



News Release

FOR IMMEDIATE RELEASE

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SAFETY BENEFITS OF ARIAD REGULATED ERYTHROPOIETIN THERAPY TO TREAT ANEMIA PRESENTED AT HEMATOLOGY MEETING

Cambridge, MA, December 7, 2001 – ARIAD Pharmaceuticals, Inc. (Nasdaq: ARIA) today announced results of preclinical studies comparing the safety profile of ARIAD's regulated erythropoietin (Epo) product candidate with that of an uncontrolled version of Epo therapy in animal models. Severe anemia resulting from the genetic blood disease, beta-thalassemia, was effectively and safely treated using ARIAD's product candidate. In contrast, all animals receiving an uncontrolled version of Epo therapy died within two months due to excessive levels of Epo and extremely high numbers of red blood cells.

The study being presented at the American Society of Hematology annual meeting by scientists from the University of Pennsylvania and ARIAD underscores the importance of dose-dependent regulation of protein therapy to achieve therapeutic benefit without life-threatening toxicity.

"The safety of new medicines always is of paramount concern," said Harvey J. Berger, M.D., chairman and chief executive officer of ARIAD. "The results announced today convincingly demonstrate the critically important safety benefit, as well as therapeutic effect, of our lead product candidate for anemia."

The abstract of the presentation by Johnston *et al* (5431), "Treatment of beta-thalassemia in the mouse by regulated expression of AAV-encoded erythropoietin," is available online at the ASH meeting website (www.abstracts-on-line.com/abstracts/hem/).

ARIAD is engaged in the discovery and development of breakthrough medicines that regulate cell signaling with small molecules. The Company's lead product candidates – treatments for bone metastases and bone pain, osteoporosis, cancer, anemia and graft-vs-host disease following T cell immunotherapy – all were developed through the integration of genomics, proteomics and structure-based drug design. ARIAD's RegTech cell-signaling regulation technologies are being used by almost 500 academic investigators providing a robust source of potential new technologies, drug targets and

product candidates that the Company may develop. ARIAD also has an exclusive license to pioneering technology related to the discovery and development of drugs that modulate the cellular protein, NF- κ B, and its associated pathways, which regulate the transcription of key genes involved in many major diseases. Additional information about ARIAD can be found on the web at www.ariad.com.

Some of the matters discussed herein are forward-looking statements. Such statements are identified by the use of words such as “anticipate,” “estimate,” “expect,” “project,” “intend,” “plan,” “believe,” and other words and terms of similar meaning in connection with any discussion of future operating or financial performance. Such statements are based on management’s current expectations and are subject to certain factors, risks and uncertainties that may cause actual results, events and performance to differ materially from those referred to or implied in such statements. These risks include, but are not limited to, risks and uncertainties regarding the Company’s preclinical studies, the Company’s ability to conduct clinical trials of its product candidates and the results of such trials, as well as risks and uncertainties relating to economic conditions, markets, products, competition, intellectual property, services and prices, key employees, future capital needs, dependence on the Company’s collaborators and other factors. These risks are identified in ARIAD’s Annual Report on Form 10-K for the fiscal year ended December 31, 2000, filed with the Securities and Exchange Commission. The information contained in this document is believed to be current as of the date of original issue. The Company does not intend to update any of the forward-looking statements after the date of this document to conform these statements to actual results or to changes in the Company’s expectations, except as required by law.

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