REATA ANNOUNCES POSITIVE PHASE 2 DATA FOR BARDOXOLONE METHYL IN CKD CAUSED BY ALPORT SYNDROME AND AUTOSOMAL DOMINANT POLYCYSTIC KIDNEY DISEASE

STATISTICALLY SIGNIFICANT IMPROVEMENT IN KIDNEY FUNCTION MAINTAINED IN ALPORT SYNDROME PATIENTS AFTER 48 WEEKS OF TREATMENT

STATISTICALLY SIGNIFICANT RETAINED BENEFIT OF 4.1 ML/MIN IN ALPORT SYNDROME PATIENTS FOLLOWING 48 WEEKS OF TREATMENT AND 4 WEEKS OF DRUG WITHDRAWAL

STATISTICALLY SIGNIFICANT IMPROVEMENT IN KIDNEY FUNCTION OBSERVED IN ADPKD PATIENTS AFTER 12 WEEKS OF TREATMENT

CONFERENCE CALL WITH MANAGEMENT SCHEDULED FOR TODAY JULY 23RD AT 8:00 AM ET

IRVING, Texas—July 23, 2018—Reata Pharmaceuticals, Inc. (Nasdaq: RETA), a clinical-stage biopharmaceutical company, today announced results from two Phase 2 studies of bardoxolone methyl (bardoxolone) in patients with chronic kidney disease (CKD). Reata reported positive one-year results for the Phase 2 portion of CARDINAL, a study of bardoxolone in patients with CKD due to Alport syndrome, and positive final results for the Phase 2 autosomal dominant polycystic kidney disease (ADPKD) cohort of PHOENIX.

In the Phase 2 portion of CARDINAL, significantly increased estimated glomerular filtration rate (eGFR) at Week 48 from baseline (n=25) of 10.4 mL/min/1.73 m^2 ($p<0.0001$) was observed in patients treated with bardoxolone. Reata collected historical eGFR data for 22 out of the 25 Phase 2 study subjects. The historical eGFR data demonstrate that the Phase 2 study subjects' kidney function was declining at an average annual rate of 4.2 mL/min/1.73 m^2 prior to study entry. The observed 10.4 mL/min/1.73 m^2 improvement after one year of treatment with bardoxolone represents a recovery of approximately two years of average eGFR loss.

Significantly increased eGFR from baseline at Week 52 after withdrawal of active drug for four weeks (the retained eGFR benefit) by a mean of 4.1 mL/min/1.73 m^2 ($p<0.05$) was also observed with bardoxolone treated patients. These results provide evidence that bardoxolone may delay or prevent kidney failure. The U.S. Food and Drug Administration (FDA) has provided Reata with guidance that, in Alport syndrome patients, a significant improvement in placebo-corrected retained eGFR after one year of bardoxolone treatment may support accelerated approval and, after two years of bardoxolone treatment, may support full approval.

With respect to safety in the Phase 2 portion of CARDINAL, no treatment-related serious adverse events have been reported, and the reported adverse events have generally been mild to moderate in intensity. Twenty-five patients were available for the analysis, and no discontinuations were due to bardoxolone treatment.

In the Phase 2 ADPKD cohort of PHOENIX, significantly increased eGFR at Week 12 from baseline, which was the primary endpoint of the study, of 9.3 mL/min/1.73 m^2 ($p<0.0001$) was observed in bardoxolone treated patients. Reata collected historical eGFR data for 29 of the 31 Phase 2 study subjects. The historical eGFR data demonstrate that these subjects' kidney function was declining at an average annual rate of 4.8 mL/min/1.73 m^2 prior to study entry. The observed 9.3 mL/min/1.73 m^2 improvement after 12 weeks of treatment with bardoxolone represents a recovery of approximately two years of average eGFR loss.

With respect to safety in the Phase 2 ADPKD cohort of PHOENIX, no treatment-related serious adverse events were reported, and the reported adverse events were generally mild to moderate in intensity. Twenty-eight patients were available for the analysis, and only one patient (3%) discontinued for a treatment-related adverse event of fatigue.

