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): **July 25, 2012**

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 1.01 Entry into a Material Definitive Agreement.

On July 25, 2012, Synta Pharmaceuticals Corp. (the "Company") entered into Subscription Agreements with the members of its Board of Directors identified below with respect to the registered direct offer and sale by the Company of an aggregate of 3,976,702 shares of the Company's common stock, par value $0.0001 per share (the "Shares"), at a purchase price of $6.49 per Share. The purchase price is equal to the consolidated closing bid price on The NASDAQ Global Market on July 25, 2012. The Shares were sold directly to the directors without a placement agent, underwriter, broker or dealer. The sale of the Shares is expected to settle on or about July 27, 2012. The net proceeds to the Company are expected to be approximately $25.8 million after deducting estimated offering expenses payable by the Company.

The sale and issuance of the Shares is being made pursuant to a prospectus supplement dated July 25, 2012 and an accompanying prospectus dated August 19, 2011, pursuant to the Company's existing effective shelf registration statement on Form S-3 (File No. 333-176022), which was filed with the Securities and Exchange Commission (the "Commission") on August 4, 2011 and declared effective by the Commission on August 19, 2011.

The members of the Company's Board of Directors purchasing Shares and the number of Shares purchased by each member are set forth below:

- Bruce Kovner, one of our directors and our largest stockholder who beneficially owns approximately 26.1% of our outstanding shares of common stock prior to this offering, will purchase 3,081,664 shares of our common stock. Upon completion of this offering, Mr. Kovner will beneficially own approximately 29.4% of our outstanding shares of common stock.

- Wyandanch Partners, L.P., which is controlled by Keith R. Gollust, one of our directors who beneficially owns approximately 3.7% of our outstanding shares of common stock prior to this offering, will purchase 770,416 shares of our common stock. Upon completion of this offering, Mr. Gollust will beneficially own approximately 4.7% of our outstanding shares of common stock.

- Robert N. Wilson, one of our directors who beneficially owns approximately 1.1% of our outstanding shares of common stock prior to this offering, will purchase 100,000 shares of our common stock. Upon completion of this offering, Mr. Wilson will beneficially own approximately 1.2% of our outstanding shares of common stock.

- Donald W. Kufe, one of our directors who beneficially owns less than 1% of our outstanding shares of common stock prior to this offering, will purchase 4,622 shares of our common stock. Upon completion of this offering, Dr. Kufe will continue to beneficially own less than 1% of our outstanding shares of common stock.

- Safi R. Bahcall, Ph.D., our President and Chief Executive Office and one of our directors who beneficially owns approximately 5.0% of our outstanding shares of common stock prior to this offering, will purchase 20,000 shares of our common stock. Upon completion of this offering, Dr. Bahcall will beneficially own approximately 4.7% of our outstanding shares of common stock.

A copy of the opinion of Mintz, Levin, Cohn, Ferris, Glovsky and Popeo, P.C. relating to the legality of the issuance and sale of the Shares is attached as Exhibit 5.1 hereto. A copy of the form of Subscription Agreement is filed herewith as Exhibit 10.1 and is incorporated herein by reference. The foregoing description of the sale and issuance of the Shares by the Company and the documentation related thereto does not purport to be complete and is qualified in its entirety by reference to such Exhibits.

ITEM 8.01 Other Events.

In connection with the sale and issuance of the Shares described in Item 1.01 of this Current Report on Form 8-K, the Company included an updated business overview in the prospectus supplement dated July 25, 2012.

A copy of the updated business overview is filed as Exhibit 99.1 to this Current Report on Form 8-K and is incorporated by reference herein.

On July 26, 2012, the Company issued a press release announcing the sale and issuance of the Shares. A copy of the press release is filed as Exhibit 99.2 to this Current Report on Form 8-K and is incorporated by reference herein.

ITEM 9.01 Financial Statements and Exhibits.

(d) Exhibits.

Exhibit No.	Description
5.1	Opinion of Mintz, Levin, Cohn, Ferris, Glovsky and Popeo, P.C.
10.1	Form of Subscription Agreement, dated July 25, 2012, by and between Synta Pharmaceuticals Corp. and each of the Purchasers.
23.1	Consent of Mintz, Levin, Cohn, Ferris, Glovsky and Popeo, P.C. (included in the opinion filed as Exhibit 5.1).
99.1	Business Overview included in Prospectus Supplement dated July 25, 2012.
99.2	Press Release, dated July 26, 2012.

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: July 26, 2012

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

EXHIBIT INDEX

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99.1	Business Overview included in Prospectus Supplement dated July 25, 2012.
99.2	Press Release, dated July 26, 2012.

Exhibit 5.1

MINTZ LEVIN

One Financial Center
Boston, MA 02111
617-542-6000
617-542-2241 fax
www.mintz.com

July 25, 2012

Synta Pharmaceuticals Corp.
45 Hartwell Avenue
Lexington, MA 02421

Ladies and Gentlemen:

We have acted as legal counsel to Synta Pharmaceuticals Corp., a Delaware corporation (the "Company"), in connection with the preparation and filing with the Securities and Exchange Commission (the "Commission") of a Prospectus Supplement, dated July 25, 2012 (the "Prospectus Supplement"), to a Registration Statement (No. 333-176022) on Form S-3 (the "Registration Statement"), filed by the Company with the Commission under the Securities Act of 1933, as amended (the "Securities Act"). The Prospectus Supplement relates to the sale of an aggregate of 3,976,702 shares (the "Shares") of the Company's common stock, $0.0001 par value per share (the "Common Stock"), pursuant to Subscription Agreements, dated July 25, 2012, by and among the Company and the purchasers set forth on the signature pages thereto (the "Subscription Agreements"). The form of Subscription Agreement will be filed as an exhibit to a Current Report on Form 8-K and incorporated by reference into the Registration Statement. This opinion is being rendered in connection with the filing of the Prospectus Supplement with the Commission. All capitalized terms used herein and not otherwise defined shall have the respective meanings given to them in the Registration Statement.

In connection with this opinion, we have examined the Company's Restated Certificate of Incorporation and Restated Bylaws, each as currently in effect, the Registration Statement and the exhibits thereto, the Prospectus Supplement and the Subscription Agreements and such other records of the corporate proceedings of the Company and certificates of the Company's officers as we have deemed relevant.

