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NKTR-102 (PEG-Irinotecan) Demonstrates Significant Tumor Growth Inhibition In Multiple Preclinical Tumor Models

Anti-Tumor Activity of NKTR-102 Associated With Increased and Sustained Exposure of Tumor Tissue to the Active Metabolite of Irinotecan

SAN FRANCISCO, Oct 25, 2007 /PRNewswire-FirstCall via COMTEX News Network/ -- Nektar Therapeutics (Nasdaq: NKTR) presented positive preclinical data today on its proprietary product candidate NKTR-102 (PEG-irinotecan) at the AACR-NCI-EORTC International Conference in San Francisco, California. The presentations highlighted the anti-tumor activity and unique pharmacokinetic profile of NKTR-102 in preclinical models of colorectal, lung and breast cancer. NKTR-102 is a PEGylated form of irinotecan developed using Nektar's innovative small molecule PEGylation technology platform and is in Phase 1 clinical development for the treatment of solid tumors.

In the two data presentations today, NKTR-102 inhibited tumor growth by greater than 90% in mouse xenograft models of colorectal (HT29), lung (NCI- H460) and breast (MCF-7) cancers as compared to controls. The studies also demonstrate that Nektar's small molecule PEGylation technology improves the pharmacokinetics of irinotecan by increasing the effective half-life of irinotecan's active metabolite in tumor tissues. In a colorectal cancer model, the half-life was increased to 15 days following NKTR-102 administration versus 4 hours following irinotecan.

"The studies presented today at the 2007 AACR-NCI-EORTC Conference highlight the superior tumor suppression properties of NKTR-102 relative to irinotecan in colorectal, lung and breast cancer models," stated Hoyoung Huh, M.D., Ph.D., Chief Operating Officer and Head of the PEGylation Business Unit at Nektar. "NKTR-102 demonstrates improved half-life and tumor exposure to irinotecan's active metabolite, effectively inhibiting the cellular replication process in solid tumors. These findings validate the potential of our small molecule PEGylation technology platform to improve a wide range of oncolytics in critical cancer treatment regimens."

NKTR-102 Preclinical Studies

Preclinical studies in mouse xenograft tumor models evaluated comparative anti-tumor activity and pharmacokinetics of NKTR-102 and irinotecan as compared to controls. In each model, a single IV administration of NKTR-102 on days 0, 4 and 8 in tumor-bearing mice resulted in substantial tumor growth inhibition in a statistically-significant dose-related manner. Anti-tumor activity was measured as changes in tumor weight, tumor growth inhibition and tumor regression.

In all models, NKTR-102 was well-tolerated with no drug toxicity deaths.

Anti-Tumor Activity of NKTR-102 in Mouse Xenograft Models of Human Colorectal, Lung and Breast Tumors

In the colorectal and lung cancer models, NKTR-102 inhibited tumor growth at all dose levels with 94% tumor suppression at the highest dose of 90 mg/kg relative to controls. Tumor regression was also observed with NKTR-102 at this same dose level, while no regression was observed with irinotecan administration. Irinotecan dosing at 90 mg/kg resulted in only modest tumor growth inhibition of 6% in colorectal and 24% in lung.

In the breast cancer model, NKTR-102 suppressed tumor growth by 92% at all dose levels relative to controls with irinotecan only resulting in tumor growth inhibition of up to 56%.

Favorable Pharmacokinetics of NKTR-102 in Lung and Colorectal Mouse Xenograft Models

Following NKTR-102 administration, exposure to the active metabolite of irinotecan in colorectal and lung tumor tissue was increased substantially relative to irinotecan. In colorectal tumor tissue, the effective half-life of the metabolite was increased from 4 hours with irinotecan to 15 days with NKTR-102. In lung tumor tissue, the effective halflife of the metabolite was increased from 15 hours with irinotecan to 6 days with NKTR-102.

This increased and prolonged metabolite exposure following administration of NKTR-102 was associated with its anti-tumor activity in lung and colorectal models using pharmacokinetic (PK) and pharmacodynamic (PD) modeling. Further, PK/PD

simulations performed with irinotecan administration show that a continuous infusion of irinotecan at 240 mg/kg would be required to achieve the equivalent tumor exposure of the active metabolite and associated tumor growth inhibition found with NKTR-102. This 240 mg/kg dose of irinotecan would be expected to cause significant mortality in mice.

Data Presentations

The two poster presentations made today at the AACR-NCI-EORTC International Conference, and prior presentations for NKTR-102, can be found on Nektar's website at http://www.nektar.com/wt/page/nktr102media

C10: "NKTR-102, a novel polyethylene glycol conjugate of irinotecan, has improved antitumor activity in three mouse xenograft models"

C157: "NKTR-102, a novel PEGylated-irinotecan conjugate, results in sustained tumor growth inhibition in mouse models of human colorectal and lung tumors that is associated with increased and sustained SN38 exposure"

About NKTR-102

Nektar is developing NKTR-102, a PEGylated form of irinotecan, which was invented by Nektar using its world-leading small molecule PEGylation technology platform. The product is currently in Phase 1 clinical development. Irinotecan is an important chemotherapeutic agent used for the treatment of solid tumors, including colorectal and lung cancers. By applying Nektar's small molecule PEGylation technology to irinotecan, NKTR-102 may prove to be a more powerful and tolerable anti-tumor agent.

Nektar PEGylation Platform

Nektar PEGylation technology can enhance the properties of therapeutic agents by increasing drug circulation time in the bloodstream, decreasing immunogenicity and dosing frequency, increasing bioavailability and improving drug solubility and stability. It can also be used to modify pharmaceutical agents to preferentially target certain systems within the body. It is a technique in which non-toxic polyethylene glycol (PEG) polymers are attached to therapeutic agents, and it is applicable to most major drug classes, including proteins, peptides, antibody fragments, small molecules, and other drugs.

Nektar PEGylation technology is also used in eight additional approved partnered products in the U.S. or Europe today, including Roche's PEGASYS(R) for hepatitis C and Amgen's Neulasta(R) for neutropenia.

About Nektar

Nektar Therapeutics is a biopharmaceutical company that develops and enables differentiated therapeutics with its industryleading PEGylation and pulmonary drug development technology platforms. Nektar PEGylation and pulmonary technology, expertise, manufacturing capabilities have enabled nine approved products for partners, which include the world's leading pharmaceutical and biotechnology companies. Nektar also develops its own products by applying its PEGylation and pulmonary technology platforms to existing medicines with the objective to enhance performance, such as improving efficacy, safety and compliance.

This press release contains forward-looking statements regarding the potential of the company's PEGylation technology platform and NKTR-102. These forward-looking statements involve important risks and uncertainties, including but not limited to: (i) preclinical testing and clinical trials for NKTR-102 are long, expensive and uncertain processes, (ii) because the NKTR-102 product development programs are in the early phases of clinical development, the risk of failure is high and can occur at any stage of development, (iii) the company may fail to obtain regulatory approval of NKTR- 102, (iv) potential competition from approved drugs or drugs under development that may be safe and effective for the same indication as that targeted by NKTR-102, and (v) the company's patent applications for NKTR-102 may fail to issue; patents that have issued may not be enforceable; or unanticipated intellectual property licenses from third parties may be required in the future. Other important risks and uncertainties are detailed in the company's reports and other filings with the SEC including its most recent Annual Report on Form 10-K and Quarterly Report on Form 10-Q. Actual results could differ materially from the forward-looking statements, whether as a result of new information, future events, or otherwise. No information regarding or presented at the scientific meetings referred to above (or contained at the Internet links provided) is intended to be incorporated by reference in this press release.

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