Cytokinetics Announces Presentation of Preclinical Data Regarding CK-2017357 at the 2012 Experimental Biology Annual Conference

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Presentation Highlights Improvement in Resistance to Muscle Fatigue

SOUTH SAN FRANCISCO, CA, Apr 26, 2012 (MARKETWIRE via COMTEX) --Cytokinetics, Incorporated (NASDAQ: CYTK) announced today that preclinical data regarding CK-2017357 were presented in a poster presentation at the 2012 Experimental Biology Annual Conference in San Diego, California. CK-2017357 is the lead drug candidate from the company's skeletal muscle contractility program. CK-2017357 selectively activates the fast skeletal muscle troponin complex by increasing its sensitivity to calcium, which increases skeletal muscle force in response to neuronal input, and delays the onset and reduces the degree of muscle fatigue.

A presentation titled "The Fast Skeletal Troponin Activator, CK-2017357, Improves Resistance to Fatigue in Healthy, Conscious Rats" was made by Adam Kennedy, Ph.D., Pharmacology Scientist, Cytokinetics and Jeffrey R. Jasper, Ph.D., Head of Pharmacology, Cytokinetics. This poster presentation describes a preclinical study that was designed to assess the effects of CK-2017357 in two models of running fatigue. The first model evaluated treadmill running time, an aerobic fatigue assay, while the second model evaluated rotarod running time, an anaerobic fatigue assay. With regards to treadmill running, the authors noted that rats showed significant improvements of 50% in running time compared to controls when administered CK-2017357 at doses of 10 mg/kg and 20 mg/kg ($p < 0.01$ and $p < 0.05$, respectively). With regards to the rotarod running, the authors found that running time at least doubled following the administration of CK-2017357 at doses of 1 mg/kg and 3 mg/kg ($p < 0.05$ and $p < 0.01$, respectively) while the administration of potential control anti-fatiguing treatments did not improve performance in this test. The authors concluded that skeletal muscle troponin activators, such as CK-2017357, are capable of substantially improving performance in an endurance-type fatigue assay and in an assay that tests motor coordination under moderately fatiguing and increasingly difficult conditions. Taken together, these data suggest a role for CK-2017357 and other skeletal muscle troponin activators in reducing muscle-related fatigue that may have utility in disease conditions in which muscle-related fatigue leads to disability.

"We are pleased that these preclinical data demonstrate the potential of CK-2017357 in resistance of fatigue in animal models," stated Fady I. Malik, MD, PhD, FACC, Cytokinetics' Vice President of Biology and Therapeutics. "This presentation, in combination with the data presented at the 64th Annual Meeting of the American Academy of Neurology, point to the potential role that CK-2017357 may have in improving function and decreasing limitations associated with fatigue in patients with debilitating diseases of impaired muscle function, such as amyotrophic lateral sclerosis."

Development Status of CK-2017357 in Amyotrophic Lateral Sclerosis (ALS)

Cytokinetics is developing CK-2017357, a skeletal muscle activator, as a potential treatment for diseases and conditions associated with aging, muscle wasting or neuromuscular dysfunction. CK-2017357 is currently the subject of a Phase II clinical development program and has been granted orphan drug designation by the U.S. Food and Drug Administration and orphan medicinal product designation from the European Medicines Agency for the potential treatment of ALS, a debilitating disease of neuromuscular impairment. In addition, CK-2017357 has received Fast Track designation from the U.S. Food and Drug Administration for the potential treatment of ALS.

Cytokinetics recently completed a two-part, Phase II safety, tolerability, pharmacokinetic and pharmacodynamic clinical trial of multiple doses of CK-2017357 in ALS patients (CY 4024). Part A of this trial, which was completed in 2011, enrolled 24 patients who were not taking riluzole. Part B of this trial enrolled 25 patients who were concurrently taking riluzole. Yesterday, at the American Academy of Neurology (AAN) 64th Annual Meeting, investigators presented data from Part B of this trial. Part B was identical in design to Part A, except that patients received riluzole at the reduced dose of 50 mg daily. In Part B, CK-2017357 appeared to be safe and well-tolerated dosed daily for two weeks at 125 mg, 250 mg, or 375 mg. Adverse events and clinical assessments during treatment with CK-2017357 appeared similar, with or without co-administration of riluzole. While the trial was not designed or powered to evaluate statistically the effects of CK-2017357 on the various outcome measures that were assessed during the study, a combined analysis of patients from Part A (without riluzole) and Part B suggests encouraging trends that appear dose-related and potentially clinically meaningful in magnitude. These clinically relevant trends were observed in the ALS Functional Rating Scale in its revised form (ALSFRS-R) and in Maximum Voluntary Ventilation (MVV). There were no statistically significant differences in outcomes measures between patients in Part A and those in Part B.

Also at the AAN Annual meeting, investigators presented data from CY 4025, a Phase II, randomized, double-blind, placebo-controlled, multiple dose clinical trial of CK-2017357 in patients with ALS receiving riluzole at the reduced dose of 50 mg daily. The primary objective of CY 4025 was to assess the safety and tolerability of CK-2017357 when administered using this twice-daily dose titration regimen to patients with ALS and to determine if the total daily dose of CK-2017357 could be increased from the 375 mg once daily dose (that had been evaluated in earlier trials of CK-2017357 in patients with ALS) to a target of 250 mg dosed twice daily in patients enrolled in this trial. The authors concluded that the twice-daily dose titration regimen evaluated in the trial was generally safe and well-tolerated, that the majority of patients could be titrated successfully to a CK-2017357 dose level of 250 mg twice daily, and that encouraging trends toward functional improvements were observed on CK-2017357 versus placebo. CY 4025 was not designed or powered to evaluate statistically the effects of CK-2017357 on the various outcome measures that were assessed during the study; nevertheless, increases in ALSFRS-R and MVV were observed on CK-2017357 relative to placebo that were similar in direction and magnitude to those observed in CY 4024.

