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): August 28, 2017

ANTHERA PHARMACEUTICALS, INC.

(Exact Name of Registrant as Specified in Charter)

Delaware	001-34637	20-1852016
(State or Other Jurisdiction of Incorporation)	(Commission File Number)	(I.R.S. Employer Identification No.)

25801 Industrial Boulevard, Suite B, Hayward, California	94545
(Address of Principal Executive Offices)	(Zip Code)

Registrant's telephone number, including area code: (510) 856-5600

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))

Indicate by check mark whether the registrant is an emerging growth company as defined in Rule 405 of the Securities Act of 1933 (17 CFR §230.405) or Rule 12b-2 of the Securities Exchange Act of 1934 (17 CFR §240.12b-2). Emerging growth company ☐

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. ☐

Item 8.01. Other Events.

On August 28, 2017, Anthera Pharmaceuticals, Inc. (the "Company") issued a press release announcing top line final data from the extension period of the Phase 2 BRIGHT-SC study of blisibimod in patients with Immunoglobulin A nephropathy (IgAN). A copy of the press release is filed herewith as Exhibit 99.1.

Item 9.01. Financial Statements and Exhibits.

(d) Exhibits.

Exhibit No.	Description
99.1	Press release dated August 28, 2017

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: August 28, 2017

Anthera Pharmaceuticals, Inc.

By: /s/ Craig Thompson

Craig Thompson
President and Chief Executive Officer
(Principal Executive Officer)

Exhibit Index

Exhibit No.	Description
99.1	Press release dated August 28, 2017

Exhibit 99.1

Anthera Announces Top Line Final Data from the Extension Period of the Phase 2 BRIGHT-SC Study of Blisibimod in Patients with IgA Nephropathy

- *Trend toward preservation of renal function (eGFR) in blisibimod treated patients*
- *Lack of disease progression, as measured by proteinuria in blisibimod treated patients*

HAYWARD, Calif., August 28, 2017 (GLOBE NEWSWIRE) -- Anthera Pharmaceuticals, Inc. (Nasdaq:ANTH) today announced top line final data from the extension of the randomized, double-blind, placebo controlled, Phase 2 BRIGHT-SC study of blisibimod in 58 patients with IgA nephropathy (IgAN). Patients were treated for up to 2 years, and all patients had the opportunity to complete at least 60 weeks of treatment.

Throughout the treatment period and for up to one year of additional follow up off treatment, blisibimod appeared to halt disease progression as measured by the mean estimate of 24-hour urinary protein excretion levels, also known as proteinuria. Specifically, in patients treated with blisibimod, the mean change in proteinuria was stable to trending slightly downward, whereas the mean levels increased for patients in the placebo arm.

Estimated 24 Hour Urine Protein Excretion

Study Week	0	24	48	60	96	120	144	168
Blisibimod - mean (grams)	2.02	2.17	1.86	1.86	1.88	2.12	2.19	1.18
Placebo - mean (grams)	2.26	2.16	2.01	2.42	2.89	3.35	3.22	4.68
N (blisibimod)	30	27	27	26	20	12	5	3
N (placebo)	28	23	17	17	11	9	4	2

Additionally, blisibimod demonstrated a trend toward preservation of renal function based upon individual rates of change in estimated glomerular filtration rate (eGFR), with an annualized improvement of +6.2mL/min/1.73 m^2 per year compared to a worsening of -4.8 mL/min/1.73 m^2 of body surface area with placebo.

As seen with previous interim analyses of the BRIGHT data, serum immunoglobulins IgA, IgG, and IgM, continue to demonstrate marked reduction throughout the treatment period.

Blisibimod was well tolerated with substantially more patients on the placebo arm discontinuing from the study than those receiving blisibimod. The most common adverse events seen in the blisibimod group over the entire study period were upper respiratory infection, nasopharyngitis, and injection site reactions. There were no incidences of serious infection in the blisibimod group and the overall rates of infection were similar in the two treatment groups.

“We are very pleased with the final extension data and believe that the recent findings, as well as the orphan designation granted by the US Food and Drug Administration (US FDA) provide a strong rationale to continue the clinical development of blisibimod for the treatment of IgA nephropathy,” shared Craig Thompson, President and CEO of Anthera. “We look forward to sharing these findings with the FDA and proceeding with a Phase 3 trial for patients with IgAN”

"It is good to see that the interim observations, that blisibimod may slow the worsening of proteinuria, persisted through the end of the study. The new observations showing long-term trends in preservation of glomerular filtration are exciting as these are goals for all IgAN therapies" said Professor Jonathan Barratt, PhD, FRCP, Reader, Department of Infection, Immunity & Inflammation, University of Leicester, U.K. "Finally, it is reassuring that blisibimod was well-tolerated; this contrasts with recent evidence of steroid risk in other trials."

