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SCHEDULE 14A

Proxy Statement Pursuant to Section 14(a) of the Securities Exchange Act of 1934 (Amendment No.)

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SYNTA PHARMACEUTICALS CORP.

(Name of Registrant as Specified In Its Charter)

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Company Name: Synta Pharmaceuticals Corp. (SNTA)
Event: Jefferies 2016 Global Healthcare Conference
Date: June 10, 2016

<<Jordan Zauderer, Analyst, Jefferies LLC>>

Good afternoon and welcome to the 2016 Jefferies Global Healthcare Conference. My name is Jordan Zauderer with the Healthcare Investment Banking team. And it's my pleasure to introduce Paul Friedman, who is expected to become the Chairman and CEO of Synta Pharmaceuticals following the completion of Synta's merger with Madrigal Pharmaceuticals.

<<Paul A. Friedman, Chairman and Chief Executive Officer elect, of Madrigal Pharmaceuticals>>

Thanks Jordan. So good afternoon. I'm sure it's been a long day and a long conference for everybody. It's Friday and it's getting near the end. I want to spend a few minutes telling you about the new Madrigal, which of course is contingent on the successful closing of the planned merger with Synta. But before I get into the presentation I'm going to ask you to note the requisite fine print regarding forward-looking statements which as you'll see is even more wordy than usual because of language around the merger. We recommend that you take a look at these statements which are available on either of the company's websites.

These are the highlights of the transaction. It is an all-stock transaction. The expected ownership split will be 36% for Synta shareholders, 64% for Madrigal shareholders and the combined company will be known as Madrigal Pharmaceuticals. There is an ongoing private placement of $9 million into Madrigal prior to closing, which will add to the anticipated cash balance at closing. The merger has been approved by the boards of both companies and by Madrigal shareholders. And, we expect the close to be no later than the third quarter of 2016 subject to approval of Synta shareholders. There is a Synta shareholder meeting that is scheduled for July 21 and obviously there are other customary closing conditions.

The new Madrigal is going to be focused on a platform of selected thyroid hormone receptor beta-agonists of which MGL-3196, a once-a-day, oral, liver-directed compound is the lead. We believe it is the first truly beta selective agonist. I'm going to show you data bearing on that in a moment. While the initial indications for 3196 are nonalcoholic steatohepatitis, or NASH, and in familial hypercholesterolemia, there are other indications for which these agents could be useful and they could be studied either with the lead 3196 or with the pre-clinical backup MGL-3745.

3196 is poised to enter two proof-of-concept trials in the third quarter, one in NASH and one in heterozygous FH. A third trial in homozygous FH is also expected to initiate this year. These data readouts will continue throughout 2016 and 2017 and they offer multiple possible value creating opportunities. And importantly our cash — of combined resources are sufficient to reach these key clinical inflection points.

We are an experienced management team with proven track records in drug discovery, development and commercialization as was mentioned by Jordan. I'm the former CEO of Incyte, before that I was at DuPont and earlier at Merck, in total about 30 years in the industry. Becky Taub is Madrigal's Founder and will serve in the new company as CMO and Executive Vice President of R&D. She's a recognized expert in liver regeneration and diseases of the liver. She led teams that discovered Eliquis, while at DuPont and MGL-3196 while at Roche. In full disclosure, Becky and I are married to one another.

We are very fortunate to have persuaded Marc Schneebaum to serve as Chief Financial Officer. Marc has over 25 years of relevant experience and has been at Synta as a CFO since 2014.

This is a busy slide, but bear with me as I show you data demonstrating why we believe 3196 is the first bonafide thyroid hormone receptor beta selective molecule. On the lower left is a representation of the thyroid axis and it shows thyroxine or T4 as a prohormone that is enzymatically converted to the active hormone T3. The liver is a major organ for this conversion and the resulting T3 is active at both alpha and beta thyroid hormone receptors.

The group that Becky led at Roche used a novel, functional, cell-based assay to identify beta receptor selective agonist. And this assay turned out to be more discriminating than a straight receptor binding assay as previously employed by

other companies. The assay takes into account, among other things, the effects that accessory proteins have on thyroid hormone signaling. On the lower right, our results from this assay, showing about a thirty-fold selectivity for 3196 as an agonist for the beta receptor over the alpha receptor, while showing no selectivity for either T3, which is what you would expect, or for earlier compounds from other companies purported to be a beta selective.

