Idera Announces Publication of New Data Demonstrating Impact of Inhibiting TLRs 7, 8, and 9 in a Preclinical Model of Autoimmune Disease

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CAMBRIDGE, Mass.--(BUSINESS WIRE)--Jan. 22, 2014-- Idera Pharmaceuticals, Inc. (Nasdaq: IDRA), a clinical stage biopharmaceutical company developing a novel therapeutic approach for the treatment of autoimmune diseases and genetically defined forms of B-cell lymphoma, today announced the publication of a study supporting the potential role of the suppression of Toll-like-receptors (TLRs) 7, 8, and 9 in the treatment of psoriasis. The data were published in the scientific journal PLOS ONE.

In the publication, entitled *Suppression of Molecular Inflammatory Pathways by Toll-Like Receptor 7, 8, and 9 Antagonists in a Model of IL-23-Induced Skin Inflammation*, data are presented from a study of an antagonist of TLRs 7 and 9 and an antagonist of TLRs 7, 8, and 9. The IL-23-induced mouse model of skin inflammation was chosen due to its histological and molecular resemblance to human psoriasis, including the involvement of the IL-17 inflammatory pathway. Gene expression analyses showed that treatment with either antagonist normalized expression of IL-17-induced genes. Additionally, both antagonists normalized aberrant expression of keratin 16, an indicator of epidermal hyperplasia. More of the IL-23 regulated genes were modulated with the antagonist of TLRs 7, 8, and 9 (36%) than with the antagonist of TLRs 7, and 9 (26%). In addition to IL-17, other inflammatory pathways, including IL-6 and interferon-gamma, were strongly suppressed by both antagonists. Further analysis showed that the antagonist of TLRs 7, 8, and 9 down-regulated the JAK-STAT, IL-23, IL-12, and IL-17 canonical pathways. The results suggest that IL-23-driven inflammation in mouse skin may be dependent on signaling mediated by TLRs 7, 8, and 9.

“These results indicate that TLRs 7, 8 and 9 could serve as novel therapeutic targets in psoriasis vulgaris and other disease with similar pathophysiology,” stated James G. Krueger, M.D., Ph.D., Head of the Laboratory for Investigative Dermatology at The Rockefeller University and senior study author.

“These data provide further insight into the mechanisms underlying the therapeutic effect which we have reported previously in our TLR antagonist clinical program in psoriasis. Currently, we are conducting a Phase 2 clinical trial of IMO-8400, an antagonist of TLRs 7, 8, and 9, for the treatment of patients with moderate-to-severe plaque psoriasis and plan to initiate clinical development in selected orphan autoimmune indications,” said Robert D. Arbeit, M.D., Vice President of Clinical Development at Idera.

Authors of the study were Mayte Suárez-Fariñas, Ph.D, Francesca S. Ortenzio, and James G. Krueger, M.D., Ph.D., from the Laboratory for Investigative Dermatology at The Rockefeller University; and Robert Arbeit, M.D., and Tim Sullivan, Ph.D., from Idera Pharmaceuticals. The publication can be found by visiting PLOS ONE.

About the IMO-8400 Phase 2 trial in moderate-to-severe plaque psoriasis

In September 2013, Idera completed enrollment of the 32 patients initially planned in the Company’s ongoing randomized, double-blind, placebo-controlled Phase 2 trial of IMO-8400 in patients with moderate-to-severe plaque psoriasis. These 32 patients were randomized for treatment at three dose levels of IMO-8400, 0.075 mg/kg, 0.15 mg/kg and 0.3 mg/kg, or placebo. The data remain blinded as the follow-up period of the trial continues. All treatments were well tolerated in the trial, and based on the observed safety profile, the Company expanded the trial to evaluate a higher dose cohort of 0.6 mg/kg and placebo in up to 12 patients.. The Company expects to report top-line data from the trial in the first half of 2014.

About Idera Pharmaceuticals, Inc.

Idera's technology platform involves creating novel synthetic RNA- and DNA-based compounds to modulate immune responses. Idera has applied this platform to develop proprietary Toll-like receptor (TLR) antagonists as immunomodulatory drug candidates. Toll-like receptor antagonists block the over-activation of immune factors which can cause a range of pathological effects. Idera is conducting clinical development of TLR antagonists in autoimmune and inflammatory diseases, and for use in B-cell lymphomas harboring the MYD88 L265P mutation. More information on Idera is available at www.iderapharma.com.

Forward Looking Statements

This press release includes statements concerning Idera Pharmaceuticals, Inc. and its future expectations, plans and prospects that constitute forward-looking statements within the meaning of The Private Securities Litigation Reform Act of 1995 and that involve a number of risks and uncertainties. For this purpose, any statements contained herein that are not statements of historical fact may be deemed to be forward-looking statements. Without limiting the foregoing, the words "believes," "anticipates," "plans," "expects," "estimates," "intends," "should," "could," "will," "may," and similar expressions are intended to identify forward-looking statements. There are a number of important factors that could cause Idera's actual results to differ materially from those indicated by such forward-looking statements, including whether results obtained in early research, preclinical studies and clinical trials will be indicative of the results that will be generated in future preclinical and clinical studies; whether products based on Idera's technology will advance into or through the clinical trial process on a timely basis or at all and receive approval from the FDA or equivalent foreign regulatory agencies; whether, if the Company's products receive approval, they will be successfully distributed and marketed; and such other important factors as are set forth under the caption "Risk Factors" in Idera's Quarterly Report on Form 10-Q for the period ended September 30, 2013, which important factors are incorporated herein by reference. Idera disclaims any intention or obligation to update any forward-looking statements.

Source: Idera Pharmaceuticals, Inc.

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