In our examination, we have assumed the genuineness of all signatures, the legal capacity of natural persons, the authenticity of all documents submitted to us as originals, the conformity to original documents of all documents submitted to us as certified or photostatic copies and the authenticity of the originals of such copies.

Our opinion is limited to the General Corporation Law of the State of Delaware and we express no opinion with respect to the laws of any other jurisdiction. No opinion is expressed herein with respect to the qualification of the Shares under the securities or blue sky laws of any state or any foreign jurisdiction.

Please note that we are opining only as to the matters expressly set forth herein, and no opinion should be inferred as to any other matters. This opinion is based upon currently existing statutes, rules, regulations and judicial decisions, and we disclaim any obligation to advise you of any change in any of these sources of law or subsequent legal or factual developments which might affect any matters or opinions set forth herein.

Based upon the foregoing, we are of the opinion that the Shares, when issued and sold in accordance with the Subscription Agreements and the Prospectus Supplement, will be validly issued, fully paid and non-assessable.

We understand that you wish to file this opinion with the Commission as an exhibit to a Current Report on Form 8-K and the Registration Statement in accordance with the requirements of Item 601(b)(5) of Regulation S-K promulgated under the Securities Act and to reference the firm's name under the caption "Legal Matters" in the Prospectus Supplement, and we hereby consent thereto. In giving this consent, we do not admit that we are within the category of persons whose consent is required under Section 7 of the Securities Act or the rules and regulations of the Commission promulgated thereunder.

Very truly yours,

/s/ Mintz, Levin, Cohn, Ferris, Glovsky and Popeo, P.C.

Mintz, Levin, Cohn, Ferris, Glovsky and Popeo, P.C.

Exhibit 10.1

SUBSCRIPTION AGREEMENT

This SUBSCRIPTION AGREEMENT (this "Agreement") is dated as of July 25, 2012, between Synta Pharmaceuticals Corp., a Delaware corporation (the "Company"), and [ ] (the "Purchaser").

WHEREAS, the Purchaser desires to subscribe for, and the Company desires to issue shares of the Company's common stock, $0.0001 par value per share (the "Common Stock"), to the Purchaser pursuant to the terms and conditions of this Agreement;

NOW, THEREFORE, upon the execution and delivery of this Agreement, the Company and the Purchaser agree as follows:

1. Subscription. The Purchaser, intending to be legally bound, hereby irrevocably subscribes for and agrees to purchase [ ] shares of Common Stock (the "Shares") at a per share purchase price of $6.49, which amount is equal to the consolidated closing bid price of the Common Stock as reported by The NASDAQ Stock Market LLC ("NASDAQ") on July 25, 2012, for an aggregate purchase of $[ ] (the "Purchase Price"), and the Company, intending to be legally bound, hereby agrees to issue and sell the Shares to the Purchaser.

2. Registration of Shares. The offering and sale of the Shares (the "Offering") are being made pursuant to (a) an effective Registration Statement on Form S-3 (File No. 333-152833) (the "Registration Statement") filed by the Company with the Securities and Exchange Commission (the "Commission") under the Securities Act of 1933, as amended (the "Securities Act"), including the prospectus contained therein (the "Base Prospectus"), which relates, among other things, to the Shares and the sale thereof from time to time in accordance with Rule 415 under the Securities Act, and (b) a prospectus supplement (the "Prospectus Supplement" and, together with the Base Prospectus, the "Prospectus") containing certain supplemental information regarding the Shares and terms of the Offering that will be filed with the Commission and delivered to the Purchaser (or made available to the Purchaser by the filing by the Company of an electronic version thereof with the Commission) no later than the second business day following the date of this Agreement.

3. Purchase and Sale of Shares. The Company agrees to issue and sell to the Purchaser and the Purchaser agrees to purchase the Shares at a closing to take place at the offices of the Company, or such other place as the Purchaser and the Company shall mutually agree, including by way of the exchange of facsimile or "pdf" copies of signatures with originally executed copies of the Agreement to follow by overnight courier (the "Closing"), no later than July 27, 2012 or such other date as the Purchaser and the Company shall mutually agree (the "Closing Date"). At the Closing, the Company shall deliver instructions to the Company's transfer agent to issue the Shares as of the Closing Date and deliver to the Purchaser a certificate evidencing the Shares against delivery of the Purchase Price, which shall be paid by the Purchaser at the Closing by wire transfer of immediately available funds to the account set forth on Schedule I hereto.

4. Legends. Any certificates evidencing the Shares shall bear a legend in substantially the following form:

THE SECURITIES REPRESENTED BY THIS CERTIFICATE ARE OWNED BY A PERSON OR PERSONS WHO MAY BE CONSIDERED AN AFFILIATE FOR PURPOSES OF RULE 144 UNDER THE SECURITIES ACT OF 1933 (THE "ACT"). NO TRANSFER OF THESE SECURITIES OR ANY INTEREST THEREIN MAY BE MADE UNLESS THE ISSUER HAS RECEIVED AN OPINION OF COUNSEL SATISFACTORY TO IT THAT SHARES MAY BE SOLD PURSUANT TO RULE 144 OR ANOTHER AVAILABLE EXEMPTION UNDER THE ACT AND THE RULES AND REGULATIONS THEREUNDER.

5. Closing Conditions.

(a) The obligations of the Company hereunder are subject to the following conditions being met:

(i) the accuracy in all material respects as of the date hereof of the representations and warranties by the Purchaser contained herein; and

(ii) the delivery by the Purchaser of the Purchase Price to the Company for the Shares as set forth herein on the Closing Date.

(b) The obligations of the Purchaser hereunder are subject to the following conditions being met:

(i) the accuracy in all material respects as of the date hereof of the representations and warranties by the Company contained herein; and

(ii) the delivery by the Company, or through the Company's transfer agent, on the Closing Date of evidence satisfactory to the Purchaser confirming that the Shares have been issued in the name of the Purchaser as of the Closing Date.

6. Representations and Warranties of the Company. As of the date hereof, the Company hereby represents and warrants to the Purchaser that:

(a) Organization. The Company is a corporation, duly organized, validly existing and in good standing under the laws of the State of Delaware.

