REATA ANNOUNCES NEW PRECLINICAL DATA DEMONSTRATING THE POTENTIAL OF OMAVELOXOLONE IN THE TREATMENT OF FRIEDREICH'S ATAXIA AND OTHER SEVERE NEUROLOGICAL DISEASES

IRVING, Texas—April 24th, 2018—Reata Pharmaceuticals, Inc. (Nasdaq:RETA) (Reata or Company), a clinical-stage biopharmaceutical company, today announced new preclinical data demonstrating that omaveloxolone potently activates the Keap1/Nrf2 pathway, significantly reduces production of reactive oxygen species, and improves mitochondrial function in two different models of severe neurological diseases. These results support the rationale for clinical studies of omaveloxolone in neurodegenerative and neuromuscular disorders, including the ongoing pivotal MOXIe trial in patients with Friedreich's ataxia.

In one study, omaveloxolone treatment dose-dependently increased the expression of key Nrf2 target genes, decreased LPS-induced expression of pro-inflammatory genes, and improved mitochondrial function in several cell lines including $hFXN^{154F}$ mouse embryonic fibroblasts expressing a mutant human frataxin gene. These data were presented by Christian Wigley, Ph.D., Reata's Vice President of Research, at the ongoing 2018 American Academy of Neurology Annual Meeting in Los Angeles, CA.

In a separate study, independent academic researchers from University College London, University of Dundee, Tohoku University, Johns Hopkins University, and University of Muenster studied the effect of omaveloxolone treatment in a rodent model of epileptogenesis and chronic seizures resulting from kainic acid-induced status epilepticus. Acute treatment with omaveloxolone significantly and dose-dependently increased glutathione and ATP levels, prevented neuronal loss, and reduced the median frequency of late spontaneous seizures by 94%. Additionally, *in vitro* results from a mixed cortical neuronal cell culture demonstrated that omaveloxolone treatment improved mitochondrial metabolism, reduced production of reactive oxygen species, and prevented neuronal cell death. These results were recently published in an original research article titled, "KEAP1 inhibition is neuroprotective and suppresses the development of epilepsy" in *Brain, a Journal of Neurology*. The article can be found online at the following link: https://doi.org/10.1093/brain/awy071.

"These studies add to the growing body of clinical and preclinical data demonstrating that omaveloxolone's mechanism of action has the potential to address a broad range of neurological and neuromuscular diseases," said Keith Ward, Ph.D., Reata's Chief Development Officer. "We are especially encouraged by the significant improvements in mitochondrial function and reduction in reactive oxygen species observed in these studies, as these are both known key drivers of pathogenesis in Friedreich's ataxia."

About Friedreich's Ataxia

Friedreich's ataxia is a rare, degenerative, life-shortening neuromuscular 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 Friedreich's ataxia.

About Omaveloxolone

Omaveloxolone 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 omaveloxolone for the treatment of Friedreich's ataxia.

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 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.

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