## 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 14, 2016

# SYNTA PHARMACEUTICALS CORP.

(Exact name of registrant as specified in its charter)

Delaware (State or other jurisdiction of incorporation) 001-33277 (Commission File Number)

45 Hartwell Avenue Lexington, MA (Address of principal executive offices) 04-3508648 (IRS Employer Identification No.)

**02421** (Zip Code)

(781) 274-8200

Registrant's telephone number, including area code

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 Thursday, April 14, 2016, Synta Pharmaceuticals Corp. ("*Synta*" or the "*Company*") held an investor conference call regarding the previously announced proposed transaction pursuant to the Agreement and Plan of Merger and Reorganization (the "*Merger Agreement*"), dated April 13, 2016, by and among the Company, Saffron Merger Sub, Inc., a Delaware corporation and a wholly owned subsidiary of Synta, Madrigal Pharmaceuticals, Inc., a Delaware corporation ("Madrigal"), a company focused on the development of novel small-molecule drugs addressing unmet needs in cardiovascular and metabolic diseases, pursuant to which the Merger Sub will be merged with and into Madrigal (the "Merger"), with Madrigal surviving the Merger as a wholly-owned subsidiary of the Company.

A copy of the transcript generated after the occurrence of this conference call is attached hereto as Exhibit 99.1 and incorporated herein by reference in its entirety.

#### Additional Information about the Merger and Where to Find It

This Form 8-K 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 and a proxy card will be filed with the SEC and will be mailed to Synta's stockholders seeking any 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., 45 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 Synta's Definitive Proxy Statement on Schedule 14A relating to the 2015 Annual Meeting of Stockholders, which was filed with the SEC on April 30, 2015. 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.

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#### **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 forwardlooking 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.

#### Item 9.01 Financial statements and Exhibits

(d) The following exhibits are furnished with this report:

Exhibit Number	Description
99.1	Transcript of Conference Call, dated April 14, 2016.
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### 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.

Date: April 14, 2016

### SYNTA PHARMACEUTICALS CORP.

/s/ Marc Schneebaum Marc Schneebaum Senior Vice President and Chief Financial Officer

### EXHIBIT INDEX

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### SYNTA PHARMACEUTICALS CONFERENCE CALL April 14, 2016

- C: Marc Schneebaum; Synta Pharmaceuticals Corp.; SVP, CFO
- C: Chen Schor; Synta Pharmaceuticals Corp.; President, CEO
- C: Paul Friedman; Cerulean Pharmaceuticals, Inc.; Executive Chairman
- C: Rebecca Taub; Madrigal Pharmaceuticals, Inc.; Founder, CEO

P: Mike King; JMP Securities; Analyst

#### Presentation

Operator: Good day, ladies and gentlemen, and welcome to the Synta/Madrigal Merger Announcement Conference Call.

At this time, for opening remarks, I will turn the call over to Marc Schneebaum, Synta's Senior Vice President and CFO. Please go ahead.

Marc Schneebaum: Good morning to everyone, and thank you for joining our call. Today we plan to discuss the merger agreement announced this morning between Synta Pharmaceuticals and Madrigal Pharmaceuticals.

Today's call is accompanied by a webcast slide presentation, which is available at www.syntapharma.com. With me on today's call are Chen Schor, President and Chief Executive Officer of Synta, Dr. Paul Friedman, who will become of Chairman and Chief Executive Officer of the combined company following the anticipated close of this merger, and Dr. Rebecca Taub, Founder and Chief Executive Officer of Madrigal and the designated Chief Medical Officer and Executive Vice President of Research and Development of the combined company.

Our plan today is to provide you with a brief overview of Madrigal. This is a new story to many of you, and a new direction for Synta, one we are very excited to be embarking on. Following our remarks, we'll open the call for a question-and-answer session.

Next slide.

Before we begin, I'd like to point out that we will be making forward-looking statements based on our current intents, beliefs and expectations, which are subject to certain risks and uncertainties. Forward-looking statements include statements with respect to financial projections and estimates, and our underlying assumptions, including, without limitation, whether and when our recently announced merger with Madrigal will close; the ability of the company to raise needed capital; the success of our merger with Madrigal, if consummated; the estimated size of the market for product candidates, the timing and success of the combined company's development and commercialization of its anticipated product candidates; and the availability of alternative therapies for the Company's target market.

