REATA PHARMACEUTICALS, INC. ANNOUNCES POSITIVE DATA FROM PART ONE OF MOXIE TRIAL OF OMAVELOXOLONE FOR FRIEDREICH'S ATAXIA

– *Omaveloxolone Induced Nrf2 and Improved Mitochondrial and Neurological Function* –

– *Company Planning to Initiate Part 2 of Trial During the Second Half of 2017* –

– *Data Presentation and Conference Call Scheduled for June 2nd* –

IRVING, Texas—June 1, 2017— Reata Pharmaceuticals, Inc. (Nasdaq: RETA) ("Reata" or "the Company"), a clinical-stage biopharmaceutical company, today announced positive data from Part 1 of the Company's Phase 2 trial (MOXIe) of omaveloxolone for the treatment of Friedreich's ataxia (FA). The trial demonstrated that in FA patients, omaveloxolone induced Nrf2, which is suppressed in FA patients, and this was associated with improvements in mitochondrial and neurological function. Dose-dependent and time-dependent effects on the modified Friedreich's Ataxia Rating Scale (mFARS) were observed at the pharmacodynamically active doses, and the maximum effect on mFARS was observed at the 160 mg dose level. The Company is planning to initiate Part 2 of MOXIe during the second half of 2017.

"We are greatly appreciative of Reata, the clinical investigators, and the study volunteers for conducting and participating in a well-designed and robust dose-escalation study. We find these results to be very exciting, and they are the ideal outcome for an early Phase 2 study. They exceed expectations in terms of safety and by demonstrating dose-dependent and clinically meaningful activity that correlated with biological activity," said Jennifer Farmer, the Executive Director of the Friedreich's Ataxia Research Alliance (FARA). "FARA and the FA community encourage urgency in advancing this program to Part 2 of the study to allow for further evaluation of efficacy and safety, as there are no approved therapies to slow progression or improve symptoms for individuals living with FA. Every day counts for our patient families."

The complete data will be presented by Dr. David Lynch, Director of the Friedreich's Ataxia Program at Children's Hospital of Philadelphia, during the afternoon of June 2, 2017 at 3:00pm EDT, after completion of the Patient-Focused Drug Development meeting hosted by FARA.

Study Design and Key Efficacy Endpoints

Part 1 of MOXIe was a double-blind, randomized, placebo-controlled, dose-ranging, multi-center, international trial that enrolled a total of 69 FA patients to receive placebo (n = 17) or omaveloxolone (n = 52) at doses of 5 mg to 300 mg given orally, once-daily for 12 weeks. Part 1 of MOXIe was designed to identify a safe and clinically active dose of omaveloxolone to study in Part 2. Part 1 randomized eight to 16 patients in a 3:1 ratio of omaveloxolone to placebo across seven dose levels and was not powered to demonstrate statistical separation from placebo at any single dose level. The study assessed pharmacodynamic measures of Nrf2 activation and mitochondrial function, as well as two key efficacy endpoints: a measure of muscle function, peak work during maximal exercise testing (primary endpoint), and a measure of neurological function that has been the primary endpoint for prior registrational studies in FA, the mFARS (secondary endpoint).

The MOXIe Part 1 dose-escalation trial identified 160 mg of omaveloxolone as the optimal dose associated with pharmacodynamic measures of Nrf2 induction and improvements in mitochondrial function. Nrf2, the target of

omaveloxolone, is suppressed in FA patients leading to impaired mitochondrial function. Impaired mitochondrial function in FA patients has been shown to correlate with impaired neurological function as assessed by the mFARS. Dose-dependent induction of Nrf2 transcriptional target proteins, ferritin and gamma-glutamyl transferase (GGT), was observed at doses as low as 20 mg and reached statistical significance from baseline at doses of 80 mg and higher. Two markers of cellular metabolism, aspartate aminotransferase (AST) and creatine kinase (CK), were monitored to assess improvements in mitochondrial function. AST delivers substrate to the mitochondria for energy production as part of the malate shuttle, and CK is involved in the utilization of adenosine triphosphate (ATP) throughout cells. Dose-dependent modulation of AST and CK was observed and statistically significant changes from baseline were observed at doses of 80 and 160 mg.

