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): **April 4, 2011**

SYNTA PHARMACEUTICALS CORP.

(Exact name of registrant as specified in its charter)

Delaware	**001-33277**	**04-3508648**
(State or other jurisdiction of incorporation)	(Commission File Number)	(IRS Employer Identification No.)

45 Hartwell Avenue
Lexington, MA 02421
(Address of principal executive offices and zip code)

Registrant's telephone number, including area code: **(781) 274-8200**

(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))

ITEM 8.01 Other Events.

On April 4, 2011, Synta Pharmaceuticals Corp. issued a press release announcing that it presented three posters at the American Association for Cancer Research (AACR) 102nd Annual Meeting reporting results from studies of ganetespib (STA-9090), a novel Hsp90 inhibitor. A copy of the press release is filed as Exhibit 99.1 to this Current Report on Form 8-K and is incorporated herein by reference.

On April 4, 2011, Synta Pharmaceuticals Corp. issued a press release announcing that it presented two posters at the American Association for Cancer Research (AACR) 102nd Annual Meeting reporting results from studies of elesclomol, a small-molecule mitochondria metabolism inhibitor. A copy of the press release is filed as Exhibit 99.2 to this Current Report on Form 8-K and is incorporated herein by reference.

ITEM 9.01 Financial Statements and Exhibits.

(d) Exhibits.

Exhibit Number	Description
99.1	Press Release, dated April 4, 2011 — Synta Announces Ganetespib Results at AACR - Inhibition of Multiple Oncogenes and Resistance Mechanisms Leads to Potent Activity in NSCLC and Strong Enhancement of Radiation Therapy
99.2	Press Release, dated April 4, 2011 — Synta Announces Results Presented at AACR Further Elucidate Mechanism by Which Elesclomol Targets Cancer Cell Metabolism

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.

SYNTA PHARMACEUTICALS CORP.

Dated: April 4, 2011

/s/ Keith S. Ehrlich
Keith S. Ehrlich
Vice President, Finance and Administration
Chief Financial Officer

EXHIBIT INDEX

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99.2	Press Release, dated April 4, 2011 — Synta Announces Results Presented at AACR Further Elucidate Mechanism by Which Elesclomol Targets Cancer Cell Metabolism

Exhibit 99.1

Synta Pharmaceuticals Corp.
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Synta Announces Ganetespib Results at AACR — Inhibition of Multiple Oncogenes and Resistance Mechanisms Leads to Potent Activity in NSCLC and Strong Enhancement of Radiation Therapy

-Simultaneous inhibition of multiple oncogenes present in NSCLC including EML4-ALK, EGFR, MET, AKT, MEK, ERK, STAT3, and HER2 leads to potent activity in a broad range of NSCLC models-

-Inhibition of DNA repair and cell cycle checkpoint resistance mechanisms enhances activity of radiation therapy-

-Clinical trial as radiosensitizer to start later in 2011-

LEXINGTON, MA — April 4, 2011 — Synta Pharmaceuticals Corp. (NASDAQ: SNTA) today presented five posters at the American Association for Cancer Research (AACR) 102nd Annual Meeting. Three posters reported results from studies of ganetespib (STA-9090), and two posters reported results from studies of elesclomol, a small-molecule mitochondria metabolism inhibitor.

Ganetespib is a potent inhibitor of heat shock protein 90 (Hsp90) currently being studied in a broad range of clinical trials, with over 350 patients treated to date and a Phase 2b/3 trial in non-small cell lung cancer (NSCLC) expected to start this quarter. Ganetespib is structurally unrelated to earlier Hsp90 inhibitors such as 17-AAG and has shown superior activity to these agents in preclinical studies. In clinical trials, ganetespib has shown single-agent activity in multiple tumor types and an absence of the serious liver and ocular toxicities seen with other Hsp90 inhibitors.

"The results presented today shed further light on the strong activity seen with ganetespib both in preclinical models and in patients," said Vojo Vukovic, M.D., Ph.D., Senior Vice President and Chief Medical Officer, Synta Pharmaceuticals. "The simultaneous inhibition of multiple critical oncogenes, and the potent anti-cancer activity seen in lung cancer models with distinct genetic profiles, suggests broad potential application for ganetespib in NSCLC. These results provide insight into our ongoing Phase 2 trial in NSCLC, in which a number of patients who have failed prior therapies such as carboplatin, Taxol, Avastin, Tarceva, and Alimta achieved durable objective responses or tumor shrinkage with single-agent ganetespib."

