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Anthera Reports Blisibimod BRIGHT-SC IgA Nephropathy Continues to Demonstrate Positive Trends in the Week 48 Analysis

- Lower proteinuria in blisibimod versus placebo treated patients
- Changes in circulating B-cells and serum immunoglobulins consistent with BAFF inhibition

HAYWARD, Calif., Dec. 06, 2016 (GLOBE NEWSWIRE) -- Anthera Pharmaceuticals, Inc. (Nasdaq:ANTH) today announced positive trends from the Week 48 analysis of the Phase 2 BRIGHT-SC proof-of-concept study in 57 patients with IgA nephropathy.

The effects of blisibimod versus placebo were assessed through at least the 48 week time point in all patients in the BRIGHT-SC study. Patients enrolled in the BRIGHT-SC study had biopsy-proven IgA nephropathy with a mean proteinuria level of 2.4 grams and an estimated glomerular filtration rate of less than 70 mL/min/1.73m^2 - indicative of stage 2 chronic kidney disease per the National Kidney Foundation. Patients were on background angiotensin converting enzyme inhibitors or receptor blockers, but corticosteroids were not permitted during the trial.

A positive trend on proteinuria of blisibimod in patients with biopsy-proven IgA nephropathy was observed. Consistent with the previously announced Week 24 analysis, blisibimod treated-patients over time demonstrated stable to slightly decreasing levels of estimated 24 hour urinary protein excretion, as assessed by urinary protein to creatinine ratio (PCR), as compared to slowly increasing levels of proteinuria in the placebo group. 44 of the original 57 patients had a Week 48 observation and 22 patients had a Week 96 observation at the time of this analysis.

Week		n	24 hour urinary protein excretion (grams; mean [SD])
Baseline	Blisibimod	30	2.02 (0.73)
	Placebo	27	2.35 (1.09)
Week 48	Blisibimod	27	1.86 (1.04)
	Placebo	17	2.07 (1.46)
Week 96	Blisibimod	14	1.87 (0.79)
	Placebo	8	3.02 (2.07)

Total B cell counts and serum immunoglobulins IgA, IgG, and IgM, continued to demonstrate marked reduction, consistent with data from the Week 24 analysis as well as other studies with blisibimod.

No safety or tolerability concerns were observed with blisibimod during routine reviews of the unblinded trial data by the study's independent Data and Safety Monitoring Board.

"The sustained effects of blisibimod versus placebo on proteinuria after up to 2 years of treatment are very encouraging for IgA nephropathy patients, for whom no treatments exist," said Jonathan Barratt, MD, Reader in the Department of Infection, Immunity & Inflammation, University of Leicester, Honorary Consultant Nephrologist at Leicester General Hospital, and Head of the Postgraduate Specialty School of Clinical Academic Training at Health Education East Midlands. "We look forward to receiving longer term data from the study which will provide more conclusive assessment of blisibimod on the progression of kidney disease."

"The Week 48 observations from the BRIGHT-SC study continue to demonstrate consistent pharmacological effects on B cells and serum immunoglobulins, and suggest an effect on proteinuria," said William Shanahan, MD, Anthera's Chief Medical Officer. "We believe proteinuria is a robust surrogate biomarker for IgA nephropathy disease activity, and we remain optimistic that a longer-term evaluation in greater numbers of patients could provide further validation of blisibimod's effects on IgA nephropathy by demonstrating continuing effects on proteinuria and preservation of glomerular function."

The full dataset from this study will be submitted and presented at an upcoming scientific conference.

About BRIGHT-SC

The study enrolled 57 patients, 44 of whom completed assessments through a minimum of 48 weeks. Patients with persistent proteinuria (1-6 g/24hrs) prior to enrollment were randomized to receive either blisibimod (300mg/wk for 8 weeks and 200mg/wk thereafter) or matching placebo over background angiotensin converting enzyme inhibitors (ACEi) or angiotensin receptor blockers (ARB) for up to 104 weeks and are followed thereafter in the absence of study drug to assess longer term outcome. All patients were treated with an optimal dose of ACEi or ARB for a minimum of 90 days prior to randomization and this therapy was continued throughout the trial as background medication for all patients. Steroid intervention was not permitted during the trial.

The BRIGHT-SC study was originally designed as a phase 2/3 study with a target enrollment of 200 patients with renal biopsy verified IgA nephropathy, 24 hour urine protein excretion of 1-6 grams by urinary protein to creatinine ratio (PCR), and an estimated glomerular filtration rate (eGFR) of more than 30 ml/min/1.73 m^2 of body surface area. The original 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 is 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 57 and the study was converted to phase 2. An observed case analysis was conducted when all patients had the opportunity to complete Week 24 and topline results were previously announced. Based upon the Week 24 results, applications for Orphan Drug and Breakthrough Status have been filed with the FDA, with responses expected by late 1Q2017.

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 Anthera Pharmaceuticals, Inc.

Anthera Pharmaceuticals is a biopharmaceutical company focused on developing and commercializing products to treat serious and life-threatening diseases, including lupus, lupus with glomerulonephritis, IgA nephropathy, and exocrine pancreatic insufficiency due to cystic fibrosis. Additional information on the Company 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 September 30, 2016. 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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