The Company's actual results could differ materially from those anticipated in these forward-looking statements as a result of various factors. Additional information regarding factors that

could cause results to differ are available in the Risk Factors section of the Company's annual report on form 10-K, filed with the U.S. Securities and Exchange Commission on March 15, 2016 and in the Company's other periodic filings with the SEC.

### Next Slide.

I would also like to note that in connection with the merger, Synta will be filing a proxy statement with the SEC. We encourage you to read it and the other relevant materials filed by Synta with the SEC because these documents have or will have important information about the proposed transaction.

I will now turn over the call to Chen for the next slide.

Chen Schor: Thank you, Mark. And thank you all for taking the time to join us today. As Mark noted, this morning we issued a press release to announce that Synta has entered into a definitive merger agreement with privately held Madrigal Pharmaceuticals. This transaction is one that the Board and the management team of Synta are very excited about for reasons we will outline in a moment.

Under the terms of the merger agreement, Synta will acquire all outstanding shares of Madrigal Pharmaceuticals in exchange for approximately 253.9 million newly issued shares of Synta common stock. Upon completion of the proposed acquisition, existing Synta shareholders will own 36% of the combined company, and Madrigal shareholders will own 64% of the combined company. The transaction has been approved by the Board of Directors of both companies and by Madrigal shareholders, and is expected to close by the end of the third quarter of 2016, subject to customary closing conditions, including approval of the merger by the shareholders of Synta.

Upon the close of the transaction, the combined company will be known as Madrigal Pharmaceuticals. An investor syndicate that includes Bay City Capital, Dr. Fred Craves, founder of Bay City Capital and SQN LLC, a corporation held by Dr. Friedman and Dr. Taub, has committed to invest up to \$9 million in Madrigal prior to the closing of the merger.

The combined company intends to use these proceeds, in addition to the cash balance at closing, to fund the development of Madrigal's lead development compound, MGL-3196, through the completion of Phase 2 clinical studies in non-alcoholic steatohepatitis, or NASH, and familial hypercholesterolemia.

### Next slide.

The decision to pursue this agreement follows an extensive review of strategic alternatives by the Board and Senior Management team here at Synta. We believe that this agreement offers shareholders a very compelling opportunity for long-term value creations, which Paul will outline for you in a moment.

At the heart of this transaction is MGL-3196, a drug candidate with a unique lipid-lowering profile that has been validated through early clinical and preclinical studies. The combination of Synta and Madrigal offers its shareholders a company that is well-capitalized, with a lead program that has both a substantial potential commercial opportunity in NASH, and the fast-to-

market strategy in genetic lipid disorders. We believe the combined Company has the potential to achieve multiple value creating milestones over the next 18 months. Also, Synta's historical assets offer the potential opportunity for monetization for the combined company.

### Next slide.

The future of Madrigal Pharmaceuticals benefits from the leadership of two very accomplished physicians and drug developers: Dr. Friedman and Dr. Taub. Paul, who is likely well-known to many of you, is the former CEO and current Director of Incyte Pharmaceuticals, as well as the former President of DuPont Pharmaceutical Research, and Associate Professor of Medicine and Pharmacology at Harvard Medical School. Becky, who is a recognized expert in liver regeneration and in addition, has led teams to discover Eliquis and MGL-3196, Madrigal's lead compound. She is CEO of Madrigal, and will become Chief Medical Officer and Executive Vice President of Research and Development. And Marc, whom you've heard from a moment ago, will remain as CFO.

The combined company's Board of Directors will be comprised of seven representatives: one from Synta, five from Madrigal, and one who will be designated by mutual agreement. The combined company will be based in the Philadelphia area.

### Next slide.

With that, I will turn the call over to Dr. Friedman to walk you through the Madrigal value proposition. Afterwards, Dr. Taub will discuss MGL-3196, Madrigal's lead development compound in greater detail.

#### Paul?

Paul Friedman: Next slide, please. I'd like to start by saying that I am really excited to again be involved with a class of molecules of which MGL-3196 in my opinion, has substantial therapeutic, commercial, and investment potential. As many as of the salient points in this introductory slide are going to come up again in Becky's presentation, and in somewhat more detail, I am going to highlight several now and then pass the presentation on to her.

MGL-3196 is first in class as a truly thyroid beta selective agonist. And it has, in my opinion, very favorable pharmacokinetic and pharmacodynamic properties, with little to no CNS penetration. It is liver directed; it looks almost certainly to be a once-a-day drug, relatively low-dose molecule. It has been shown to be safe in long-term animal studies, and to-date in humans.