Omaveloxolone improved neurological function in a dose- and time-dependent manner as assessed by mFARS. Across all doses, omaveloxolone significantly improved (by reducing) mFARS by 2.5 points from baseline ($p<0.001$) and by 1.1 points versus placebo (non-significant). At the 160 mg dose, omaveloxolone improved mFARS by 3.8 points versus baseline ($p=0.0001$) and by 2.3 points versus placebo ($p=0.06$).

Week 12 mFARS Change from Baseline

	All Patients		No Foot Deformity	
	n	Week 12	n	Week 12
Placebo	17	-1.4	7	-1.6
Omaveloxolone				
All	52	-2.5**	30	-3.3**
80 mg	6	-2.9*	4	-4.2**
160 mg	12	-3.8**	4	-6.0**
*p<0.05 vs baseline				
**p<0.005 vs baseline				

Omaveloxolone produced greater improvements in mFARS in patients that did not have a preexisting musculoskeletal foot deformity that causes high arched feet, called *pes cavus*. Presence of this deformity can affect the patients' ability to use their legs, walk, and perform neurologic and exercise testing independent of their ataxia. In patients without this foot deformity, omaveloxolone significantly improved mFARS by 3.3 points from baseline ($p<0.001$) and by 1.7 points from placebo (non-significant). In this group, the maximum effect on mFARS was observed at the 160 mg dose level. At this dose, omaveloxolone improved mFARS by 6.0 points from baseline ($p<0.0001$) and by 4.4 points versus placebo ($p=0.01$). Twelve weeks of treatment at the optimal dose of 160 mg resulted in a placebo-corrected improvement in neurological function that is equivalent to recovering approximately one to two years' of disease progression.

Across all patients, omaveloxolone did not improve peak work as measured during the maximal exercise test. The Company received reports from clinical sites that patients with *pes cavus* were not able to reach peak muscle exhaustion during the maximal exercise test due to pain associated with their foot deformity. In the subset of patients without the *pes cavus* foot deformity, across all doses omaveloxolone increased peak work by 6.5 watts relative to baseline ($p=0.002$) and by 4.3 watts versus placebo (non-significant). In these patients, omaveloxolone produced a

dose- and time-dependent increase in peak work, and the maximum effect on peak work was observed at the 160 mg dose level. At this dose, omaveloxolone increased peak work 11.5 watts from baseline (p=0.03) and 9.0 watts versus placebo (non-significant), which is a 13% relative improvement.

Colin Meyer, M.D., Chief Medical Officer of Reata, said, "We are very pleased that data from Part 1 of the MOXIe trial demonstrated clear evidence of biological activity that was associated with improvements in neurological function. Even though the study was not powered to demonstrate statistical separation from placebo, at the optimal dose, the mFARS results approached significance with only 12 active and 17 placebo patients and achieved significance in the subgroup that will be enriched in the confirmatory phase of the trial. We believe these results are quite meaningful since omaveloxolone-treated patients had substantial improvements in mFARS scores from baseline and versus placebo with only 12 weeks of treatment. The data suggest that omaveloxolone may not just slow disease progression but instead may promote the recovery of lost neurological function."

Safety and Tolerability

The trial was overseen by an independent data safety monitoring board (DSMB). No safety concerns were identified by the DSMB. Only two serious adverse events were reported, and both events occurred in placebo-treated patients. The most common adverse events in excess to placebo in the omaveloxolone group were upper respiratory tract infections and nasopharyngitis, which were generally mild in severity. Omaveloxolone was well-tolerated with only a single discontinuation in a 40 mg patient who developed a skin rash. One placebo patient discontinued prematurely due to withdrawal of consent.

Design of Part 2 of MOXIe

The Company plans to initiate Part 2 of the MOXIe trial during the second half of 2017. Part 2 of MOXIe will be a double-blind, randomized, placebo-controlled, multi-center, international trial designed to evaluate the safety and efficacy of omaveloxolone in patients with FA. The trial will enroll approximately 100 FA patients randomized evenly to either 150 mg of omaveloxolone or placebo. A 150 mg dose of omaveloxolone should produce similar systemic exposure while reducing the number of capsules per administration when compared to a 160 mg dose. Patients will be stratified by *pes cavus* status, and the proportion of *pes cavus* patients will be limited. Study drug will be administered orally, once daily for 24 weeks. The proposed primary endpoint of the study will be the change from baseline in mFARS of omaveloxolone compared to placebo at 24 weeks. A key secondary endpoint of the study will be the change from baseline in peak work during maximal exercise testing compared to placebo at 24 weeks. The study will also assess changes in the FA activities of daily living scale (ADL), the 25-foot timed walk test, the 9-hole peg test, the frequency of falls, and SF-36 scores.