And it's important to note that the beta selectively, as well as liver targeting, are characteristics of 3196 and are key to beneficial metabolic actions and avoid safety issues that earlier compounds have stumbled by having. 3196 has shown excellent safety, in particular, unlike another company's earlier thyroid receptor agonist. There have been no cartilage findings in chronic toxicology or ALT increases in human studies.

So why 3196 for NASH and why does the beta selectivity matter? Well, first I'd like to point out what I think most people know. The commercial potential for successful NASH therapy is huge. The potential worldwide NASH market is estimated to be as high as $35 billion to $40 billion. 2% to 3% of the U.S. population is believed to have NASH in the most conservative estimates, with those estimates rising to as high as 10%, driven by the high rates of diabetes and obesity. And while there are many compounds in development, there are not yet any approved.

And with respect to beta selectivity, it's well documented that thyroid hormone receptor-beta—thyroid hormone receptor-beta is key in the liver and that indicate that appropriate stimulation at this receptor is required for proper homeostasis of cholesterol and triglycerides and for maintaining liver health, in particular, avoiding fat accumulation and concomitant inflammation.

We believe that 3196 is a liver-directed selective agonism of the beta receptor with its result in pleiotropic upstream effects, which positions 3196 as a potential ideal therapy for NASH. These effects include reversing features of the metabolic syndrome and fatty liver disease and I'll be showing you data bearing on those points in just a moment.

I think it's important to note that the systemic hypothyroidism is more common in people with NASH than in the age matched general population. And while this is only an association, this observation raises the possibility, that having low thyroid function, as well as risk factors for NASH might put an individual at greater risk of developing the disease or for having a more severe circumstance should they develop the disease.

A more direct link is published data that show liver-specific hypothyroidism in NASH patients, which results from increased levels of a thyroid hormone degrading enzyme deiodinase 3, which is produced by increased numbers of activated fibrosis producing cells known as stellate cells in the inflamed NASH liver.

So NASH patients with advanced fibrosis, don't die of liver failure. They tend to die cardiovascular disease. There is increased risk of cardiovascular events and that's the primary mode of death. The fact that 3196 lowers LDL cholesterol and I'll show you this fairly profoundly, would indicate that it could be of a cardiovascular benefit to NASH patients.

And lastly, in the end, effectively treating the metabolic syndrome and the lipotoxicity in inflammation of fatty liver rather than the reactive fibrosis is the key to addressing the disease is recognized by the FDA, which has provided guidance that indicates that resolution of NASH without reducing fibrosis is an approvable endpoint. Much like the situation with hepatitis C virus in which after curing the patient and allowing the liver to regenerate eventually results in a lessening of fibrosis which can take a substantial amount of time. It's believed now that if you can treat NASH that eventually the same thing will happen in the NASH liver. But the concept of using fibrosis as your endpoint, that is the important aspect in NASH therapy, I think is misguided.

Now, with that background, let me show you what I think are some compelling preclinical and clinical data. This slide shows an example of improved safety of 3196 versus activated thyroid hormone, T3 in mice treated with either T3 or 3196 after establishment of diet-induced obesity. While both 3196 and T3 lower cholesterol profoundly in a dose dependent manner show on the left, T3 at all doses tested significantly decreases bone mineral density. 3196 does not do that. This propensity toward causing osteoporosis is but one example of T3 agonism at the alpha receptor which precludes its use in treating NASH.

Further, since as I mentioned, NASH patients breakdown T3 in the liver or want to try to treat NASH with a hormone. It would become even more of a non-starter as even more hormone would have to be administered in NASH, leading to unwanted peripheral effects.

One other point to note here which is going to pertain to the next couple of slides as well, is that the doses used here in these rodent models, give exposures in the rodents that are the same as the exposures that we have seen in humans, when I show you the human data. So we're not as is often the case with rodent models forced to use industrial doses of the agent to get the effects that we're looking for.