"The findings that ganetespib can inhibit critical components of repair mechanisms to the double-strand DNA breaks induced by irradiation are very encouraging and suggest important potential application as a radiosensitizer," continued Dr. Vukovic. "There is a high unmet need for improving the outcome of radiotherapy in human cancers: 50-60 percent of all cancer patients receive radiotherapy as part of their treatment. The favorable safety profile observed with ganetespib in the clinic is a critical advantage for this application. We have established a strong collaboration with a leading radiotherapy group, which will be initiating a clinical trial of ganetespib as a radiosensitizer later this year."

"Our ganetespib clinical program has benefited not only from thorough *in vitro* and *in vivo* studies, but also from a Phase 1 clinical trial in dogs with spontaneous cancers," continued Dr.

Vukovic. "The results from our collaborators at Ohio State University have been instrumental in helping us think through clinical trial choices for ganetespib on dose, schedule, and biomarker evaluation. The single-agent activity seen in dogs with cancer has been encouraging, and consistent with single-agent activity that has been seen in human clinical trials."

Synta is currently conducting a Phase 2 clinical trial of ganetespib as a single agent in NSCLC. A Phase 2b/3 trial of ganetespib in combination with docetaxel in 2nd-line advanced NSCLC patients is expected to initiate in Q2 of 2011 with preliminary results from the Phase 2b portion of the trial expected either late this year or in Q1 2012. Additional details regarding upcoming trials will be announced as those trials initiate.

In January of this year, the Multiple Myeloma Research Foundation announced funding of up to $1 million to support a study of ganetespib in combination with bortezomib (VELCADE®) in multiple myeloma.

Ganetespib posters and publications are available at www.syntapharma.com or by contacting Synta directly.

Ganetespib (STA-9090) Posters

Title: Potent anticancer actions of the Hsp90 inhibitor STA-9090 in wild-type EGFR models of lung cancer

Poster Presentation April 4, 8:00 a.m. ET
Abstract Number: 1638

Abstract:

Non-small cell lung cancer (NSCLC) is a heterogeneous disease that can be sub-classified based on the specific alterations in oncogenes that drive it. While EGFR and KRAS are most often implicated in the molecular epidemiology of NSCLC, aberrations in several other genes have been shown to contribute to oncogenesis. These include mutation and/or amplification of MET, mutation in BRAF or chromosomal rearrangements involving ALK. Targeted therapy against these kinases has shown signs of therapeutic success; however, acquired drug resistance universally develops.

Heat Shock Protein 90 (Hsp90) is a molecular chaperone that mediates the post-translational stability of its protein substrates, many of which are validated oncogenes. Hsp90 is emerging as an important target in cancer therapy because its inactivation results in the abrogation of multiple signaling pathways simultaneously, irrespective of the mutational status of its substrate. STA-9090 is a second-generation, synthetic, small-molecule Hsp90 inhibitor that has shown potent and selective activity preclinically and is currently in Phase 2 trials in a number of indications. We show here that in the presence of STA-9090, upregulation of the MET pathway, either through transient stimulation by its ligand, HGF, or through amplification of MET itself, is incapable of maintaining survival in EGFR-inhibitor-resistant NSCLC. To identify additional genetic lesions sensitive to Hsp90 inhibition, we screened a panel of wild-type EGFR NSCLC cell lines for viability in the presence of STA-9090. All the cell lines assayed, driven by mutations in genes such as PDGFR α, BRAF, PI3K and EML4-ALK or amplification of wild-type EGFR, were sensitive to STA-9090, with IC50 values between 10 and 150 nM.

Further analysis demonstrated that STA-9090 potently destabilized the oncogenic driver for each cell line. *In vivo*, STA-9090 showed strong single-agent activity in xenograft models of

human NSCLC carrying either a BRAF mutation or EML4-ALK fusion, in accordance with the sensitivity of these client proteins to the effects of STA-9090 action. Inhibition of Hsp90 activity therefore presents a promising approach for combating NSCLC induced by mutations in genes other than EGFR, as well as by compensatory pathways upregulated in the context of EGFR-inhibitor resistance.

Title: Novel Hsp90 inhibitor, STA-9090, for combination with radiotherapy

Poster Presentation April 4, 1:00 p.m. ET
Abstract Number: 2677

Abstract:

Introduction: Radiation is accepted as an important standard therapy for locally unresectable cancers, and as such is given to approximately 60% of cancer patients. However, radio-resistance and repair of sublethal radiation damage can limit its efficacy.

