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): **May 31, 2018**

MADRIGAL PHARMACEUTICALS, INC.
(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.)

Four Tower Bridge **200 Barr Harbor Drive, Suite 400** **West Conshohocken, Pennsylvania**	**19428**
(Address of principal executive offices)	(Zip Code)

(484) 380-9263
Registrant's telephone number, including area code

(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:

o Written communications pursuant to Rule 425 under the Securities Act (17 CFR 230.425)
o Soliciting material pursuant to Rule 14a-12 under the Exchange Act (17 CFR 240.14a-12)
o Pre-commencement communications pursuant to Rule 14d-2(b) under the Exchange Act (17 CFR 240.14d-2(b))
o Pre-commencement communications pursuant to Rule 13e-4(c) under the Exchange Act (17 CFR 240.13e-4(c))

Indicate by check mark whether the registrant is an emerging growth company as defined in Rule 405 of the Securities Act of 1933 (§230.405 of this chapter) or Rule 12b-2 of the Securities Exchange Act of 1934 (§240.12b-2 of this chapter).

Emerging growth company o

If an emerging growth company, indicate by check mark if the registrant has elected not to use the extended transition period for complying with any new or revised financial accounting standards provided pursuant to Section 13(a) of the Exchange Act. o

Item 8.01 Other Events.

On May 31, 2018 Madrigal Pharmaceuticals, Inc. (the "Company") issued a press release announcing positive top-line, 36-week results from a Phase 2 clinical trial of the Company's MGL-3196 in patients with biopsy-proven non-alcoholic steatohepatitis. A copy of the press release is attached hereto as Exhibit 99.1 and is incorporated herein by reference.

Item 9.01 Financial Statements and Exhibits.

(d) Exhibits

Exhibit Number	**Description**

99.1	Press Release Dated May 31, 2018.

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.

MADRIGAL PHARMACEUTICALS, INC.

By: /s/ Marc R. Schneebaum
Name: Marc R. Schneebaum
Title: Chief Financial Officer

Date: May 31, 2018

Madrigal's MGL-3196 Achieves Liver Biopsy Endpoints in Patients with Non-alcoholic Steatohepatitis (NASH) at 36 Weeks in Phase 2 Clinical Trial

— Statistically significantly more patients treated with MGL-3196 compared with placebo treated patients achieved a two point reduction in NAS (NAFLD activity score) on biopsy —

— Statistically significantly more patients treated with MGL-3196 compared with placebo treated patients achieved resolution of NASH on biopsy —

— A ≥30% fat reduction (MRI-PDFF) in MGL-3196 treated patients, at Week 12 predicted an improved NASH histologic response at Week 36, including 39% NASH resolution, which was statistically significant relative to placebo —

— MGL-3196 was well-tolerated with mostly mild and a few moderate AEs; 7 SAEs, none drug related, none occurring more than once, 5 in MGL-3196, 2 in placebo-treated patients (study randomized 2:1) —

— Conference call scheduled for 8:30 AM Eastern Time today —

CONSHOHOCKEN, Pa., May 31, 2018 — Madrigal Pharmaceuticals, Inc. (Nasdaq: MDGL) today announced positive top-line, 36-week results from a Phase 2 clinical trial in patients with biopsy-proven non-alcoholic steatohepatitis (NASH). In this trial, MGL-3196, a first-in-class, oral, once-daily, liver-directed, thyroid hormone receptor (THR) β-selective agonist, demonstrated statistical significance in the primary endpoint (p<0.0001), relative reduction of liver fat on magnetic resonance imaging-estimated proton density fat fraction (MRI-PDFF) at 12 Weeks in December 2017, and, reported here, statistically significant results in multiple Week 36 endpoints including key secondary endpoints, reduction and resolution of NASH (see Table below).