"The results announced today add to the large body of clinical evidence that bardoxolone treatment has the potential to prevent or delay kidney failure in rare forms of chronic kidney disease" said Warren Huff, Reata's President and Chief Executive Officer. "Importantly, today's CARDINAL data demonstrate that one year of bardoxolone treatment can improve kidney function in Alport syndrome patients that have had progressive loss of kidney function while on standard of care. Further, the magnitude of the observed retained eGFR benefit after withdrawal of drug versus the historical rate of eGFR loss suggests that the Phase 3 portion of CARDINAL is conservatively powered with respect to the key secondary endpoint of retained eGFR benefit. Additionally, the eGFR increase at Week 12 observed in patients with ADPKD suggests that long-term improvements from treatment with bardoxolone in other forms of CKD may translate to patients with ADPKD."

Reata management will host a call to discuss these results today, July 23, 2018 at 8:00 a.m. ET.

CONFERENCE CALL INFORMATION

Date:	Monday, July 23, 2018
Time:	8:00 a.m. E.T.
Audience Dial-in (toll-free):	(844) 348-3946
Audience Dial-in (international):	(213) 358-0892
Passcode:	4992288
Webcast Link:	https://edge.media-server.com/m6/p/uyivv65d

About the Retained eGFR Benefit Analysis

FDA has accepted for approval in rare forms of CKD the placebo-corrected "retained eGFR benefit" after withdrawal of drug. The "on treatment" eGFR improvement is the true clinical benefit to the patient, but FDA requires additional evidence that an intervention may delay kidney failure. Withdrawal of drug after long-term treatment provides evidence that a drug either protected or harmed the kidney during treatment. If retained eGFR is higher than placebo, the drug treatment protected kidney function, and if retained eGFR is lower than placebo, the drug treatment harmed kidney function. A positive retained eGFR benefit provides evidence that drug treatment may delay kidney

failure. In April 2018, the FDA approved tolvaptan for ADPKD on a placebo-corrected (but below baseline) retained eGFR benefit of 1.27 mL/min/1.73 m^2 (tolvaptan: -2.34, placebo: -3.61; Torres *et al* NEJM 2017).

About the CARDINAL Clinical Study

CARDINAL is an international, multi-center, Phase 2/3 study enrolling patients from 12 to 60 years old with a confirmed genetic or histological diagnosis of Alport syndrome, baseline eGFR values between 30 to 90 mL/min/1.73 m^2, and on stable renin-angiotensin-aldosterone system blockade unless contraindicated. The Phase 2 portion of CARDINAL is open-label and enrolled 30 patients. The Phase 3 portion of CARDINAL is double-blind, placebo-controlled, and will randomize approximately 150 patients on a 1:1 basis to once-daily, oral bardoxolone or placebo.

The Phase 3 primary efficacy endpoint is the on-treatment eGFR change from baseline in bardoxolone-treated patients relative to placebo at Week 48. The key secondary endpoint of the Phase 3 portion of the trial is the change from baseline in retained eGFR benefit after 48 weeks on-treatment and four weeks off-treatment relative to placebo and is designed to demonstrate that bardoxolone has disease-modifying activity in Alport syndrome patients. Based upon guidance from the FDA, the 52-week retained eGFR benefit data may support accelerated approval under subpart H. After withdrawal, patients will be restarted on study drug with their original treatment assignments and will continue on study for a second year. The second-year on-treatment eGFR change will be measured after 100 weeks and the retained eGFR benefit will be measured after withdrawal of drug for four weeks at Week 104. Based upon guidance from the FDA, the year-two retained eGFR benefit data may support full approval.

About the PHOENIX Clinical Study

The Phase 2 PHOENIX program is studying bardoxolone in patients with ADPKD, IgA nephropathy, focal segmental glomerulosclerosis, and CKD associated with type 1 diabetes. Patients receive bardoxolone open-label, orally, once-daily for 12 weeks, and the primary efficacy endpoint is change from baseline in eGFR after 12 weeks of treatment. Endpoints will be assessed for each cohort separately.