Becky will make the listed points about the opportunity in NASH in more detail, but in my strong opinion, the pleiotropic affects of MGL-3196 make it particularly promising in treating globally this increasingly prevalent clinical condition. In particular, the rapid and substantial decrease as seen in humans in lipid parameters, and related molecules as well as other key pathogenic parameters position MGL-3196 well, again in my opinion, to compete with other molecules in development.

In familial hypercholesterolemia, although eprotirome had significant safety issues and has been shown to not have significant selectivity from beta-thyroid receptor, it did show significant LBL-lowering on top of conventional therapy in Phase 3 studies in familial hypercholesterolemia.

Since these effects in liver are felt to be predominately mediated through the beta receptor, there is, in my opinion, highly reassuring proof of concept clinical data for a safe selective beta agonist such as MGL-3196.

With those introductory remarks, let me turn the presentation over to Becky.

Rebecca Taub: If we move now to slide 10. We believe, as Paul mentioned, that MGL-3196 is the first bona fide thyroid receptor beta selective molecule. This is based on the assays that Roche Pharmaceuticals, where MGL-3196 was discovered, used to select thyroid beta molecules. And this was a novel functional assay that went beyond what previous companies had done, which was a simple receptor biding assay.

The earlier compounds, as shown in the graph on the right, were reported to be thyroid beta receptor selective, but they actually showed no functional selectivity and, like thyroid hormone, the blue circle, activate the thyroid alpha receptor equally well as the beta receptor. Why is this important? Well, there are safety issues associated with activation of the alpha receptor, particularly in the heart and bone, and beta selectivity in liver targeting are key to the beneficial metabolic actions of thyroid hormone.

MGL-3196 has an excellent safety profile based on both pre-clinical and early clinical studies, and unlike prior analog mentioned by Paul, eprotirome, there were no cartilage findings in product toxicology studies with MGL-3196 in any species, at any dose. And no liver enzyme increases in human studies during a time period where other thyroid analogs showed a mild increase in ALT.

On slide 11, Madrigal's timeline is shown, the fact that MGL-3196 is Phase 2 ready and two significant indications, NASH and FH, or familial hypercholesterolemia. There is a backup molecule, which is pre-clinical; it has a similar profile to MGL-3196. And then we would also like to mention that additional thyroid analogs, or MGL-3196, could have activity and diseases of biliary system where there are genetic mutations that result in disorders that may be modulated favorably by Thyroid Beta agonists, sub-clinical hypothyroidism, genetic dyslipidemias, and other orphan thyroid diseases that are on the list of potential indications for these analogs.

The next slide, slide 12. Discussing the NASH opportunity, which we all recognize and we believe is a very large market, estimated at \$35 billion to \$40 billion by analysts. The epidemiology data varies; we're predicting at least 2% to 3% of the US population is thought to have NASH. Now, many KOLs here at the EASL meeting said the incidents are as high as 10% to 12% in the US population. And this number is expected to grow given the high rate in the growth of obesity and diabetes. Currently, there is no FDA approved treatment option.

MGL-3196 is a novel differentiated therapy relative to other NASH targets, and it is importantly, a cardio-protective therapy. There is a strong disease link; thyroid beta receptor activity is deficient in NASH livers. This is supported by both pre-clinical and clinical data, and we believe that both central hypothyroidism and liver-specific hypothyroidism contribute to this deficiency. As I mentioned, the clear differentiation of MGL-3196 is its cardiovascular benefit. MGL-3196 demonstrates LDL cholesterol lowering up to 30% in humans based on the Phase 1 studies. This

is a critical point of differentiation in NASH disease, which is more likely to lead to death by cardiovascular disease than liver disease.

At this point, there is a relatively well-defined regulatory path based on both EMA and FDA statements that indicate that resolution of NASH without reducing fibrosis is an approvable end-point. Recognition, from our perspective and we believe, that liver fibrosis will decrease with time after NASH resolves. This is similar to the reduction of fibrosis that occurs after cure of HCV. As the liver regenerates and repairs itself, the fibrosis reduces as well.

On slide 13, the thyroid beta receptor benefits across the spectrum of early to advance NASH are detected. This is evidence from human and animal data from both Madrigal, and multiple published studies. Beginning with the fatty liver, the metabolic benefits of thyroid beta agonists are well-known: reduction in liver fat and lipotoxicity are the proposed mechanism of metabolic benefit in the fatty liver; there is also an increase in insulin sensitivity, and as mentioned, reduction in LDL cholesterol and other lipids, including triglycerides and a reduction in atherosclerosis in animal models.