About BRIGHT-SC

Fifty-eight (58) patients aged 18-65 with historical kidney biopsy proven IgAN (Oxford classification), persistent proteinuria (1-6 g/24hrs) despite treatment with angiotensin converting enzyme inhibitors (ACEi) and/or angiotensin receptor blockers (ARB), and estimated glomerular filtration rate > 30mL/min/1.73m^2 were equally randomized to receive either blisibimod (300mg/wk for 8 weeks and 200mg/wk thereafter; n=30) or matching placebo (n=28) for at least 24 weeks. Patients with advanced glomerular or tubulointerstitial scarring or rapidly progressive glomerulonephritis were excluded. After Week 24, patients were given the opportunity to receive blinded treatment for up to 104 weeks, discontinue treatment but continue to be followed, or discontinue from the study. Fifty-seven (57) patients received at least one dose of study medication and were included in the efficacy (mITT) and safety evaluations. The study was ended when all ongoing patients had completed at least Week 60. Forty seven patients (27 blisibimod, 20 placebo) completed at least 24 weeks of evaluation, 43 completed at least 60 weeks of evaluation, and 22 completed assessments through at least 108 weeks.

The study population was balanced between treatment groups with respect to age, weight, BMI, demographic and disease characteristics. Patients were enrolled from Asia (74%) and Europe (26%). All patients were taking an ARB and 52% were also taking an ACEi. Mean time since diagnosis was 2.2 years, mean estimated 24-hour urine protein was 2.14 grams, mean estimated glomerular filtration rate 68.25 ml/min/1.73 m^2 body surface area, and mean kidney biopsy Oxford Classification Derived Score was 6.2.

All patients were treated with an optimal, stable dose of ACEi and/or ARB for a minimum of 90 days prior to randomization and this therapy was continued throughout the trial as background medication. Patients were not allowed to receive corticosteroids for the treatment of IgA nephropathy within 3 months or immunosuppressives within 6 months of screening; however, these medications could be initiated as rescue treatment after randomization.

The BRIGHT-SC study was originally designed as a two-part Phase 2/3 study with a target enrollment of 200 patients. Part A was a 24-Week study of the effects of blisibimod on proteinuria, and Part B was an extension phase in which long term effects on the prevention of end stage renal disease would be assessed. The primary endpoint of the study was the number of patients who achieved a partial or complete response in urinary protein excretion at Week 24. A partial response was defined as achieving proteinuria ≤1g/24hrs, and a complete response as follows: for patients with baseline proteinuria ≥1g/24hrs but ≤2g/24hrs, achievement of proteinuria ≤1.0g/24hr AND a 50% reduction from baseline at 2 consecutive visits; for patients with baseline proteinuria > 2g/24hrs, achievement of proteinuria ≤1.0g/24hr OR a 50% reduction from baseline at 2 consecutive visits. Due to slow recruitment, enrollment was curtailed at 58 patients and the study was converted to a Phase 2 study. An observed case, interim analysis was conducted when all patients had the opportunity to complete Week 24 and another at Week 48, results of which were previously announced. Mean effects by treatment group on proteinuria and certain measures of expected pharmacology (circulating B cells and B cell subpopulations, serum immunoglobulins) were analyzed and reported to Anthera by an independent statistician, with the treatment blind maintained for the patient, investigator, and sponsor personnel.

About IgA Nephropathy

IgA nephropathy (IgAN, also known as Berger's disease) is the most common cause of primary glomerulonephritis worldwide, occurring more frequently in Asia than in Europe or North America. Patients express under-glycosylated immunoglobulin A1 (IgA1) which is immunogenic and targeted by other immunoglobulins. The resulting IgA-containing immune complexes are deposited in the kidney, causing inflammation with consequent blood and protein leakage into the urine. The disease typically progresses slowly, but as many as 40-50% of adults will eventually develop end-stage-renal disease and require dialysis or kidney transplant. The current management of IgAN is non-specific treatment aimed at blood pressure control and reduction of proteinuria with angiotensin converting enzyme inhibitors (ACEI) and/or angiotensin II receptor blockers (ARBs); corticosteroids and immunosuppressive therapy are used in some patients but benefits are uncertain.

About Blisibimod

Blisibimod is a selective peptibody antagonist of the B-cell activating factor (BAFF) cytokine. BAFF is a tumor necrosis family member and is critical to the development, maintenance and survival of B-cells. It is primarily expressed by macrophages, monocytes and dendritic cells and interacts with three different receptors on B-cells: BAFF receptor, or BAFF-R; B-cell maturation antigen, or BCMA; and transmembrane activator and cyclophilin ligand interactor, or TACI. BAFF-R is expressed primarily on peripheral B-cells. Blisibimod consists of a novel BAFF binding domain fused to the N-terminus of the Fc region of human IgG1 antibody. Blisibimod binds to both membrane and soluble BAFF, and inhibits the interaction of BAFF with its receptors.

About Anthera Pharmaceuticals, Inc.

Anthera Pharmaceuticals is a biopharmaceutical company focused on developing and commercializing products to treat serious and life-threatening diseases, including exocrine pancreatic insufficiency and IgA nephropathy. Additional information on Anthera can be found at www.anthera.com.

Safe Harbor Statement

Any statements contained in this press release that refer to future events or other non-historical matters, including statements that are preceded by, followed by, or that include such words as "estimate," "intend," "anticipate," "believe," "plan," "goal," "expect," "project," or similar statements, are forward-looking statements made pursuant to the safe harbor provisions of the Private Securities Litigation Reform Act of 1995. These forward-looking statements are based on Anthera's expectations as of the date of this press release and are subject to certain risks and uncertainties that could cause actual results to differ materially as set forth in Anthera's public filings with the SEC, including Anthera's Quarterly Report on Form 10-Q for the quarter ended June 30, 2017. Anthera disclaims any intent or obligation to update any forward-looking statements, whether because of new information, future events or otherwise, except as required by applicable law.

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