(b) Authority and Validity. The Company has all requisite corporate power and authority to execute, deliver and perform its obligations under this Agreement and to consummate the transactions contemplated hereby. The execution, delivery and performance by the Company of this Agreement and the consummation of the transactions contemplated hereby have been duly and validly authorized by all necessary action required on the part of the Company, and no other proceedings on the part of the Company are necessary to authorize this Agreement or for the Company to perform its obligations under this Agreement. This Agreement constitutes the lawful, valid and legally binding obligation of the Company,

enforceable in accordance with its terms, except as the same may be limited by applicable bankruptcy, insolvency, reorganization, moratorium or similar laws affecting the enforcement of creditors' rights generally and general equitable principles regardless of whether such enforceability is considered in a proceeding at law or in equity.

(c) Valid Issuance of Common Stock. The Shares, when issued, sold and delivered in accordance with the terms hereof for the Purchase Price, will be duly and validly authorized and issued, fully paid and nonassessable and free of restrictions on transfer other than restrictions on transfer under this Agreement and applicable state and federal securities laws.

(d) Registration Statement. The Registration Statement is in full force and effect and no cease and desist order or other suspension of the Registration Statement exists, has been imposed or, to the knowledge of the Company is threatened by the Commission.

(e) No Violation or Conflict. The execution, delivery and performance of this Agreement and the transactions contemplated hereby do not (i) violate, conflict with or result in the breach of any provision of the Company's Restated Certificate of Incorporation or Restated Bylaws, (ii) conflict with or violate any law, rule, regulation, order, judgment or decree applicable the Company or any of its assets, properties or businesses, or (iii) conflict with, result in any breach of, constitute a default (or event that with the giving of notice or lapse of time, or both, would become a default) under, require any consent under, or give to others any rights of termination, amendment, acceleration, suspension, revocation or cancellation of, or result in the creation of any encumbrance on any of the assets or properties of the Company, pursuant to any note, bond, mortgage or indenture, contract, agreement, lease, sublease, license, permit, franchise or other instrument or arrangement to which the Company is a party except, in the case of clauses (ii) and (iii), to the extent that such conflicts, breaches, defaults or other matters would not, individually or in the aggregate, reasonably be expected to have a material adverse effect on the Company.

(f) Governmental/Regulatory Consents and Approvals. Except for filings under federal securities laws and, if required, The NASDAQ Marketplace Rules, the execution, delivery and performance of this Agreement by the Company do not, and the consummation of the transactions contemplated hereby do not and will not, require any permits, consents, approvals, orders, authorizations of, or declarations to or filings with any federal, state, local or foreign government or regulatory authority, which has not already been obtained, effected or provided.

7. Representations and Warranties of the Purchaser. As of the date hereof, the Purchaser hereby represents and warrants to the Company that:

(a) The Purchaser has received (or otherwise had made available to him by the filing by the Company of an electronic version thereof with the Commission) the Base Prospectus which is a part of the Registration Statement, and the documents incorporated by reference therein (collectively, the "Disclosure Package"), prior to or in connection with the execution of this Agreement.

(b) The Purchaser (a) is knowledgeable, sophisticated and experienced in making, and is qualified to make decisions with respect to, investments in shares presenting an investment decision like that involved in the purchase of the Shares, including investments in securities issued by the Company and investments in comparable companies and has reviewed such information and made such inquiries regarding the Company and the purchase of the Shares as he has deemed appropriate and (b) in connection with his decision to purchase the Shares, has received (or had full access to) and is relying only upon the Disclosure Package and the documents incorporated by reference therein.

(c) The Purchaser understands that nothing in this Agreement, the Disclosure Package or any other materials presented to the Purchaser in connection with the purchase and sale of the Shares constitutes legal, tax or investment advice. The Purchaser has consulted such legal, tax and investment advisors and made such investigations as he, it his sole discretion, has deemed necessary or appropriate in connection with his purchase of the Shares.

(d) No person or entity acting on behalf of, or under the authority of, the Purchaser is or will be entitled to any broker's, finder's, or similar fees or commission payable by the Company.

8. Governing Law. This Agreement shall be governed by, and construed in accordance with, the laws of the State of Delaware, without regard to its conflicts of laws principles.

9. Entire Agreement. This Agreement constitutes the entire agreement between the Company and the Purchaser with respect to the matters covered hereby and supersedes all prior agreements and understanding with respect to such matters between the Company and the Purchaser.

10. Severability. In case any provision contained in this Agreement should be invalid, illegal or unenforceable in any respect, the validity, legality and enforceability of the remaining provisions contained herein will not in any way be affected or impaired thereby.

11. Counterparts; Facsimile or "pdf" Copies. This Agreement may be executed in counterparts, each of which, when executed, shall be deemed an original but all of which, taken together, shall constitute one and the same Agreement. Delivery of an executed copy of a signature page to this Agreement by facsimile or "pdf" transmission shall be as effective as delivery of a manually executed copy of this Agreement and shall be as effective and enforceable as the original.

[SIGNATURES FOLLOW ON NEXT PAGE]

IN WITNESS WHEREOF, each of the undersigned has executed and delivered this Agreement on the day and year first above written.

SYNTA PHARMACEUTICALS CORP.

By: _____
Name:
Title:

[ ]

Overview

We are 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. We have two drug candidates in clinical trials for treating multiple types of cancer and several drug candidates in the preclinical stage of development. Each of our drug candidates was discovered and developed internally using our proprietary, unique chemical compound library and integrated discovery engine. We retain full ownership of all of our drug candidates.

Oncology Programs

We have two clinical-stage programs and one preclinical-stage program in oncology:

Ganetespib (Hsp90 Inhibitor)

Summary

Ganetespib is a novel, potent, small molecule inhibitor of heat shock protein 90 (Hsp90), a molecular chaperone which is required for the proper folding and activation of many cancer-promoting proteins. Inhibition of Hsp90 by ganetespib leads to the simultaneous degradation of many of its client proteins and the subsequent death or cell cycle arrest of cancer cells dependent on those proteins. A number of Hsp90 client proteins are also involved in the resistance of cancer cells to other anti-cancer treatments, such as chemotherapy. The ability to reduce cancer-cell drug resistance suggests the combination of ganetespib with chemotherapies or other agents may provide greater benefit than those agents administered alone. In preclinical studies, ganetespib has shown potent anti-cancer activity in a broad range of solid and hematologic cancers, both as a monotherapy and in combination with certain widely used anti-cancer agents.