These are two studies showing improved health liver health in DIO mice, diet-induced obesity mice, with established disease. In the upper panel, we see that 3196 normalizes liver size. Liver tends to get big in this model. 3196 profoundly lowers liver triglycerides and improves insulin tolerance — improves insulin sensitivity, excuse me. The effects of liver size and triglycerides are greater than you see with rosiglitazone in these experiments. And rosiglitazone was reported in a 2011 publication to have modestly beneficial efficacy in human NASH.

The experiment in the lower panel shows impressive lowering. This is a longer term model where you begin to get elevated liver enzyme. You see normalization of liver enzymes and marked improvement histologically in the level of fat in the liver.

This is a slide showing a heat map analysis of NASH relevant proinflammatory and profibrotic gene expression again in the DIO model and again at the same doses as before. The transcripts measured are shown on the left up regulation of these transcripts in mice fed the high-fat diet is shown in the far left panel and is apparent and reflected in the red coloring when compared to a lean mouse control, which is the second from the left panel. And what you see in the dose dependent manner again is that 3196 normalizes expression of the NASH relevant genes and it does so to a greater extent than does rosiglitazone, which is shown in the far right panel.

And moving now to human studies both single and multiple ascending dose human studies have been completed. The latter was a 14-day study once-a-day dosing from five to 200 milligrams. And although the multiple ascending dose study was in otherwise healthy volunteers, they were selected to have cholesterols of at least 110 milligrams per deciliter to give us a chance to see effects on LDL cholesterol. 3196 is well-tolerated at all doses tested. No effects were seen on vital signs including heart rate. There was no perturbation of the central thyroid axis, for example, no changes in TSH. And, there were no effects on liver enzymes.

This last observation, I think is particularly notable in that earlier thyroid hormone receptor agonist that were tested in man from other companies elevated ALT within the first two weeks of treatment. The Phase 1 study of 3196 in combination with statins has also been completed. With API, with drug product and a completed chronic toxicology package in hand, long-term dosing in humans is enabled.

Now, here are the data from the 14-day multiple ascending dose human study, with once-daily oral treatment, showing a highly significant and dose dependent reduction of up to 30% for apolipoprotein B. Total LDL and non-HDL-cholesterol, as well as a very strong trend in triglyceride reduction of up to 60% were seen. Near-maximal effect for the 14-day dosing period is observed with the 80 milligram dose which is the fourth. I'll turn here to this dose. This one, two, three, four, this dose. This is the 80 milligram dose. That's the 100 milligram dose and that's a 200 milligram dose.

So you can see that by 80 milligrams you pretty much maxed out the effect at 14 days. And you see lipid decreases as early as two days after initiating therapy and obviously we don't know whether this is the maximum decrease that you would see on any one of these doses up to dose longer than the 14 days that were utilized in this MAD study.

So to summarize, 3196 is a once-a-day oral agent. I'm not showing the distribution data, but 3196 has high liver uptake and shows low tissue penetration which includes high protein binding in plasma outside the liver. It lowers insulin resistance, LDL cholesterol, triglycerides in ApoB, I'm going to get back to this lowering of Lp(a) a highly atherogenic and hard to treat particle, when I get into our plans in a moment for familial hypercholesterolemia.

The pre-clinical animal study show changes in their potential to be a benefit in the NASH setting and the safety profile is remarkably benign, there's no suppression of the central thyroid axis, there are no effects, no alpha effects, heart rate, bone, CNS effects, there have been no bone or cartilage findings in the long-term animal toxicology studies, the drug is not mutagenic. It's a stable entity chemically and there are no clinical liver enzyme elevations.

This all points to 3196 having the potential with its pleiotropic and cardio-beneficial actions to be positively differentiated from other compounds being developed for NASH. Just to mention three, obeticholic acid elevates LDL

cholesterol. You would think that that would potentially raise a regulatory bar for approval and limit the market opportunity. Elafibranor shows only modest maybe 10% reduction in LDL cholesterol. And anti-fibrotics don't address the basic pathophysiology of the disease, they treat. They're treating the end — one of the aspects of NASH.