Recent studies have shown that Heat Shock Protein 90 (Hsp90), a molecular chaperone that mediates maturation and activation of client proteins, plays a critical role in establishing resistance to radiation therapy. Inhibiting Hsp90 has been reported to sensitize tumors to radiation, resulting in tumor growth suppression and augmenting therapeutic cell death induction. Unfortunately, many of the Hsp90 inhibitors currently in clinical trials exhibit hepatotoxicity as well as ocular toxicity, hindering their clinical use. Taken together, development of clinically acceptable Hsp90 inhibitors for combination with radiation could serve as an important strategy for improving radiotherapy success.

Ganetespib is a second generation Hsp90 inhibitor that has shown potent preclinical activity and is currently in twelve Phase II trials across a broad range of indications. Ganetespib has demonstrated encouraging activity in a Phase II trial in patients with stage IIIB and IV non-small cell lung cancer. Importantly, ganetespib has displayed a favorable safety profile with substantially lower incidence of hepatic or ocular toxicity than that reported for other Hsp90 inhibitors.

Results: We evaluated the radiosensitizing potential of ganetespib *in vivo*. Monotherapy treatment with either ganetespib or 2 Gray (Gy) ionizing irradiation resulted in moderate reductions in human tumor growth rates in a mouse xenograft model. Combination of ganetespib with 2 Gy irradiation resulted in substantial tumor regression. Increasing the dose of radiation in the combination arm to 4 Gy further enhanced tumor regression, resulting in a 50% reduction in tumor volume. In summary, ganetespib offers an effective strategy for improving the outcome of radiotherapy in human cancers.

Title: Phase I evaluation of STA-1474, a pro-drug of the novel HSP90 inhibitor STA-9090, in dogs with spontaneous cancer

Poster Presentation April 4, 8:00 a.m. ET
Abstract Number: 1282

Abstract:

Purpose: The novel water soluble compound STA-1474 is metabolized to ganetespib (formerly STA-9090), a potent HSP90 inhibitor previously shown to kill canine tumor cell lines *in vitro* and inhibit tumor growth in the setting of murine xenografts. The purpose of the following study was

to extend these observations and investigate the safety and efficacy of STA-1474 in dogs with spontaneous tumors.

Experimental Design: This was a Phase 1 trial in which dogs with spontaneous tumors received STA-1474 under one of three different dosing schemes. Pharmacokinetics, toxicities, biomarker changes, and tumor responses were assessed.

Results: Twenty-five dogs with a variety of cancers were enrolled. Toxicities were primarily gastrointestinal in nature consisting of diarrhea, vomiting, inappetence and lethargy. Upregulation of HSP70 protein expression was noted in both tumor specimens and PBMCs within 7 hours following drug administration. Measurable objective responses were observed in dogs with malignant mast cell disease (n=3), osteosarcoma (n=1), melanoma (n=1) and thyroid carcinoma (n=1), for a response rate of 24% (6/25). Stable disease (>10 weeks) was seen in 3 dogs, for a resultant overall biological activity of 36% (9/25).

Conclusions: This study provides evidence that STA-1474 exhibits biologic activity in a relevant large animal model of cancer.

About Ganetespib

Ganetespib (formerly STA-9090) is a potent, synthetic, small-molecule inhibitor of heat shock protein 90 (Hsp90). Hsp90 is a molecular chaperone required for the proper folding and activation of many cancer-promoting proteins, and is recognized as a key facilitator of cancer cell growth and survival. In preclinical experiments, ganetespib has shown activity in multiple tumor models both as a single agent and in combination with certain widely used cancer agents. Ganetespib is currently being evaluated in a broad range of cancer clinical trials. In these trials, ganetespib has shown clinical activity in heavily pretreated patients and has been well tolerated to date with no evidence of severe liver or common ocular toxicities seen with other Hsp90 inhibitors. The most common adverse event seen to date has been diarrhea, which has been manageable with standard supportive care. Information on clinical trials with ganetespib can be found at www.clinicaltrials.gov.

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 invented by Synta scientists using our compound library and discovery capabilities. For more information, please visit www.syntapharma.com.

