Eagle Pharmaceuticals Announces Positive Results of Study Conducted to Evaluate Neuroprotective Effects of RYANODEX Secondary to Nerve Agent Exposure

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Eagle Pharmaceuticals, Inc. (Nasdaq: EGRX) ("Eagle" or "the Company") today announced positive results of an initial study in over 50 rodents to evaluate the neuroprotective effects of RYANODEX® (dantrolene sodium) in an established rodent model of Nerve Agent-(NA) induced seizures and seizure-related brain damage.

Scientific evidence indicates that elevated intracellular calcium levels may have a role in seizure-related brain damage resulting from induced seizures and status epilepticus secondary to NA exposure. As in other conditions, including acute hyperthermic and hypermetabolic disorders, intracellular calcium overload leads to severe brain and other organ damage. RYANODEX (dantrolene sodium) is a well-characterized ryanodine receptor antagonist that inhibits intracellular calcium overload secondary to different triggers. Ryanodine receptors are widely distributed in the body, including skeletal muscle, heart and brain tissues. The active ingredient in RYANODEX is the only approved drug that inhibits the ryanodine receptors, modulating the intracellular calcium levels.

Animals treated with RYANODEX in combination with nerve gas antidotes and anti-epileptic drugs (AEDs) after exposure to NAs had better performance in neurobehavioral testing compared to animals treated with AEDs alone, as well as substantially less brain damage.

"We believe these study data support further investigation of RYANODEX as a neuroprotective therapy in Nerve Agent casualties, a potential new indication for the drug," said Scott Tarriff, Chief Executive Officer of Eagle. "Our next steps are to continue to engage with the military and meet with the FDA. We will continue to develop our animal research with the expectation that indications like these are approved under the Animal Rule[1]."

In this study, animals were exposed to a high dose of the NA soman and treated with the known antidote for acute poisoning (atropine and HI-6). All surviving study animals developed severe status epilepticus and were treated with standard AEDs according to protocol. Study animals were randomly assigned to receive RYANODEX or control vehicle as added treatment.

Neurobehavioral testing (NBT) was conducted between two and four weeks after exposure to soman, at which time brain neuropathology (NP) was also evaluated. NBT included Forced Swim Test and Sucrose Preference Test.

Animals treated with RYANODEX + AEDs had better performance in NBT, compared to animals treated with AEDs only. Analysis of NP showed a substantially lower level of brain cell necrosis in animals treated with RYANODEX + AEDs, compared to AEDs alone. Animals treated with standard therapy showed a mean necrosis score of 2.6 in fronto-parietal cortex, compared to a group of RYANODEX treated animals showing a score of 0.6 in the same anatomical region. The scoring system for cell necrosis ranges between 0 (normal, no necrosis) to 5 (cellular necrosis greater than 80%) In addition, no safety issues were observed in animals treated with RYANODEX.

About Nerve Agents

NAs were first widely used in World War II (WWII); their impact became a significant public health issue thereafter. NAs acquired their name because they affect the transmission of nerve impulses in the nervous system. NA include compounds such as sarin, VX and soman.

NAs, whether gas, aerosol or liquid, are extremely toxic and have a very rapid effect. The NA enters the body through inhalation or through the skin. Poisoning may also occur through consumption of liquids or foods contaminated with NAs. NA survivors will likely experience severe symptoms, including neurologic consequences.

NAs produce seizures and seizure-related brain damage. The seizures quickly develop into status epilepticus and usually become refractory to standard antiepileptic therapy.

Medical care for NA casualties will likely be delayed beyond the therapeutic window of opportunity to terminate NA-induced seizures, and seizure-related brain damage will continue along the pathological cascade. Thus, there is a need for adjunct drug therapy that is capable of interrupting the pathologic cascade and augmenting neuroprotection when administered in combination with antiepileptic drugs during the refractory phase of NA-induced seizures.

Scientific evidence supports a pivotal role of elevated intracellular calcium levels in seizure-related brain damage resulting from induced seizures and status epilepticus secondary to NA exposure. In addition, there are several reports that a neuroprotective approach, aimed at attenuating delayed calcium overload, combined with antiepileptic treatment, may lead to greater protection against seizure-related brain damage than anti-epileptics alone.

About Eagle Pharmaceuticals, Inc.

Eagle is a specialty pharmaceutical company focused on developing and commercializing injectable products that address the shortcomings, as identified by physicians, pharmacists and other stakeholders, of existing commercially successful injectable products. Eagle's strategy is to utilize the FDA's 505(b)(2) regulatory pathway. Additional information is available on the Company's website at www.eagleus.com.

Forward-Looking Statements

This press release contains forward-looking information within the meaning of the Private Securities Litigation Reform Act of 1995, as amended and other securities laws. Forward-looking statements are statements that are not historical facts. Words such as "will," "may," "can," "could be," "believe," "intends," "anticipate(s)," "plan," "enables," "potentially," "entitles," and similar expressions are intended to identify forward-looking statements. These statements include statements regarding future events including, but not limited to: the ability of Eagle to generate data sufficient to support the grant

of an indication from the FDA for the use of Ryanodex as a neuroprotective therapy in treating the effects of NA exposure; the willingness of the FDA to grant approval for such an indication; and the ability of the Company to develop and sustain a market for such indication; and other factors that are discussed in Eagle's Annual Report on Form 10-K for the year ended December 31, 2016, and its other filings with the U.S. Securities and Exchange Commission ("SEC"). All of such statements are subject to certain risks and uncertainties, many of which are difficult to predict and generally beyond Eagle's control, that could cause actual results to differ materially from those expressed in, or implied or projected by, the forward-looking information and statements. Such risks include, but are not limited to whether Eagle's management and/or board of directors will be effective in managing Eagle's business and future growth, whether Eagle will maintain successful compliance with FDA and other governmental regulations applicable to manufacturing facilities, products and/or businesses, as well as the other risks described in Eagle's filings with the SEC. Readers are cautioned not to place undue reliance on these forward-looking statements that speak only as of the date hereof, and we do not undertake any obligation to revise and disseminate forward-looking statements to reflect events or circumstances after the date hereof, or to reflect the occurrence of or non-occurrence of any events.

[1] FDA Guidance for Industry 'Product Development Under the Animal Rule' (October 2015).
https://www.fda.gov/downloads/Drugs/GuidanceComplianceRegulatoryInformation/Guidances/UCM399217.pdf

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