Ganetespib is currently being evaluated in over 20 clinical trials, including trials evaluating monotherapy administration in certain genetically-defined targeted patient populations, such as our trials in ALK+ lung cancer, HER2+ breast cancer, and triple-negative breast cancer, as well as trials evaluating combination treatment in a broader patient population, such as our GALAXY lung cancer trial. The safety profile across these trials, involving over 600 patients treated with ganetespib to date, has been consistent and favorable. Ganetespib has shown no evidence of the serious liver or common ocular toxicities reported with other Hsp90 inhibitors, or the neurotoxicity, bone marrow toxicities, and alopecia characteristic of many chemotherapies. The most common adverse event reported with ganetespib has been transient, mild or moderate diarrhea, which can be prevented or effectively managed with standard supportive care.

In clinical trials, ganetespib has shown promising activity in a broad range of cancers, both as a monotherapy and in combination:

- *Monotherapy:*
  - Objective responses or anti-tumor activity have been seen in patients with ALK+ lung cancer, mutant BRAF lung cancer, mutant KRAS lung cancer, mutant KRAS gastric cancer, HER2+ breast cancer, triple-negative breast cancer, renal cancer, colorectal cancer, and melanoma.
- *Combination*: We recently announced encouraging interim results from our randomized, Phase 2b/3 GALAXY trial evaluating ganetespib plus docetaxel vs. docetaxel alone in second-line non-small cell lung cancer (NSCLC). Key findings from this interim analysis include:
  - Increases in progression-free survival (PFS), objective response rate, and disease control rate in patients treated with ganetespib. These increases were observed in patients with elevated lactate dehydrogenase (LDH) and patients with mutant KRAS, which are the two pre-specified co-primary endpoint populations, as well as in all adenocarcinoma patients
  - An encouraging improvement in overall survival (OS) in the all adenocarcinoma population
  - A profile suggestive of anti-angiogenic activity, consistent with preclinical results showing that ganetespib potently inhibits the hypoxia-induced factor (HIF) pathway that drives VEGF production in tumors. This includes: (a) enhanced activity in patients with elevated markers of tumor hypoxia (LDH), (b) favorable results in patients with adenocarcinoma histology, and (c) lack of benefit in patients with squamous cell histology, with possible safety concerns, including risk of bleeding, e.g. hemoptysis. This profile is consistent with known anti-angiogenic agents, e.g. direct VEGF inhibitors.
  - Favorable safety of the ganetespib plus docetaxel combination in adenocarcinoma patients

The results observed with ganetespib monotherapy administration suggest promising potential for treating specific, targeted populations: patients with cancers driven by increased expression or mutations in genes encoding “strong” Hsp90 clients. This

represents a sizable unmet need and commercial opportunity. Our CHIARA trial, for example, evaluates ganetespib in patients with ALK+ lung cancer. There are an estimated 40,000-70,000 new patients diagnosed worldwide each year with this cancer type. Our ENCHANT trial evaluates ganetespib in patients with HER2+ or triple-negative breast cancer. Each of these subpopulations is estimated at 15-20% of the 1.4 million patients diagnosed with breast cancer worldwide each year.

The results observed to date in our GALAXY trial suggest an even broader unmet need and commercial opportunity for the combination therapy approach. An estimated 600,000 patients worldwide die each year from NSCLC with adenocarcinoma histology, the patient population being evaluated in our GALAXY trial. In addition, over 500,000 patients receive taxanes each year, across all cancer indications. The ability to combine with taxanes with minimal additional toxicity and possible enhanced activity represents a promising opportunity not only in lung cancer but in breast, prostate, ovarian, gastric, bladder, and head and neck cancers as well. In preclinical models, ganetespib has shown ability to enhance the activity of a number of other widely used anti-cancer agents, in addition to the taxanes, including pemetrexed, gemcitabine, bevacizumab, cytarabine, irinotecan, etoposide, doxorubicin, carboplatin, cisplatin, vincristine, tamoxifen, fulvestrant, temsirolimus, lapatinib, crizotinib, vemurafenib, selumetinib, and bortezomib. Combination trials with a number of these agents have recently been initiated or are planned for later this year.

Ganetespib Mechanism of Action and Preclinical Results

Ganetespib is a novel, small-molecule inhibitor of Hsp90 structurally unrelated to first-generation, ansamycin-family compounds, such as 17-AAG or 17-DMAG. In preclinical studies, ganetespib has shown 10-100 times greater potency than 17-AAG across a broad range of cancer cell types as well as activity in animal models that are resistant to treatment with 17-AAG.

Hsp90 is a molecular chaperone required for the proper folding and activation of many cancer-promoting proteins. Many of the client proteins of Hsp90, such as ALK, AKT, BCR-ABL, BRAF, KIT, MET, EGFR, FLT3, HER2, HIF-1alpha, PDGFRA, and VEGFR, are the targets of clinically validated cancer drugs such as Avastin, Erbitux, Gleevec, Herceptin, Nexavar, Sutent, Tarceva, Votrient, Xalkori, and Zelboraf. In preclinical studies, inhibition of Hsp90 by ganetespib results in simultaneous degradation of these client proteins, resulting in cancer cell death or cell cycle arrest.

Ganetespib also inhibits known mechanisms by which cancer cells evade or recover from other anti-cancer treatments. For example, cancer cells can evade DNA damage caused by chemotherapy or radiation therapy through modification of cell cycle dynamics and activation of DNA repair processes. Many of the cell cycle and DNA repair components — such as ATM, ATR, CHK1, BRCA1, and WEE1 — are Hsp90 client proteins. Ganetespib has shown activity both as a monotherapy and in combination in a broad range of *in vitro* and *in vivo* models of cancer. Combination activity has been observed in models of ALK+ NSCLC with Xalkori, KRAS mutant NSCLC with docetaxel, EGFR mutant NSCLC with Avastin, HER2+ breast cancer with Tykerb, colorectal cancer with radiation or platinum therapy, BRAF mutant melanoma with Zelboraf, hormone refractory prostate cancer with mTOR inhibitors, and AML with cytarabine.

Results published in *Molecular Cancer Therapeutics* in December 2011 highlighted certain physicochemical properties of ganetespib believed to contribute to its improved safety and activity relative to other Hsp90 inhibitors. These include smaller size, greater potency, improved ability to passively enter cells, improved interaction with the drug target, absence of a molecular component known to cause liver toxicity, and ability to penetrate deep into tumor tissues.