Safe Harbor Statement

This media release may contain forward-looking statements about Synta Pharmaceuticals Corp. Such forward-looking statements can be identified by the use of forward-looking terminology such as “will”, “would”, “should”, “expects”, “anticipates”, “intends”, “plans”, “believes”, “may”, “estimates”, “predicts”, “projects”, or similar expressions intended to identify forward-looking statements. Such statements, including statements relating to the timing, developments and progress of our clinical and preclinical programs, reflect our current views with respect to future events and are based on assumptions and subject to risks and uncertainties that could cause

actual results to differ materially 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, 2010 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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Synta Announces Results Presented at AACR Further Elucidate Mechanism by Which Elesclomol Targets Cancer Cell Metabolism

-Elesclomol induces cancer cell apoptosis by elevating oxidative stress directly within cancer cell mitochondria, disrupting mitochondrial energy metabolism-

-Biomarkers related to cellular metabolic state, LDHA and TRX-1, are predictive of elesclomol activity in models of lung cancer-

LEXINGTON, MA — April 4, 2011 — Synta Pharmaceuticals Corp. (NASDAQ: SNTA) today presented five posters at the American Association for Cancer Research (AACR) 102nd Annual Meeting. Three posters reported results from studies of ganetespib (STA-9090), a novel Hsp90 inhibitor, and two posters reported results from studies of elesclomol.

Elesclomol is a novel small molecule inhibitor of cancer cell metabolism that kills cancer cells with normoxic, or normal oxygen, metabolism. In three randomized clinical trials elesclomol has shown clinical activity in patients whose cancers show signatures of the normoxic metabolic state. Elesclomol is currently being evaluated in clinical trials in leukemia and ovarian cancer, with a trial in lung cancer expected to initiate later this year.

"The results presented today demonstrate that elesclomol induces apoptosis through a unique means of selectively targeting cancer cell mitochondria and elevating reactive oxygen species," said Vojo Vukovic, M.D., Ph.D., Senior Vice President and Chief Medical Officer, Synta Pharmaceuticals. "The anti-cancer activity of elesclomol is highly dependent on the redox potential of its copper complex. This observation suggests a unique means of selectively targeting cancer cells: exploiting electrochemical differences between cancer cell and normal cell mitochondria. This represents an entirely novel approach to treating cancer, distinct from chemotherapy or cell signaling inhibition."

"Additional results presented today show that two serum markers directly related to tumor metabolic state are predictive of elesclomol *in vitro* activity in lung cancer," said Dr. Vukovic. "Our collaborators at the University of Miami, the V.A. Medical Research Center in Miami, and the M.D. Anderson Cancer Center have shown that low levels of thioredoxin reductase 1 (TRX-1), an antioxidant, which is associated with resistance to cisplatin — a common first-line treatment of lung cancer — leads to high levels of reactive oxygen species (ROS) in lung cancer cells and enhanced sensitivity to elesclomol. Results also showed that low levels of LDHA were associated with increased sensitivity to elesclomol, consistent with results observed in clinical trials. These results further validate the mechanism of action, and suggest promising potential for using biomarkers to select those patients most likely to benefit from treatment."

"All ongoing and planned trials with elesclomol incorporate the use of biomarkers related to tumor metabolic state," continued Dr. Vukovic. "We are excited to pioneer a novel personalized medicine approach for this first-in-class, promising drug candidate."

Clinical trials of elesclomol in acute myeloid leukemia (AML) and ovarian cancer are currently underway and Synta expects to initiate a Phase 2 trial of elesclomol in non small cell lung

cancer later this year. The ovarian cancer trial is being conducted by the Gynecologic Oncology Group (GOG) and supported by the National Cancer Institute (NCI).

Elesclomol posters and publications are available at www.syntapharma.com or by contacting Synta directly.

Title: Elesclomol-Cu chelate selectively targets mitochondria to induce oxidative stress

Poster Presentation April 4, 1:00 p.m. ET
Abstract Number: 2093

Abstract:

Introduction: Elesclomol is a first-in-class investigational drug that exerts anticancer activity through elevating the level of reactive oxygen species (ROS) and oxidative stress. We recently reported that elesclomol selectively chelates Cu(II) in plasma, which causes a change in conformation that enables its uptake into cells. A cell-free assay system showed that elesclomol-Cu(II) generated ROS via the reduction of Cu(II) to Cu(I). A correlation has been observed between the redox potential and anticancer activity for Cu chelates of elesclomol and its analogs, suggesting that the ability to promote redox cycling of Cu(II) to Cu(I) is necessary for anticancer activity. Here, we demonstrate that elesclomol-Cu carries copper into mitochondria, leading to an increase in oxidative stress and apoptosis due to mitochondrial stress.