In selecting mFARS as the primary endpoint for the study, the Company has worked closely with FARA and key opinion leaders in the FA community, who have strongly encouraged the use of the mFARS. mFARS was developed by clinical scientists in the FA community and FARA with input from the Division of Neurology Products (the Division) of the FDA from the Friedreich's Ataxia Rating Scale (FARS), a neurological exam with a validated scale developed for FA patients. The mFARS is designed to exclude elements of the FARS that are so-called "neurological signs" and, therefore, focuses only on the subset of elements that reflect patient function. Most important, FARA and the Collaborative Clinical Research Network in FA have conducted a large, longitudinal outcome measure and natural

history study in FA patients for the last 15 years (the FA Natural History Study) that demonstrates that changes in FARS and mFARS scores are highly correlated with patients' ability to perform activities of daily living and with disease progression.

During 2014, the Division provided the Company with guidance regarding the clinical endpoints and other design elements of MOXIe. The Division asked the Company to confirm that it planned to use the mFARS as an endpoint in MOXIe and stated that the mFARS would likely be considered an intermediate clinical endpoint that could support accelerated approval for drugs treating serious or life-threatening illnesses under Subpart H of the FDA act (Subpart H approval). Recently, the Company received a comment from the Division suggesting that the Company select an endpoint that measures a clinically meaningful drug benefit such as the FA ADL scale, rather than the mFARS. The statement made no reference to the Division's prior guidance regarding either use of the mFARS in MOXIe or use of mFARS as an intermediate endpoint that could support Subpart H approval. The Company believes that the FDA comment referring to clinical endpoints like the ADL was made to provide a path to full approval, rather than Subpart H approval. The FA Natural History Study data have established that patient reported ADL scores are highly variable and using them as a primary endpoint would require clinical studies to include thousands of patients to be adequately powered for efficacy. Since studies of that size are not feasible in FA, most registrational studies in FA to date have been conducted using mFARS as the primary endpoint. The Company is seeking clarification from the Division that it stands by its prior guidance that it considers the mFARS an intermediate endpoint that, depending on the trial results, could be appropriate to support approval under Subpart H.

Presentation of MOXIe Data

The complete data will be presented by Dr. David Lynch, Director of the Friedreich's Ataxia Program at Children's Hospital of Philadelphia, at 3:00pm EDT on June 2, 2017, after the completion of the Patient-Focused Drug Development meeting hosted by FARA. The presentation will be streamed online at the following link:

Safety, Efficacy, and Pharmacodynamics of Omav (Omaveloxolone) in Friedreich's Ataxia Patients (MOXIe Trial): Part 1 Results

Reata management will host a call at 4:00pm EDT during the afternoon of June 2, 2017, to discuss the results of Part 1 and to provide details on the timing and design of Part 2 of MOXIe. Slides for the event hosted by Reata will be available at 4:00pm EDT on our website at www.reatapharma.com. Dial-in and webcast information for this call is below:

Audience Dial-in:	(213) 358-0892
Audience Dial-in (toll-free):	(844) 348-3946
Audience Dial-in (international)	(213) 358-0892
Passcode:	33435422
Webcast Link:	http://edge.media-server.com/m/p/6r3b8jrz

About Friedreich's Ataxia

FA is a rare, degenerative, life-shortening neuro-muscular disorder that affects children and adults and involves the loss of strength and coordination usually leading to wheelchair use; diminished vision, hearing and speech; scoliosis

(curvature of the spine); increased risk of diabetes; and a life-threatening heart condition. Currently, there are no FDA-approved treatments for FA.

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 methyl and omaveloxolone, target the important transcription factor Nrf2 to restore mitochondrial function, reduce oxidative stress, and resolve inflammation.

Forward-Looking Statements

This press release includes certain disclosures which 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 are 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.

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