Results presented at the AACR-EORTC-NCI meeting in November 2011 and at the American Society of Clinical Oncology (ASCO) meeting in June 2012 demonstrated that common ocular toxicities seen with some Hsp90 inhibitors, but not observed in clinical trials with ganetespib or with 17-AAG, are associated with physicochemical properties that affect drug distribution to the eye.

Ganetespib Clinical Trials

Ganetespib is being evaluated in over 20 clinical trials ongoing, currently initiating, or recently completed, including trials in lung, breast, colon, gastric, prostate, melanoma, and pancreatic cancers, as well as in certain types of blood cancers. Many of these trials are sponsored by individual investigators or groups of investigators. We are sponsoring three principal trials evaluating ganetespib activity:

- GALAXY: a randomized Phase 2b/3 trial evaluating ganetespib in combination with docetaxel versus docetaxel alone as second-line therapy in patients with advanced NSCLC (Ganetespib Assessment in Lung cAncer with docetaXel);

- CHIARA: a Phase 2 trial evaluating ganetespib monotherapy in patients whose tumors have a genetic profile characterized by rearrangement of the ALK gene (ALK+) (Chaperone Inhibition in ALK Rearranged lung cAncer), and

- ENCHANT: a Phase 2 trial evaluating ganetespib monotherapy in patients with newly diagnosed HER2+ and triple-negative metastatic breast cancer (EvaluatiNg Chaperone inhibition by gANetespib in breasT cancer)

Ganetespib in combination with chemotherapy: the GALAXY Trial

Cancer treatments are often given in combination in order to maximize benefit to patients. A challenge with combination therapy is that the added toxicities from combining two or more potent anti-cancer agents may not be tolerable, particularly if the toxicity profiles from distinct treatments overlap. The favorable safety profile seen to date with ganetespib and the non-overlapping toxicities with many standard-of-care agents support such a combination therapy approach.

Results to date suggest potential for combining ganetespib and taxanes. These include a strong scientific rationale based on multiple mechanisms of synergistic anti-cancer activity, strong synergestic results in *in vitro* and *in vivo* experiments, and the encouraging safety profile seen in our Phase 1 combination study of ganetespib and docetaxel.

GALAXY Trial Design

In 2011 we initiated the GALAXY trial, a Phase 2b/3 program in patients with advanced NSCLC who have received one prior treatment for advanced disease, i.e., a second-line treatment setting. The GALAXY trial compares treatment with docetaxel alone, which is approved for second-line treatment, versus treatment with ganetespib plus docetaxel. The first stage, Phase 2b portion is designed to establish the clinical benefit and safety profile of ganetespib in combination with docetaxel relative to docetaxel alone, and to identify the patient populations, by biomarker or other disease characteristics, which may be most responsive to combination treatment. The first stage of this program is intended to build the clinical and operational experience needed to optimize the design and execution of the second stage, Phase 3 portion.

Patients in both arms receive a standard regimen of docetaxel 75 mg/m2 on day 1 of a 21-day cycle. Patients in the combination arm also receive ganetespib 150 mg/m2 on days 1 and 15. Treatment continues until disease progression per Response Evaluation Criteria in Solid Tumors (RECIST) criteria. Enrollment is stratified by ECOG performance status, LDH, smoking status, and time since diagnosis of metastatic disease to ensure balance of these prognostic factors between the two arms.

The Phase 2b portion of the trial was designed to enroll 240 second-line NSCLC patients, without selecting for specific biomarkers, in order to evaluate several pre-specified hypotheses on which patients might be most responsive to combination treatment. On initial design the co-primary endpoints were progression-free survival in all patients (the ITT or intent-to-treat population) and overall survival in patients with elevated baseline level of serum LDH. Several months after trial initiation, but before any substantial patient enrollment, the trial was amended to elevate improvement in progression-free survival in patients with mutant KRAS (the mKRAS population) from a secondary endpoint to a co-primary endpoint, based on clinical results observed in a separate ganetespib trial around that time. Both LDH and mutant KRAS were pre-specified for evaluation from blood and tumor tissue, respectively, by an independent, central laboratory.

Enhanced activity in the elevated LDH population was chosen as a primary hypothesis because an isoform of LDH is a marker of lack of oxygen, or hypoxia, in a tumor. Under low oxygen conditions, hypoxia-induced factor (HIF-1alpha) rises in cancer cells and has been shown in preclinical studies to increase tumor metastasis, angiogenesis, and resistance to chemotherapy. Inhibition of HIF-1alpha has been shown in preclinical studies to reduce metastases, improve survival, and enhance the anti-cancer activity of chemotherapy. Because ganetespib potently suppresses HIF-1alpha, which is an Hsp90 client protein, it was proposed that patients with elevated levels of baseline LDH might show enhanced benefit from ganetespib treatment. Recent trials with other agents whose activity depends on tumor oxygen state, including VEGF and mTOR inhibitors, have shown a correlation of activity with serum LDH levels, further supporting this hypothesis. Generally, one quarter to one third of patients in comparable trials present with elevated baseline levels of LDH.

Enhanced activity in the mutant KRAS population was chosen as a primary hypothesis based on both the known Hsp90 dependence of pathways active in this type of lung cancer and observations from other Synta trials. In a Phase 2 trial evaluating ganetespib monotherapy in lung cancer, as reported at the ASCO June 2011 meeting, approximately two thirds of patients with mutant KRAS showed tumor shrinkage following ganetespib treatment. In addition, in an investigator-sponsored Phase 2 trial in patients with esophagogastric cancer, a patient with mutant KRAS gastric cancer experienced a complete response following treatment with ganetespib monotherapy. The patient recently entered the 23rd month of treatment with ganetespib. Approximately 15% to 30% of NSCLC patients are estimated to have tumors with KRAS mutation.

The GALAXY trial was designed to enroll patients with all histologies — including both adenocarcinoma and squamous cell. Earlier this year enrollment of patients with squamous cell histology was terminated based on the lack of benefit observed in patients with this histology; possible safety concerns, including risk of bleeding; and the consistency of the emerging ganetespib profile with

known anti-angiogenic agents, for which patients with squamous cell histology are commonly excluded from clinical trials or labeled indications. The trial was amended at that time to enroll a total of 240 patients with adenocarcinoma histology only.