There are also direct anti-inflammatory and anti-fibrotic actions of thyroid beta, reduction in liver injury, promotion of liver regeneration. In addition, it has been shown that there is an increased incidence of hepatocellular carcinoma in hypothyroid individuals. We are hypothesizing that this analog may also be beneficial in prevention of HCC, which is increased in NASH as well as prevention of cardiovascular disease.

Slide 14 shows some preclinical data using MGL-3196 and a mouse model, a high fat diet model. Several studies were conducted of different duration of treatment with a high fat diet. What was consistently observed was with relatively low doses of 0.3 to 3 mg/kg of MGL-3196 as compared with Rosiglitazone, a PPAR gamma agonist. MGL-3196 shows a robust reduction in liver size, reduction of liver triglycerides.

These animals develop a fatty liver and MGL-3196 reduces hepatic triglycerides more than 50%. It showed similar insulin sensitivity as Rosiglitazone and reduction of liver enzyme ALT. Histology shown in the picture in the lower panel, you see there, is an almost complete reduction to normal of liver fat with following treatment with MGL-3196. In this particular study, these were all three-week treatment. So, this is a very rapid effect.

Slide 15 shows another experiment which is a heat map of gene expression study. This study was conducted over six months in duration, and what you can see is comparison between lane one and two, a high fat diet treated mice for six months show elevated inflammation in fibrosis in their liver where the lean animals do no show these elevations. And treatment with human comparable doses of MGL-3196, 0.3 milligram and 1 milligram shows an almost complete resolution of these fibrotic and inflammation transcripts, which are elevated in NASH, down to the level of the lean liver.

A comparator with Rosiglitazone, which has been shown to have some efficacy in humans, indicates the MGL-3196 performs better than Rosiglitazone in this study.

The next slide shows data — this is slide 16 — shows data from a 14-day multiple Phase 1 study in humans showing robust LDL and triglyceride lowering was observed with MGL-3196. This was

a once-daily oral treatment at doses from 5 mgs to 200 mgs per day. What you see is a highly significant and dose-dependant up to 30% reduction of LDL cholesterol and other lipid markers, such as apolipoprotein D, non-HDL-cholesterol, and there were strong trends in triglyceride reduction up to 60% reduction. There was a near-maximal effect in this study at 80 mgs per day dose.

Slide 17 shows a positioning of MGL-3196 in the NASH landscape. The upper panel shows the properties that we believe will be demonstrated in NASH patients treated with MGL-3196. We will see a reduction in NASH score, which is a measure of NASH activity. We believe there will be a small benefit in the fibrosis score in part because the fibrosis will reduce over time, following treatment with MGL-3196, a reduction in liver lipids, a prevention of further worsening of NASH, improved insulin sensitivity, and reduction in atherogenic lipids such as LDL cholesterol and triglycerides, which will lead to CV benefit.

MGL-3196 has been well tolerated in all human clinical studies to-date. These pleiotrophic and cardio-beneficial actions position MGL-3196 as a potential best-in-class NASH therapeutic, in our opinion. As a medical regulator, it reduces lipotoxicity, the driver of NASH and fibrosis. And cardio-vascular disease is the number one killer of NASH patients. MGL-3196 potently reduces LDL cholesterol and triglycerides.

There are opportunities based on these properties for differentiation from other NASH agents such as Intercept's obeticholic acid, which elevates LDL cholesterol, potentially in our opinion raising the regulatory bar for approval, and limiting the market opportunity. And Elafibrinor demonstrates only modest reduction in LCL cholesterol based on the recently published Phase 2 data. Anti-fibrotics do not address the underlying cause of NASH.

Efficacy on NASH and cardiovascular endpoints position MGL-3196 as an ideal NASH drug to be used in combination with anti-fibrotic, or antiinflammatory agents.

I will move on to slide 18 to discuss FH, which is a severe genetic dyslipidemia, particularly HoFH with early onset of cardiovascular disease and childhood, and certainly below the age of 20. The incidence of HeFH is estimated at 1 to 200 over 1 to 500 and HoFH is much rarer, but it has been demonstrated recently that these FHs are more common than has previously been thought, particularly in certain genetically homogeneous populations. Despite current or newer therapies, including the PCSK9 antibodies, HoFH and many HeFH, particularly the 10% of severe HeFH, are not achieving treatment goals, which in our opinion, provides a significant commercial opportunity for MGL-3196.