	MGL-3196	MGL-3196 MRI-PDFF Responders(1)	Placebo
Number of patients with baseline and end-of-study liver biopsies(2)	73	46	34
≥ 2 Point Decrease in NAS	56%	70%	32%
	p=0.02	p=0.001	
NASH Resolution	27%	39%	6.5%
	p=0.02	p=0.001	

(1)MGL-3196 MRI-PDFF Responders = MGL-3196 treated patients with >=30% relative fat reduction on Week 12 MRI-PDFF

(2)does not include one end-of-study liver biopsy that was inadequate

- MGL-3196 treated patients with ≥ 30% fat reduction on MRI-PDFF at Week 12 demonstrated a higher percentage of NAS reduction and NASH resolution
- In patients with NASH Resolution, 35% of MGL-3196 treated and no placebo patients had baseline NAS ≥5
- In MGL-3196 patients with NASH resolution, fibrosis also resolved in 50% of patients and was decreased statistically significantly relative to all placebo patients.

Other Week 36 endpoints and safety:

- Sustained, highly statistically significant (p<0.0001) reduction in liver fat compared with placebo on Week 36 MRI-PDFF; mean relative fat reduction MGL-3196 37%; placebo, 8.9%
- Sustained, statistically significant reductions in low-density lipoprotein cholesterol (LDL-C), triglycerides, ApoB and lipoprotein(a)
- Statistically significant reductions in liver enzymes, of greater magnitude with longer duration of MGL-3196 treatment. Statistically significantly more MGL-3196 treated than placebo patients had normalization of ALT
- Statistically significant reductions in fibrosis biomarkers in treated compared with placebo patients
- On liver biopsy, fibrosis was reduced by at least 1 point in 23% of placebo and 29% of MGL-3196 treated patients
- Very good all subject tolerability: mostly mild and a few moderate AEs which were balanced between drug treated and placebo patients
- An increase in incidence of a transient mild diarrhea at beginning of study, often a single episode, in MGL-3196-treated compared with placebo

"NASH is a common liver disease in the United States, with a growing prevalence, for which no FDA approved treatment is yet available," said Dr. Stephen Harrison, M.D., Principal Investigator of the study as well as Medical Director for Pinnacle Clinical Research, San Antonio, Texas, and Visiting Professor of Hepatology, Oxford University. "Compared with Week 12, at Week 36 MGL-3196 showed sustained

effects to reduce liver fat on MRI-PDFF, and more reduction in liver enzymes than placebo. MGL-3196 demonstrates improvement relative to placebo on measurements of NASH on liver biopsy, including resolution of NASH. Importantly, this study is the first to demonstrate a correlation between efficacy in a non-invasive imaging test, MRI-PDFF at 12 weeks, and improvement in NASH on liver biopsy at 36 weeks"

"The degree of NASH resolution, an approvable FDA endpoint, in patients who received MGL-3196 for 9 months we believe suggests a high likelihood of success in a larger trial with a somewhat longer treatment period in a Phase 3 study designed similarly to this Phase 2 study, pending regulatory agreement with such a design. Further, considering what we have learned regarding drug exposure and dosing, we believe there is potential to resolve NASH in as little as 9 months in 30-40% of patients receiving only MGL-3196, a well-tolerated once a day oral therapy," stated Paul Friedman, M.D., Chief Executive Officer of Madrigal.

Becky Taub, M.D., CMO and Executive VP, Research & Development of Madrigal stated, "We are excited by the results of this study that demonstrate that MGL-3196 has the potential to show a clear benefit in patients with NASH, including both reduction and resolution of NASH and improvement in multiple atherogenic lipids. Cardiovascular disease is the primary reason for death in patients with NASH. We look forward to advancing MGL-3196 in a Phase 3 clinical trial in patients with NASH."

Conference Call and Webcast Information

Madrigal will hold a conference call and webcast this morning at 8:30 a.m. ET. To access the conference call, please dial 833-660-2754 for domestic callers or 409-350-3497 for international callers. When prompted, provide the conference identification number, 3387639.

The conference call will also be webcast live and can be accessed at http://www.madrigalpharma.com/newsroom/presentations/ in the "Events and Presentations" section of the Madrigal website.

If you are unable to participate, a replay of the conference call will be available on the website under http://www.madrigalpharma.com/newsroom/presentations/.