The current co-primary endpoints of the first-stage, Phase 2b portion are: PFS in patients with elevated LDH and PFS in patients with mutant KRAS. Key secondary endpoints, to be evaluated with the statistical gatekeeping methodology, include OS and PFS in the all adenocarcinoma population. The Phase 2b stage is 90% powered to detect a PFS improvement from 6 to 12 weeks in patients with elevated LDH and from 5 weeks to 10 weeks in patients with mutant KRAS. For all adenocarcinoma patients, GALAXY is 88% powered to detect an improvement in PFS from 3 to 4.5 months, and 73% powered to detect an improvement in OS from 6 to 8.5 months. All powering assumptions are based on a 1-sided alpha of 0.05.

GALAXY Interim Results

In June 2012 we reported top line results from a planned interim analysis of the GALAXY trial. The analysis was planned for when approximately 50% of patients had been enrolled and had sufficient follow up, defined as one post-baseline scan. At the time of this analysis, completed in June, a total of 114 adenocarcinoma and 69 non-adenocarcinoma patients had been enrolled.

The table below lists primary and key secondary endpoints relating to the two co-primary patient populations, as well as the all adenocarcinoma population. At the time of the interim analysis, there were 31 patients identified as elevated LDH, and 20 patients as mutant KRAS. Of 73 adenocarcinoma samples successfully evaluated for KRAS status by the time of this analysis, 53 and 20 were identified as wild-type and mutant KRAS, respectively. Of the elevated LDH and mutant KRAS groups, 9 patients were positive for both markers. Responses were assessed per RECIST 1.1 criteria; there have been no complete responses seen in this trial. Results reported are for adenocarcinoma patients only.

Outcomes of GALAXY subgroups from the June interim analysis

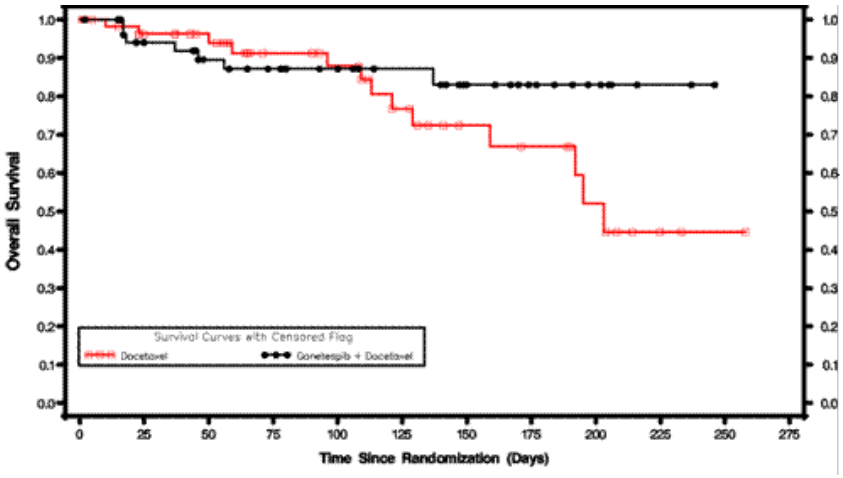
	Elevated LDH (N=31)		**Mutant KRAS (N=20)**		**All adeno (N=114)**	
	D (N=15)	**G+D (N=16)**	**D (N=11)**	**G+D (N=9)**	**D (N=59)**	**G+D (N=55)**
Primary endpoint						
median PFS	1.4 mo	4.2 mo	1.6 mo	4.2 mo	2.9 mo	4.2 mo
PRs (%)	0	2 (13)	1 (9)	2 (22)	5 (8)	8 (15)
Number of events						
# PFS events	12	8	5	3	31	23
# deaths	6	4	2	1	13	7

G: ganetespib, D: docetaxel
PR: partial response

These results are encouraging and consistent with the pre-specified hypotheses of enhanced activity in both the elevated LDH and the mutant KRAS populations.

We also reported interim OS results. Below is a Kaplan-Meier analysis of survival events in the all adenocarcinoma population. The Y-axis represents the fraction of patients alive in each arm of the study.

Overall survival in the GALAXY all adenocarcinoma population at the June interim analysis



Additional details will be provided at an upcoming medical meeting.

The adverse event profile has been comparable between both arms of the GALAXY trial. The proportion of adenocarcinoma patients with at least one adverse event (AE) was 64% vs. 82%; with grade 3 or greater AEs was 32% vs. 36%; with AEs leading to treatment discontinuation was 7% vs. 9%; and with AEs with outcome of death were 5.1% vs. 5.5%, for D (N=59) vs. G+D (N=55), respectively. The most common AEs, all grades were neutropenia (48% vs. 44%), diarrhea (10% vs. 40%) and fatigue (19% vs. 26%), for D vs. G+D, respectively. Diarrhea and fatigue were predominantly grade 1 and grade 2; the incidence of grade 3 or greater diarrhea was 0% vs. 2% and grade 3 or greater fatigue was 3% vs. 0% in D vs. G+D, respectively. The most common grade 3 or greater AEs were neutropenia (29% vs. 33%), leukopenia (5% vs. 4%), and nausea (3% vs. 4%). Trials with some other Hsp90 inhibitors have reported a high incidence of ocular toxicities. In this trial, there has been one report of an ocular-related adverse event (grade 2, transient blurred vision) in the G+D arm (2%) vs. no reports in the D arm.

Summary of GALAXY Trial Findings and Near-Term Plans

Key findings from the recent interim analysis include:

- Increases in PFS, objective response rate, and disease control rate in patients treated with ganetespib across the three major patient populations of interest: elevated LDH, mutant KRAS, and all adenocarcinoma patients. This activity was enhanced in the pre-specified biomarker groups, consistent with the preclinical and clinical rationale
- Encouraging improvement in OS in the all adenocarcinoma population
- A profile suggestive of anti-angiogenic activity, consistent with preclinical results showing that ganetespib potently inhibits the hypoxia-induced factor (HIF) pathway that drives VEGF production in tumors. This includes: (a) enhanced activity in patients with elevated markers of tumor hypoxia (LDH), (b) favorable results in patients with adenocarcinoma histology, and (c) lack of benefit in patients with squamous cell histology, with possible safety concerns, including risk of bleeding, e.g. hemoptysis.
- Favorable safety of the ganetespib plus docetaxel combination in adenocarcinoma patients

Our near term plans for the GALAXY program include:

- Conduct the next planned interim analysis and present these results at an upcoming medical meeting
- Meet with regulatory agencies, review the interim results, and discuss plans for the Phase 3 portion of the trial later this year
- Complete enrollment and transition to the Phase 3 portion of the trial later this year

Based on our current plans and projections, we anticipate final data from the Phase 2b portion of the trial in the first half of 2013, and final data from the Phase 3 portion in the first half of 2014.

