Synta Presents New Data on Elesclomol (Formerly STA-4783) at AACR-NCI-EORTC International Conference

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LEXINGTON, Mass.--(BUSINESS WIRE)--Oct. 25, 2007--Synta Pharmaceuticals Corp., (NASDAQ: SNTA) announced it presented new preclinical data on elesclomol (formerly STA-4783) at the AACR-NCI-EORTC International Conference on Molecular Targets and Cancer Therapeutics sponsored by the American Association for Cancer Research (AACR), the National Cancer Institute (NCI) and the European Organization for Research and Treatment of Cancer (EORTC), in San Francisco, CA.

Data presented at the conference confirmed that the primary mechanism of action of elesclomol is through the induction of reactive oxygen species (ROS) and provided details of the sequence of events by which programmed cell death (apoptosis) is induced. The results presented at the conference also provided evidence that elesclomol enhances the efficacy of several leading first-line cancer agents including Rituxan(R), Gemzar(R), and Taxotere(R), with minimal additional toxicity. Finally, the results showed that elesclomol has single agent anti-tumor activity.

"Oxidative stress induction by elesclomol represents a novel and promising anti-cancer strategy," said James Barsoum, Ph.D., Senior Vice President of Research at Synta Pharmaceuticals. "Cancer cells operate at a much higher level of oxidative stress than normal cells. By causing the level of ROS in cancer cells to exceed sustainable levels, elesclomol selectively induces apoptosis in cancer cells, with minimal effects on normal cells. The elevation of ROS also appears to sensitize cancer cells to killing by other agents acting through the mitochondrial apoptosis pathway."

"Taken together, the encouraging results presented at the conference on mechanism and combination activity provide continuing evidence that oxidative stress induction has broad potential application across multiple tumor types and in combination with widely used anti-cancer agents," said Dr. Barsoum. "Importantly for potential clinical applications, elesclomol appears to provide this enhanced combination activity with minimal added toxicity."

Highlights of Preclinical Results

Two posters on elesclomol were presented at the meeting and are also available on the company's website.

In the first poster, additional details on the novel mechanism of action by which elesclomol kills cancer cells were described. Elesclomol was found to rapidly induce oxidative stress in a wide variety of human cancer cell lines, but not in most normal cells. Elesclomol also induced the expression of ROS-regulated genes characteristic of a protective response to oxidative stress. However, this protective response was ultimately futile, and the sustained induction of ROS rapidly led to mitochondrial membrane depolarization, release of cytochrome c and activation of caspase-3. These results demonstrate that induction of oxidative stress by elesclomol activates the

well-characterized intrinsic mitochondrial apoptotic cascade, leading to cancer cell death.

ROS and oxidative stress are known to potentiate the activities of many different anti-cancer therapies. Results from murine tumor model studies presented in a second poster showed that not only was elesclomol active as a single agent, but that it may also enhance several different widely used anti-cancer therapies that act through distinct mechanisms of action. These include the microtubule inhibitor docetaxel (Taxotere(R)), the nucleoside analog gemcitabine HCl (Gemzar(R)), the anti-CD20 monoclonal antibody rituximab (Rituxan(R)) and fractionated ionizing radiation. The data also demonstrated that elesclomol was active not only in melanoma, but also in colon, lung and lymphoma tumor models.

About Elesclomol (Formerly STA-4783)

Elesclomol is a novel, injectable, investigational drug candidate that kills cancer cells by elevating oxidative stress levels beyond a breaking point, triggering programmed cell death. In preclinical models elesclomol showed anti-cancer activity against a broad range of cancer cell types, as well as the ability to enhance the efficacy of certain anti-cancer therapies, with minimal additional toxicity.

In a recent 21-center, double-blind, randomized, controlled Phase 2b clinical trial in 81 patients with metastatic melanoma, elesclomol in combination with paclitaxel met the primary endpoint - doubling the median time patients survived without their disease progressing - compared to paclitaxel alone ($p = 0.035$). The most common adverse events in the elesclomol plus paclitaxel group included fatigue, alopecia, constipation, nausea, hypoaesthesia, arthralgia, insomnia, diarrhea, and anemia. Synta is initiating a confirmatory Phase 3 clinical trial of elesclomol in combination with paclitaxel in metastatic melanoma (the SYMMETRY(SM) trial). Phase 2 trials in other indications, and in combination with other agents, are planned. Elesclomol has received Fast Track designation from the FDA for development in metastatic melanoma.

About Synta Pharmaceuticals

Synta Pharmaceuticals Corp. is a biopharmaceutical company focused on discovering, developing, and commercializing small molecule drugs to extend and enhance the lives of patients with severe medical conditions, including cancer and chronic inflammatory diseases. Synta has a unique chemical compound library, an integrated discovery engine, and a diverse pipeline of clinical- and preclinical-stage drug candidates with distinct mechanisms of action and novel chemical structures. All Synta drug candidates were discovered and developed internally. Synta has a global collaboration agreement with GlaxoSmithKline (GSK) for the joint development and commercialization of its lead investigational drug candidate, elesclomol. For more information, please visit www.syntapharma.com.

Safe Harbor Statement

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from those expressed or implied by such forward-looking statements, including those described in "Risk Factors" of our Form 10-K for the year ended December 31, 2006 as filed with the Securities and Exchange Commission. Synta undertakes no obligation to publicly update forward-looking statements, whether because of new information, future events or otherwise, except as required by law.

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