Clinical Program Summaries for MGL-3196

NASH

Non-alcoholic Steatohepatitis (NASH) is a common liver disease in the United States and worldwide, unrelated to alcohol use, that is characterized by a build-up of fat in the liver, inflammation, damage (ballooning) of hepatocytes and increasing fibrosis. Although people with NASH may feel well and often do not know they have the disease, NASH can lead to permanent damage, including cirrhosis and impaired liver function in a high percentage of patients.

In October 2016, the first patient was treated in the ongoing Phase 2 trial of MGL-3196 for the treatment of NASH. The randomized, double-blind, placebo-controlled, multi-center Phase 2 study enrolled 125 patients 18 years of age and older with liver biopsy-confirmed NASH and included approximately 25 clinical sites in the United States.

Patients were randomized to receive either MGL-3196 or placebo in a 2:1 ratio.

The primary endpoint of the study, the reduction of liver fat at 12 weeks compared with baseline (relative change), assessed by MRI-PDFF, was achieved. Liver fat was reduced by 36.3% in all MGL-3196 treated patients (78) and 42.0% in a pre-specified group of high exposure MGL-3196 treated patients (44/78), as compared with 9.6% median reduction in liver fat in 38 placebo treated patients. These results were statistically significant ($p<0.0001$) for both MGL-3196 treatment groups. Further, 75% of the high-exposure MGL-3196 treated patients showed liver fat reductions of $\geq$30%. MGL-3196 treated patients also showed significant reductions relative to placebo in multiple atherogenic lipids including LDL-cholesterol, Lp(a), Apo B and triglycerides. At Week 12, there were statistically significant reductions in MGL-3196 compared with placebo patients in liver enzyme, inflammatory and fibrosis biomarkers, consistent with a potential of MGL-3196 to reduce NASH and fibrosis.

Between Weeks 12 and 36, 4 placebo patients discontinued, 1 of the 4 had a repeat liver biopsy (Week 26) and was considered a completer, and one 36 week completer did not have a Week 36 liver biopsy; 4 MGL-3196 patients discontinued, and one week 36 patient had a repeat liver biopsy that was inadequate. Six of eight discontinuations were either lost to follow up or patient decision; one in each treatment group was related to an AE. No SAEs were related to treatment or occurred more than once. Most SAEs were related to a pre-existing condition and resolved or improved with treatment. The two SAEs in the placebo group were choledocholithiasis and pain from an umbilical hernia; in the MGL-3196 group, the SAEs were an aborted CVA occurring a few days after randomization, a kidney stone, a C. difficile colitis, an intra-abdominal bleed post end-of-study liver biopsy, and a worsening of spinal stenosis. In the placebo group, one patient's end-of-study liver biopsy had progressed from Stage 2 fibrosis at baseline to Stage 4, cirrhosis.

In addition, based on liver enzyme inclusion criteria, some patients are receiving extended treatment beyond 36 weeks for up to 36 additional weeks. All patients in this extension study will receive MGL-3196 and only non-invasive assessments will be made, including serial MRI-PDFF, safety labs, and circulating biomarkers. Additional information about the study [NCT02912260] can be obtained at www.ClinicalTrials.gov.

HeFH

Heterozygous familial hypercholesterolemia (HeFH), and a much rarer form called homozygous familial hypercholesterolemia (HoFH), are severe genetic dyslipidemias typically caused by inactivating mutations in the LDL receptor. Both forms of FH lead to early onset cardiovascular disease. HeFH, the most common dominantly inherited disease, is present in up to 1 in 200 people and as many as 40 percent of HeFH patients are not able to reach their cholesterol (LDL-C) reduction goals on these therapies, reflecting the lifetime burden of cholesterol buildup in their bodies.