Ganetespib as Monotherapy

ALK+ NSCLC: In June and July 2011 we presented results from a Phase 2 trial of ganetespib administered as a monotherapy in patients with advanced NSCLC at the ASCO Annual Meeting and the International Association for the Study of Lung Cancer (IASLC) 14th World Conference on Lung Cancer, respectively. Patients in this trial had failed to respond to, or experienced disease progression following, numerous prior therapies. In this trial, as in other trials, ganetespib treatment was associated with favorable safety.

Encouraging evidence of clinical activity was also observed in this trial, as evident by the durable objective tumor responses achieved in certain patients, as evaluated by RECIST. The disease control rate, using the standard definition of complete response plus partial response plus stable disease, was 54%. This rate compares favorably with disease control rates observed in trials for approved and experimental agents in a similar broad, pre-treated, advanced NSCLC patient population.

Results presented at these meetings also showed a connection between single-agent ganetespib clinical activity and certain tumor genetic profiles. Four of eight patients who were ALK+, i.e., for whom tumor genetic testing revealed rearrangements in the ALK gene, experienced confirmed partial responses following treatment with ganetespib (a 50% objective response rate, using the standard definition of complete response plus partial response). These responses were durable, with the responding patients remaining on therapy an average of about one year (range 7 to >21 months). Six of these eight patients experienced tumor shrinkage in target lesions, and seven of these eight patients (88%) achieved disease control for eight weeks or more. These results are encouraging when compared to results typically seen with chemotherapy and other agents in these advanced NSCLC treatment settings, for which objective response rates have been in the range of 5-10%.

Although only eight critzonib-untreated, ALK+ patients were reported in this trial, these results are comparable to those seen with the direct ALK inhibitor Xalkori® (crizotinib), which was granted accelerated approval in August 2011 by the FDA for the treatment of ALK+ NSCLC. In a Phase 1 trial enrolling 136 ALK+ patients and in a single-arm Phase 2 trial in 119 ALK+ patients , crizotinib demonstrated a 50% and a 61% objective response rate, respectively, by investigator review, and a 42% and 51% objective response rate, respectively, by independent review.

Ganetespib has also been shown to be active as monotherapy and in combination with crizotinib in preclinical models of ALK+ NSCLC. Importantly, ALK inhibition via direct inhibition of Hsp90 supports a complementary, rather than competitive, mechanism with crizotinib and other direct ALK inhibitors. Combined with clinical observations so far, these results present strong evidence that Hsp90 inhibition with ganetespib is a promising approach for treating ALK+ NSCLC patients.

To further characterize ganetespib activity in this treatment setting, we recently initiated the CHIARA trial to evaluate ganetespib monotherapy in ALK+ NSCLC patients who have not been previously treated with a direct ALK inhibitor. We expect preliminary results from this trial by the end of the year. In addition to CHIARA, a number of cancer centers and cooperative groups have approached us with proposals to support trials evaluating ganetespib in combination with other agents in ALK+ disease. An investigator-sponsored Phase 1/2 trial evaluating ganetespib and crizotinib combinations in ALK+ patients began enrolling patients at Memorial Sloan-Kettering Cancer Center (MSKCC) in New York City earlier this year.

HER2+ and triple negative metastatic breast cancer: At the San Antonio Breast Cancer Symposium in December 2011, researchers from MSKCC presented results from a Phase 2 trial evaluating ganetespib monotherapy in patients with metastatic breast canc\er who had been previously treated with multiple lines of chemotherapy or other anti-cancer agents. Results showed that 15% (2/13) of the HER2+ patients experienced a confirmed partial response and an additional 46% (6/13) achieved stable disease. These results with ganetespib in HER2+ disease are consistent with results from an earlier Phase 2 study of 17-AAG, a first-generation Hsp90 inhibitor, in patients who had progressed following treatment with one line of trastuzumab (Herceptin). In that trial 22% (6/27) of patients achieved a partial response and an additional 37% (10/27) achieved stable disease. While in the latter study 17-AAG was given in combination with trastuzumab, in the former study ganetespib was given as a monotherapy. Together, these studies present strong evidence that Hsp90 inhibition is a promising approach for treating HER2+ breast cancer.

Results with ganetespib in patients with triple-negative breast cancer (TNBC) were also reported in December 2011. One of three evaluable patients in the Phase 2 clinical trial experienced significant tumor shrinkage following three doses of ganetespib. An objective response was also reported in a patient with TNBC participating in a ganetespib Phase 1 trial. TNBC represents a difficult-to-treat disease, for which no targeted therapies are currently approved. These results are encouraging, and suggest that ganetespib is active in TNBC.

We recently initiated the ENCHANT trial designed to evaluate ganetespib monotherapy as first-line treatment for both metastatic HER2+ breast cancer and TNBC. In addition, MSKCC has announced that it will initiate a Phase 1/2 trial evaluating

ganetespib in combination with paclitaxel and Herceptin in HER2+ breast cancer, and ganetespib in combination with paclitaxel in TNBC.

Additional oncology indications

In addition to the clinical trials we plan to initiate and continue in 2012, a number of ganetespib trials sponsored by third parties, including cooperative groups, foundations, and individual investigators, have recently initiated or are expected to initiate in 2012. These include

- the trials evaluating ganetespib in breast cancer and in ALK+ lung cancer sponsored by MSKCC described above
- a randomized trial evaluating the combination of fulvestrant and ganetespib in patients with hormone receptor-positive, metastatic breast cancer, being conducted at the Dana-Farber Cancer Institute, which began enrolling patients earlier this year
- a trial evaluating the combination of ganetespib with capecitabine and radiation in patients with locally advanced rectal cancer being conducted at Emory University, which began enrolling patients earlier this year
- a trial evaluating both ganetespib monotherapy and the combination of ganetespib and bortezomib in multiple myeloma, supported by a grant of up to $1 million by the Multiple Myeloma Research Foundation, which began enrolling patients earlier this year
- a trial evaluating ganetespib in combination with pemetrexed and cisplatin in patients with malignant pleural mesothelioma, being sponsored by Cancer Research UK, which is expected to initiate later this year
- a randomized trial evaluating the combination of ganetespib and the chemotherapy drug ara-C in elderly patients with acute myeloid leukemia (AML)

Additional ongoing investigator-sponsored trials include trials in prostate cancer, pancreatic cancer, liver cancer, melanoma, and ocular melanoma.