About Alport Syndrome

Alport syndrome is a rare, genetic form of CKD caused by mutations in the genes encoding type IV collagen, which is a major structural component of the glomerular basement membrane (GBM) in the kidney. The abnormal expression of type IV collagen causes loss of GBM integrity, abnormal leakage of proteins through the GBM, and excessive reabsorption of protein in the proximal tubules of the kidney. Like other forms of CKD, excessive reabsorption of protein in the tubules induces oxidative stress, chronic inflammation, and renal interstitial inflammation and fibrosis.

Alport syndrome affects approximately 30,000 to 60,000 people in the United States according to the Alport Syndrome Foundation. A majority of patients with Alport syndrome develop CKD and approximately 50% of male patients with x-linked disease require dialysis or a kidney transplant by the age of 25. There are currently no approved therapies to treat Alport syndrome.

About Autosomal Dominant Polycystic Kidney Disease

ADPKD is a genetic form of CKD caused by mutations in PKD1 and PKD2 genes leading to inflammation that stimulates the formation of fluid-filled cysts in the kidneys that cause pain and progressive loss of kidney function. ADPKD is the leading inheritable cause of kidney failure with an estimated 400,000 patients in the United States.

About Bardoxolone

Bardoxolone is an experimental, oral, once-daily activator of Nrf2, a transcription factor that induces molecular pathways that promote the resolution of inflammation by restoring mitochondrial function, reducing oxidative stress, and inhibiting pro-inflammatory signaling. The FDA has granted orphan designation to bardoxolone for the treatment of Alport syndrome and pulmonary arterial hypertension. The European Commission has granted orphan designation in Europe to bardoxolone for the treatment of Alport syndrome. In addition to CARDINAL and PHOENIX bardoxolone is currently being studied in CATALYST, a Phase 3 trial for the treatment of connective tissue disease-associated pulmonary arterial hypertension and AYAME, a Phase 3 trial for the treatment of diabetic kidney disease.

About Reata Pharmaceuticals, Inc.

Reata is a clinical-stage biopharmaceutical company that develops novel therapeutics for patients with serious or life-threatening diseases by targeting molecular pathways involved in the regulation of cellular metabolism and inflammation. Reata's two most advanced clinical candidates, bardoxolone and omaveloxolone, target the important transcription factor Nrf2 that promotes the resolution of inflammation by restoring mitochondrial function, reducing oxidative stress, and inhibiting pro-inflammatory signaling.

Forward-Looking Statements

This press release includes certain disclosures that contain "forward-looking statements," including, without limitation, statements regarding the success, cost and timing of our product development activities and clinical trials, our plans to research, develop and commercialize our product candidates, and our ability to obtain and retain regulatory approval of our product candidates. You can identify forward-looking statements because they contain words such as "believes," "will," "may," "aims," "plans," and "expects." Forward-looking statements are based on Reata's current expectations and assumptions. Because forward-looking statements relate to the future, they are subject to inherent uncertainties, risks, and changes in circumstances that may differ materially from those contemplated by the forward-looking statements, which are neither statements of historical fact nor guarantees or assurances of future performance. Important factors that could cause actual results to differ materially from those in the forward-looking statements include, but are not limited to, (i) the timing, costs, conduct, and outcome of our clinical trials and future preclinical studies and clinical trials, including the timing of the initiation and availability of data from such trials; (ii) the timing and likelihood of regulatory filings and approvals for our product candidates; (iii) the potential market size and the size of the patient populations for our product candidates, if approved for commercial use, and the market

opportunities for our product candidates; and (iv) other factors set forth in Reata's filings with the U.S. Securities and Exchange Commission, including its Annual Report on Form 10-K, under the caption "Risk Factors." The forward-looking statements speak only as of the date made and, other than as required by law, we undertake no obligation to publicly update or revise any forward-looking statements, whether as a result of new information, future events, or otherwise.

Contact
Reata Pharmaceuticals, Inc.
(972) 865-2219
info@reatapharma.com
http://news.reatapharma.com

Investor Relations:
Vinny Jindal
Vice President, Strategy
(469) 374-8721
ir@reatapharma.com

Media:
Matt Middleman, M.D.
LifeSci Public Relations
(646) 627-8384
matt.middleman@lifescipublicrelations.com