associated societal costs. It is estimated that in 2004 5.3 million patients in the United States suffered from chronic heart failure. Many chronic heart failure patients frequently suffer acute episodes. Over 4.5 million patients had a primary or secondary diagnosis of acute heart failure in 2004. These numbers are increasing due to the aging population and an increased likelihood of survival following acute myocardial infarctions. The costs to society attributable to the prevalence of heart failure are high. The annual cost of heart failure to the nation's health care system is estimated to be \$38 billion dollars. 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 \$300 million for acute heart failure. Despite currently available therapies, readmission rates for patients over the age of 65 remain high 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 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. 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 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 mechanism of action 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 may address areas of significant unmet clinical needs. Cytokinetics' development activities are primarily directed to advancing multiple drug candidates through clinical trials with the objective of determining the intended pharmacodynamic effect or effects in two principal diseases: heart failure and cancer. 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 believes that ispinesib has demonstrated clinical activity in Phase II monotherapy clinical trials in breast cancer, ovarian cancer and non-small cell lung cancer and recently initiated an additional 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 on a more dose-dense schedule than previously studied. Cytokinetics is also conducting a Phase I/II trial of SB-743921 on a similar more dose-dense schedule in non-Hodgkin and Hodgkin lymphomas. GSK has obtained an option for the joint development and commercialization of ispinesib and SB-743921. Cytokinetics and GSK are conducting collaborative research activities 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, subject to Cytokinetics' option to co-fund certain later-stage development activities and to co-promote any resulting approved drug in North America. 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 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. Cytokinetics' focus on the cytoskeleton enables it to develop novel and potentially safer and more effective classes of drugs directed at treatments for cancer and cardiovascular disease. 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 Safe Harbor for forward-looking statements contained in the Act. Examples of such statements include, but are not limited to, statements relating to Cytokinetics' and its partners research and development programs, including the design, conduct and results of clinical trials, the expected timing for the availability of clinical trial data and the presentation of clinical trial data; CK-1827452's potential with respect to drug-drug interactions; the size and growth of potential markets for drug candidates arising out of Cytokinetics' heart failure program, including for CK-1827452; the potential benefits of CK-1827452 and Cytokinetics' other drug candidates and potential drug candidates; the enabling capabilities of Cytokinetics' cytoskeletal focus; and Cytokinetics' potential receipt of funds and anticipated role in development and commercialization activities under its collaboration and option agreement with Amgen. 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, 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 clinical trials may be difficult or delayed, Cytokinetics' 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; GSK may decide to postpone or discontinue 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, 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 our partners, including 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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