Elesclomol (Mitochondria-Targeting Agent)

Elesclomol is a first-in-class, investigational drug candidate that triggers programmed cell death (apoptosis) in cancer cells through a novel mechanism: disrupting cancer cell mitochondrial metabolism. In preclinical experiments, anti-cancer activity of elesclomol has been shown to correlate with certain biomarkers, including LDH, which can distinguish between active mitochondria (sufficient oxygen) and inactive mitochondria (insufficient oxygen). Consistent with these findings in three randomized clinical trials, LDH was an important predictor of elesclomol treatment outcome.

Our current clinical program for elesclomol includes a clinical trial of elesclomol as a monotherapy in AML. In December 2009, we presented results at the American Society for Hematology (ASH) meeting showing that elesclomol was highly active against AML cell lines and primary blast cells from AML patients. In February 2011, we announced that the first patient had been treated in a Phase 1 dose escalation study of elesclomol as a single agent in patients with AML. This trial will enroll up to 36 patients with relapsed or refractory AML and total baseline serum LDH level less than 0.8 times ULN. Patients will be treated with elesclomol sodium on a once-weekly schedule at a starting dose of 200 mg/m2, with dose escalation planned based on safety, tolerability and pharmacokinetic considerations. The trial is being conducted at Princess Margaret Hospital in Toronto, Canada and at MSKCC in New York.

We are also evaluating the use of elesclomol in combination with paclitaxel in ovarian cancer. In March 2011, the Gynecological Oncology Group (GOG), initiated a Phase 2 clinical trial of elesclomol in combination with paclitaxel for the treatment of persistent or recurrent ovarian, fallopian tube or primary peritoneal cancer for patients with total baseline serum LDH level less than 0.8 times ULN. The GOG is a non-profit organization with the purpose of promoting excellence in the quality and integrity of clinical and basic scientific research in the field of gynecologic malignancies. The National Cancer Institute is providing financial support of up to approximately $300,000 for the trial through its Cancer Therapy Evaluation Program.

STA-9584 (Vascular Disrupting Agent)

STA-9584 is a novel, injectable, small molecule compound that appears to disrupt the blood vessels that supply tumors with oxygen and essential nutrients, and is in preclinical development. In March 2011, we received a $1 million grant from the United States Department of Defense (DoD) for the development of STA-9584 in advanced prostate cancer.

Inflammatory Disease Programs

We have two preclinical-stage programs focusing on treatments for inflammatory diseases. Both of our inflammatory disease programs focus on oral, disease- modifying drug candidates that act through novel mechanisms and could potentially target multiple indications.

CRACM Ion Channel Inhibitors

We have developed novel, small molecule inhibitors of CRACM ion channels expressed on immune cells. Our CRACM ion channel inhibitors have shown strong anti-inflammatory activity in preclinical studies both *in vitro* and *in vivo*, inhibiting T cell and mast cell activity, including cytokine release, degranulation, and immune cell proliferation. Potential applications include a wide range of inflammatory diseases and disorders for which modulating T cell and mast cell function has been shown to be critical, including rheumatoid arthritis (RA), asthma, chronic obstructive pulmonary disease (COPD), allergy, transplant rejection, and other autoimmune diseases and inflammatory conditions. We have several promising CRACM inhibitors in preclinical development. Because there are a number of CRACM ion channel targets on immune cells, we believe that CRACM inhibitor compounds can be developed that target different diseases.

Roche CRACM Inhibitor Alliance

In December 2008, as amended in February 2010, February 2011 and July 2011, we formed a strategic alliance with Roche to discover, develop, and commercialize small-molecule drugs targeting CRACM channels, which we refer to as the Roche Agreement. The goal of this alliance was to develop a novel category of oral, disease-modifying agents for the treatment of RA and other autoimmune diseases and inflammatory conditions. The Roche Agreement was terminated by Roche effective on February 16, 2012.

As a result of termination of the Roche Agreement, the research, development and commercialization licenses granted to Roche by us have terminated. Ownership of all rights to all Licensed Compounds (as defined in the agreement) (including the scientific data relating to those compounds) has reverted to us. We have also received an exclusive license to use Roche's patent rights and know-how to research, develop, manufacture, commercialize and import any collaboration compound, including the Licensed Compounds. We are obligated to pay a low single digit royalty on a country-by-country and Licensed Product-by-Licensed Product (as defined in the agreement) basis upon commercialization of any Licensed Product.

IL-12/23 Inhibitors

We have identified several small molecule IL-12/23 inhibitors that represent a promising opportunity to develop drug candidates that could be administered orally and potentially address a wide range of serious inflammatory diseases with high unmet medical needs.

Exhibit 99.2

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Synta Announces $25.8 Million Registered Direct Offering of Common Stock

LEXINGTON, MA — July 26, 2012— Synta Pharmaceuticals Corp. (NASDAQ: SNTA) today announced that it has entered into common stock subscription agreements with members of its Board of Directors for the sale of 3,976,702 shares of its common stock in a registered direct offering at a price of $6.49 per share, for gross proceeds of approximately $25.8 million. The shares were offered directly to the purchasers without a placement agent, underwriter, broker or dealer. The sale and issuance of the shares is expected to close on or about July 27, 2012.

The net proceeds from the offering will be used to fund Synta's operations, including advancement of Synta's lead drug candidate, ganetespib, other research and development, clinical trials, manufacturing, intellectual property protection and enforcement, and working capital, and for other general corporate purposes.

The shares described above are registered under the Securities Act of 1933, as amended, pursuant to Synta Pharmaceuticals' effective shelf registration statement.

This press release shall not constitute an offer to sell or the solicitation of an offer to buy any securities of Synta Pharmaceuticals Corp. nor shall there be any sale of securities in any state or jurisdiction in which such an offer, solicitation or sale would be unlawful prior to registration or qualification under the securities laws of any such state or jurisdiction.

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.

Safe Harbor Statement

This media release contains forward-looking statements about Synta Pharmaceuticals Corp., including, but not limited to, statements relating to the anticipated use of proceeds from the sale. Such statements, 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, 2011 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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