One more notable point that should it make sense clinically is to combine agents in a market that's this large and in a population that's this large. There is no reason that you would not be able to combine something like 3196 with other agents that would have anti-fibrotic and/or anti-inflammatory activity.

This is the current study design that we have for our proof-of-concept Phase 2 study. It's a three-armed, blinded study. There'll be two doses of MGL-3196 and a placebo arm. These will be in patients who have liver biopsy proven NASH. This study will have just over 100 patients in it. We're debating now and discussing with the thought leaders whether the study duration will be six or nine months. We're leaning heavily toward nine months at this point. The primary endpoint will be a MRI-PDFF evaluation of liver fat at three months. And secondary endpoints will be biomarkers measurements at 12 and 24 weeks with a liver biopsy at the end of the study which would be either at six or nine months looking for reduction or resolution of NASH.

Now switching gears and moving to the second clinical area and which we're planning to evaluate 3196. Homozygous FH and heterozygous FH, which are caused primarily by inactivating mutations of either both or one LDL receptor gene, respectively, lead to early onset of cardiovascular disease resulting from a lifetime burden of elevated LDL cholesterol. While homozygous FH is rare, you can see that the heterozygous disease is not so uncommon.

Despite current and newer therapies such as the PCSK9 antibodies, homozygous and many especially severe heterozygous still do not achieve treatment goal because of the lifetime LDL cholesterol burden. And also because the aim of — and also because of the lifetime burden and the aim of reducing atherogenic plaque by attempting to reverse to recognize that substantial atherosclerosis is not uncommon at diagnosis in these patients. Very low levels of LDL cholesterol are sort down to as low as 70 milligrams per deciliter.

Additionally, some of the newer agents carry FDA label warnings of hepatotoxicity. We believe there's a significant commercial opportunity in FH for 3196 and that it will deliver additional LDL cholesterol and Lp(a) lowering safely on top of conditional, conventional treatment. I'm going to show you some data that bears on that. There's a word missing here it should say both LDL receptor dependent and independent cholesterol lowering are part of the mechanism of agonizing the thyroid beta receptor. This occurs by stimulating cholesterol, breakdown and elimination, by lowering ApoB and Lp(a), and interestingly by a decreasing levels of PCSK9, which this mechanism has been demonstrated to do. It actually looks a little bit like a PCSK9 heterozygous phenotype, which gives a very high level of cardio protection versus people who are homozygous.

And interestingly, angiopoietin-like protein 3, have been shown in gene expression experiments to also be driven down by agents that agonize the thyroid beta receptor. This is the target that Regeneron reported on within the last few weeks in four homozygous FH patients and got fairly dramatic LDL cholesterol lowering.

We've shown that 3196 lowers LDL in concert with statins in clinical and preclinical studies. It's known that thyroid agonist will lower cholesterol in LDL receptor knockout mice. Importantly eprotirome, which while it is a nonselective thyroid receptor agonist in the liver, it is certainly working through the beta receptor in a Phase 3 in heterozygous FH. It showed low statistically significant lowering of LDL cholesterol and of Lp(a). It had toxicology issues and safety issues which precluded it being further developed. But what this says is that if you agonize the thyroid beta receptor in these patients safely, you will lower LDL and Lp(a). And we believe therefore that 3196 has a very high probability of showing that in this patient population.

And the other important point here is that there are very few things that will lower the Lp(a) and 3196 almost certainly will do that by virtue of data that we have and by virtue of the fact that eprotirome was already been demonstrated to do it.

The study being proposed in the heterozygous is three-armed, blinded, and placebo-controlled with two doses of 3196 being tested. Patients on statins, as well as Zetia are eligible and we're finalizing whether a subgroup of PCSK9 antibody therapy will be limited in number or not in — of such subjects eligible. Duration is three months. The endpoints are those you would expect to see in such a study. And we expect this study will begin in the third quarter.

The HoFH trial is much smaller as you might expect. It is open-label. The patient serves as his or her own control. And in this study, 3196 maybe titrated. The endpoints are the same and we expect that this study will start this year.

So this is my last slide. It shows the timing of potential value drivers, with the three studies initiating in 2016 and a series of data readouts beginning in late 2016 and continuing throughout 2017. And for what I've shown you, we believe that the probability for efficacy in all three trials is substantial and we further believe the drug will be well-tolerated.