Results: The subcellular distribution of elesclomol was tracked using preformed elesclomol-Cu chelates. Cytosolic, nuclear and mitochondrial fractions of HL60 cells were prepared from cells treated with elesclomol-Cu, and total copper levels were determined for each cellular fraction. Elevated copper levels were observed only in the mitochondrial fraction, suggesting that elesclomol-Cu selectively transported copper into the mitochondria. To verify that the increased mitochondrial copper levels were from elesclomol-Cu, a copper complex of elesclomol was preformed with 65Cu. This elesclomol-65Cu complex was incubated for 2h with HL60 cells that were previously enriched with 63Cu using 63Cu-supplemented media, and subcellular distributions of 63Cu and 65Cu determined by ICP-MS. Control mitochondria contained minimal levels of endogenous 65Cu. In contrast, 65Cu was markedly increased in the mitochondrial fraction of elesclomol-65Cu treated cells but not in the cytosolic or nuclear fractions, confirming the selective mitochondrial uptake of copper with elesclomol-Cu. Next, we compared the mitochondrial uptake of elesclomol-Cu and disulfiram(DSF)-Cu using isolated mitochondria. DSF is a Cu chelator, and Cu has been shown to enhance DSF-mediated growth inhibition and apoptosis in cancer cells through the generation of ROS. As expected, an increase in copper levels was observed in mitochondria treated with elesclomol-Cu, yet no increase in mitochondrial copper was seen following treatment with DSF-Cu at its cytotoxic concentration, emphasizing the novel mitochondrial selectivity of elesclomol-Cu. To investigate whether the mitochondrial entry of elesclomol-Cu triggered the generation of ROS, ROS in isolated mitochondria was measured. Mitochondrial ROS was immediately increased by adding elesclomol-Cu while no change in mitochondrial ROS was seen using DSF-Cu or free Cu2+. These results show that elesclomol induces apoptosis through elevating ROS directly in cancer cell mitochondria.

Title: Downregulation of thioredoxin-1 confers resistance to cisplatin and sensitivity to the ROS generating agent elesclomol

Poster Presentation April 4, 8:00 a.m. ET

Abstract Number: 1692

Abstract:

We have previously discovered a unique and important finding that all of our cisplatin resistant (CR) lung cancer cell lines, regardless of their signaling mechanisms, possess high levels of ROS (Reactive Oxygen Species) when compared to their parental cancer cell counterparts as well as normal cells. Importantly, these CR cells are sensitive to elesclomol, a new compound which kills cancer cells by generating ROS(2). The question remains why these CR cells possess intrinsically higher levels of ROS.

It is known that one of the pharmacologic actions of cisplatin is the disruption of redox system through inhibition of thioredoxin reductase-1 (TrxR1) TrxR catalyses the NADPH-dependent reduction of the redox protein thioredoxin-1 (TRX1). TRX-1 is an important protein that acts as an antioxidant by facilitating the reduction of other enzymes. Using our CR cell models, we have found that TrxR1 activities as well as TRX-1 levels are significantly decreased. To further verify that TRX-1 is an important contributory factor to the higher ROS levels seen in CR cells, we knocked down TRX-1 protein expression in parental cells using siRNA. These TRX-1 knocked down cells generated significantly higher levels of ROS and were resistant to cisplatin as well as hypersensitive to elesclomol treatment. Correspondingly, over-expression of TRX-1 protein in the CR cells using the pCMV6 vector containing full length TRX-1 cDNA, resulted in decreased ROS production but increased sensitivity to cisplatin. These TRX-1 overexpressing cells also became more resistant to elesclomol treatment.

Moreover, we found that all CR cells have 3-5 fold lower levels of lactate dehydrogenase A (LDHA) levels. Interestingly, it has been reported that diminished elesclomol activity is influenced by high LDHA levels. Here, we found that TRX-1 overexpression cells also exhibit higher LDHA levels and confer resistance to elesclomol. Our findings suggest another novel approach to selectively kill CR lung tumors which intrinsically produce higher ROS and express lower TRX-1 and LDHA levels.

About Elesclomol

Elesclomol is a first-in-class, investigational drug candidate that triggers programmed cell death (apoptosis) in cancer cells through a novel mechanism: disrupting mitochondrial energy metabolism.

Elesclomol binds copper in plasma, which causes a change in conformation that enables its uptake through membranes and into cells. Elesclomol binds copper in an oxidative, positively charged, state called Cu(II). Once inside mitochondria, an interaction with the electron transport chain reduces the copper from Cu(II) to Cu(I), resulting in a cascade of redox reactions, a rapid increase of oxidative stress, disruption of mitochondrial energy production, and the initiation of the mitochondrial apoptosis pathway.

Mitochondria generate energy for cells, but also can induce apoptosis under certain conditions, such as a high level of oxidative stress. By sensitizing mitochondria and reducing barriers to apoptosis, elesclomol may provide a means to overcome resistance to traditional chemotherapy or targeted therapy.

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 invented by Synta scientists using our compound library and discovery capabilities. For more information, please visit www.syntapharma.com.

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