On slide 19 we are focusing on again, the pleiotropic mechanisms of lipid-lowering that is observed with the thyroid beta pathway, which leads to cholesterol breakdown and elimination, inhibition of PCSK9, reduction of ApoB, a severely atherogenic particle, which is called Lp(a) that is frequently elevated in FH and not well treated by existing therapeutics. This is a complimentary mechanism with statins and other mechanisms of cholesterol lowering.

As Paul mentioned, this is a clinically validated target in HeFH based on data from an earlier thyroid agonist.

On the final slide, I show potential near and long-term value drivers that are depicted across the next 18 months or so, in which we expect to start Phase 2 clinical trials in NASH and HeFH, followed by a small group of concept study in HoFH, and there will be data on early safety data, the top-line SH data, the data on the first part of NASH Study, which is reduction in liver fat at three months of treatment. And then there will be liver biopsy data at or near the end of 2017 from the NASH phase 2 Study.

And with that I will turn the call back to Paul.

Paul Friedman: I suspect this story is new to many of you, and we wanted to provide the opportunity if there are questions for you to be able to ask them now. So, I'd ask the operator to open the call up for any questions you may have.

### Q&A

Operator: (Operator Instructions) - Mike King, JMP Securities.

Mike King: I had a couple questions if you didn't mind. I wanted to start first from the Synta side. I was just wondering, I know you guys have AML 18 and 19, and I think also I-SPY running. And if those trials should provide positive readouts for ganetespib, would there be any plans to continue to take ganetespib forward in either of those indications?

Chen Schor: This is a new era for the combined Company, and the focus of the Company will be on MGL-3196. Indeed, for ganetespib there's a couple of ongoing clinical studies. The two key-studies that are still ongoing are the ovarian cancer and the sarcoma studies.

Our exposure to these studies is mainly to provide drug products and once data are available from these studies, there can be a decision what is the best step forward. Currently what we expect is that both ganetespib and STA-12-8666 provides a good opportunity for the future monetization of these assets so that the focus of the company will, indeed, be on MGL-3196 as the key outline in the clinical development in Madrigal's plan.

Does that answer your question?

Mike King: Yes it does. Just turning to Madrigal. What cursory work I could do on it on one of our data bases — couple of questions arising from that. First on MGL-3196, it looks like the molecule first entered clinical development in 2011 and then the updates only are as recent as 2013.

I'm just wondering what's transpired between 2013 and today; that's the first question. The second question is there's a mention of Via Pharmaceuticals in there. Could you just help us understand what the relationship is between Madrigal, Via, and any of the — are there any residual interests on the Via shareholders to milestones, royalties, anything like that?

Rebecca Taub: Yes. Via, which was a public company, underwent an ABC transaction at the end of 2011 in which the assets were bought by Madrigal — first bought by Bay City and then by Madrigal. So there was a transfer of assets, no transfer of shareholders or any of that.

So it was a clean transaction as far as that part of your question. Studies that have been pursued with Madrigal in the past couple years have focused on gathering (inaudible — microphone inaccessible) data leads. We mentioned that we wanted to have long-term toxicology data in both to de-risk the mechanism, but also to allow us to construct a NASH Study of sufficient duration to provide confidence that we would be able to achieve the type of registrational end-points that are required for that disease.

Mike King: And how long were those studies?

Rebecca Taub: So those were the standard chronic toxicology study ----

Mike King: It was like a two year tox? Was it [Cardon] Study, too?

Rebecca Taub: No. The repeat does study, that sixth-month in rabbit and nine-month in dog that completed at the end of 2015.

Operator: (Instructions) And I'm not showing any further questions at this time. I'd like to turn the call back to Paul Friedman.

Paul Friedman: Well, thank you for the question, Mike. And thanks for others who have listened. We're looking forward, going forward, to seeing or talking with as many of you as we can to familiarize you with what, in our opinion, is an exciting and promising opportunity therapeutically, commercially, and from an investment standpoint.

And we hope to see as many of you as we can in the weeks ahead and help you to get to know the Madrigal story in greater detail. And with that, Operator, let's terminate the call.

Good morning and thank you all so much.

Operator: Ladies and gentlemen, this does conclude today's presentation you may now disconnect. Have a wonderful day.