Thank you very much for your attention.

Additional Information about the Merger and Where to Find It

This Schedule DEFA14A does not constitute an offer to sell or the solicitation of an offer to buy any securities or a solicitation of any vote or approval. A definitive proxy statement on Schedule 14A and a proxy card were filed with the Securities and Exchange Commission ("SEC") on June 8, 2016 and was mailed to Synta's stockholders on or about June 13, 2016 seeking the required stockholder approvals in connection with the proposed transactions. The proxy statement will contain important information about Synta, Madrigal, the transaction and related matters. BEFORE MAKING ANY VOTING OR INVESTMENT DECISION, INVESTORS AND STOCKHOLDERS ARE URGED TO READ THE PROXY STATEMENT (INCLUDING ANY AMENDMENTS OR SUPPLEMENTS THERETO) AND ANY OTHER RELEVANT DOCUMENTS THAT SYNTA MAY FILE WITH THE SEC WHEN THEY BECOME AVAILABLE BECAUSE THEY WILL CONTAIN IMPORTANT INFORMATION ABOUT THE PROPOSED TRANSACTIONS. Stockholders may obtain, free of charge, copies of the definitive proxy statement and any other documents filed by Synta with the SEC in connection with the proposed transactions at the SEC's website (http://www.sec.gov), at Synta's website under the heading "Investors / SEC Filings", or by directing a written request to: Synta Pharmaceuticals Corp., 125 Hartwell Avenue, Lexington, MA 02421, Attention: Wendy Rieder, Esq.

Synta and its directors and executive officers and Madrigal and its directors and executive officers may be deemed to be participants in the solicitation of proxies from the stockholders of Synta in connection with the proposed transaction. Information regarding the special interests of these directors and executive officers in the merger will be included in the proxy statement referred to above. Additional information regarding the directors and executive officers of Synta is also included in the definitive proxy statement on Schedule 14A as filed with the SEC on June 8, 2016. This document is available free of charge at the SEC web site (www.sec.gov), at Synta's website under the heading "Investors / SEC Filings", or by directing a written request to Synta as described above.

Cautionary Statement Regarding Forward-Looking Statements

Any statements made herein relating to future financial or business performance, conditions, plans, prospects, trends, or strategies and other financial and business matters, including without limitation, the potential closing date of the transaction, the amount of Synta's net cash at closing, the prospects for commercializing or selling any drug candidates, are forward-looking statements within the meaning of the Private Securities Litigation Reform Act of 1995. In addition, when or if used in this press release, the words "may," "could," "should," "anticipate," "believe," "estimate," "expect," "intend," "plan," "predict" and similar expressions and their variants, as they relate to Synta, Madrigal or the management of either company, before or after the aforementioned merger, may identify forward-looking statements. Synta cautions that these forward-looking statements are subject to numerous assumptions, risks, and uncertainties, which change over time. Important factors that may cause actual results to differ materially from the results discussed in the forward-looking statements or historical experience include risks and uncertainties, including (i) the timing and

completion of the Company's merger with Madrigal, including its ability to satisfy the closing conditions of the Merger Agreement with Madrigal, including the closing condition that Synta have a minimum net cash amount of $28.5 million, (ii) the Company's continued listing on NASDAQ, (iii) the failure by Synta or Madrigal to secure and maintain relationships with collaborators; (iv) risks relating to clinical trials; (v) risks relating to the commercialization, if any, of Synta's or Madrigal's proposed product candidates (such as marketing, regulatory, product liability, supply, competition, and other risks); (vi) dependence on the efforts of third parties; (vii) dependence on intellectual property; and (viii) risks that Synta or Madrigal may lack the financial resources and access to capital to fund proposed operations. Further information on the factors and risks that could affect Synta's business, financial conditions and results of operations are contained in Synta's filings with the U.S. Securities and Exchange Commission, which are available at www.sec.gov. The forward-looking statements represent Synta's and Madrigal's estimate as of the date hereof only, and Synta and Madrigal specifically disclaim any duty or obligation to update forward-looking statements.