In a completed Phase 2 HeFH trial, patients who were not at their LDL-C goal were randomized in a 2:1 ratio to receive either MGL-3196 or placebo, in addition to their current cholesterol lowering regimen, which included approximately 75% taking high intensity statins (20/40 mg rosuvastatin or 80 mg atorvastatin), and about 2/3 of patients also taking ezetimibe. MGL-3196 treated patients (placebo corrected) achieved highly significant ($p<0.0001$) LDL-C lowering of 18.8%, and 21% LDL-C lowering in those on an optimal dose of MGL-3196. LDL-C lowering was 28.5% in MGL-3196 treated compared to placebo in a prespecified group of patients who did not tolerate high intensity statin doses. Highly significant reductions ($p<0.0001$) relative to placebo were also observed with ApoB, triglycerides (TG)

(25-31%), apolipoprotein CIII (Apo CIII) and Lp(a) (25-40%) in all MGL-3196 treated patients and prespecified subgroups, irrespective of statin treatment.

MGL-3196 was well-tolerated with primarily mild and some moderate AEs, the numbers of which were balanced between placebo and drug-treatment groups.

About MGL-3196

Among its many functions in the human body, thyroid hormone, through activation of its beta receptor, plays a central role in controlling lipid metabolism, impacting a range of health parameters from levels of serum cholesterol and triglycerides to the pathological buildup of fat in the liver. Attempts to exploit this pathway for therapeutic purposes in cardio-metabolic and liver diseases have been hampered by the lack of selectivity of older compounds for the thyroid hormone receptor (THR)-β, chemically-related toxicities and undesirable distribution in the body.

Madrigal recognized that greater selectivity for thyroid hormone receptor (THR)-β and liver targeting might overcome these challenges and deliver the full therapeutic potential of THR-β agonism. Madrigal believes that MGL-3196 is the first orally administered, small-molecule, liver- directed, truly β-selective THR agonist. MGL- 3196 has demonstrated the potential for a broad array of therapeutically beneficial effects, improving components of both metabolic syndrome, such as insulin resistance and dyslipidemia, and fatty liver disease, including lipotoxicity and inflammation. These pleiotropic actions, coupled with an excellent safety profile, suggest that MGL-3196 could be the preferred treatment option for NASH.

About Madrigal Pharmaceuticals

Madrigal Pharmaceuticals, Inc. (Nasdaq: MDGL) is a clinical-stage biopharmaceutical company pursuing novel therapeutics that target a specific thyroid hormone receptor pathway in the liver, which is a key regulatory mechanism common to a spectrum of cardio-metabolic and fatty liver diseases with high unmet medical need. Madrigal's lead candidate, MGL-3196, is a first-in- class, orally administered, small-molecule, liver-directed, thyroid hormone receptor (THR) β-selective agonist that is currently in Phase 2 development for NASH and HeFH. For more information, visit www.madrigalpharma.com.

Forward-Looking Statements

This communication contains "forward-looking statements" made pursuant to the safe harbor provisions of the Private Securities Litigation Reform Act of 1995. Such statements contain words such as "expect," "could," "may," "will," "believe," "estimate," "continue," "future," or the negative thereof or comparable terminology and the use of future dates. Forward-looking statements reflect management's current knowledge, assumptions, judgment and expectations regarding future performance or events. Although management believes that the expectations reflected in such statements are reasonable, they give no assurance that such expectations will prove to be correct and you should be aware that actual results could differ materially from those contained in the forward-looking statements. Forward-looking statements are subject to a number of risks and uncertainties including, but not limited to, the company's clinical development of MGL-3196, the timing and outcomes of clinical studies of MGL- 3196, and the uncertainties inherent in clinical testing. Undue reliance should not be placed on forward- looking statements, which speak only as of the date they are made. Madrigal undertakes no obligation to update any forward-looking statements to reflect new information, events or circumstances after the date they are made, or to reflect the occurrence of unanticipated events. Please refer to Madrigal's filings with the U.S. Securities and Exchange Commission for more detailed information regarding these risks and uncertainties and other factors that may cause actual results to differ materially from those expressed or implied.

Investor Contact:
Marc Schneebaum, Madrigal Pharmaceuticals, Inc. IR@madrigalpharma.com

Media Contact:
Mike Beyer, Sam Brown Inc. mikebeyer@sambrown.com 312 961 2502