Cytokinetics has met with the U.S. Food and Drug Administration's Center for Drug Evaluation and Research's Division of Neurology Products and with the European Medicines Agency to discuss its progress in the development of CK-2017357 as a potential treatment for patients with ALS and the company's plans for its further development, including potential registration strategies. Cytokinetics is assessing options that may enable the initiation of a registration program for CK-2017357 and anticipates having additional interactions with U.S. and European regulatory authorities during 2012 to discuss the further development of CK-2017357 as a potential treatment for patients with ALS, including potential registration strategies.

Background on Cytokinetics Skeletal Muscle Contractility Program

Skeletal muscle contractility is driven by the sarcomere, the fundamental unit of skeletal muscle contraction. The sarcomere is a highly ordered cytoskeletal structure composed of skeletal muscle myosin, the cytoskeletal motor that is directly responsible for converting chemical energy into mechanical force, as well as actin, and a set of regulatory proteins, troponins and tropomyosin, which make the actin-myosin interaction dependent on changes in intracellular calcium levels. Cytokinetics' skeletal muscle contractility program is focused to the discovery and development of small

molecule skeletal sarcomere activators and leverages Cytokinetics' expertise developed in its ongoing discovery and development of cardiac sarcomere activators, including the cardiac myosin activator omecamtiv mecarbil, now in Phase II clinical development as a potential treatment for heart failure. CK-2017357, a fast skeletal muscle troponin activator, is the lead drug candidate from the company's skeletal muscle contractility program. CK-2017357 selectively activates the fast skeletal muscle troponin complex by increasing its sensitivity to calcium, leading to an increase in skeletal muscle force. This mechanism of action has demonstrated encouraging pharmacological activity in preclinical models that may relate to the potential treatment of diseases associated with aging, muscle wasting or neuromuscular dysfunction. In addition, CK-2017357 has shown pharmacological activity in healthy volunteers, in patients with ALS, and in patients with peripheral artery disease and claudication. The clinical effects of muscle wasting, fatigue and loss of mobility can range from decreased quality of life to, in some instances, life-threatening complications. By directly improving skeletal muscle function, a small molecule activator of the skeletal sarcomere may potentially enhance physical performance and quality of life in patients suffering from diseases or medical conditions characterized or complicated by muscle weakness or wasting.

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' lead drug candidate from its cardiac muscle contractility program, omecamtiv mecarbil, is in Phase II clinical development for the potential treatment of heart failure. Amgen Inc. holds an exclusive license worldwide (excluding Japan) to develop and commercialize omecamtiv mecarbil and related compounds, subject to Cytokinetics' specified development and commercialization participation rights. Cytokinetics is independently developing CK-2017357, a skeletal muscle activator, as a potential treatment for diseases and conditions associated with aging, muscle wasting or neuromuscular dysfunction. CK-2017357 is currently the subject of a Phase II clinical trials program and has been granted orphan drug designation and fast track status by the U.S. Food and Drug Administration and orphan medicinal product designation by the European Medicines Agency for the potential treatment of amyotrophic lateral sclerosis, a debilitating disease of neuromuscular impairment in which CK-2017357 demonstrated potentially clinically relevant pharmacodynamic effects in Phase IIa trials. Cytokinetics is also conducting research of compounds that inhibit smooth muscle contractility and which may be useful as potential treatments for diseases and conditions associated with excessive smooth muscle contraction, such as bronchoconstriction associated with asthma and chronic obstructive pulmonary disease (COPD). 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 activities, including the conduct, design and results of clinical trials for CK-2017357, the significance and utility of preclinical study and clinical trial results for CK-2017357, anticipated interactions with regulatory authorities, plans with respect to a potential registration program for CK-2017357, and the properties and potential benefits of CK-2017357 and Cytokinetics' other drug candidates and potential drug candidates, including CK-2017357's potential utility in the treatment of patients with ALS. 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 Cytokinetics' 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, Cytokinetics' drug candidates may have adverse side effects or inadequate therapeutic efficacy, the U.S. Food and Drug Administration (FDA) or foreign regulatory agencies may delay or limit Cytokinetics' or its partners' ability to conduct clinical trials, the FDA may not grant CK-2017357 orphan drug market exclusivity even if it is approved for marketing, 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, initiation, conduct, timing and continuation of development activities for omecamtiv mecarbil; Cytokinetics will require significant additional funding to conduct the registration program for CK-2017357 for the potential treatment of ALS and may be unable to obtain such additional funding on acceptable terms, if at all; funding from the National Institute of Neurological Disorders and Stroke may not be available in future periods; Cytokinetics may incur unanticipated research and development and other costs; Cytokinetics may be unable to enter into future collaboration agreements for its drug candidates and programs on acceptable terms, if at all; standards of care may change, rendering Cytokinetics' drug candidates obsolete; competitive products or alternative therapies may be developed by others 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 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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