Idera Pharmaceuticals Presents Data from a Multiple-Dose Phase 1 Clinical Trial of IMO-3100, Lead Drug Candidate for the Treatment of Autoimmune Diseases

April 4, 2011 1:03 PM EDT

- Suppression of multiple cytokines was maintained during the treatment period -

CAMBRIDGE, Mass., Apr 04, 2011 (BUSINESS WIRE) --

Idera Pharmaceuticals, Inc. (Nasdaq: IDRA) today announced the presentation of data from its multiple-dose Phase 1 clinical trial of IMO-3100 in healthy subjects. The trial was designed to evaluate the safety and mechanism of action of IMO-3100, a Toll-like Receptor (TLR) 7 and TLR9 dual antagonist that is Idera's lead drug candidate in development to treat autoimmune and inflammatory diseases. In the trial, IMO-3100 was well tolerated over the four weeks of treatment. In addition, target engagement of TLR7 and TLR9 was observed, confirming the intended mechanism of action of IMO-3100. The presentation (Abstract #333) entitled "IMO-3100, a novel toll-like receptor antagonist for autoimmune and inflammatory diseases: safety and pharmacodynamics in a multiple-dose Phase 1 clinical trial" is being made today at the Keystone Symposia meeting "Immunoregulatory Networks" being held in Breckenridge, Colorado from April 1-6, 2011.

"We have completed two Phase 1 clinical trials of IMO-3100 involving a total of 60 healthy subjects. The results of this multiple-dose trial are consistent with those from our earlier single-dose study, with demonstration of a good safety profile and evidence of the intended mechanism of action," said Robert Arbeit, M.D., Vice President of Clinical Development of Idera Pharmaceuticals. "The Phase 1 data support our plans to evaluate IMO-3100 in a Phase 2 clinical trial in a selected autoimmune disease indication."

"One of our objectives in the Phase 1 clinical development program was to establish that IMO-3100 engages TLR7 and TLR9, its intended mechanism of action. We are very pleased that in both trials IMO-3100 inhibited TLR7- and TLR9-mediated immune responses, and that in this study suppression of key cytokines such as IL-6 and IFN-a was maintained throughout the four-week treatment period," said Tim Sullivan, Ph.D., Vice President of Development Programs and Alliance Management at Idera. "We are currently conducting nonclinical studies of IMO-3100 which we expect to complete during the first half of 2011 to enable submission of a Phase 2 clinical trial protocol during the third quarter of this year."

"Our scientific rationale for the use of IMO-3100 to treat autoimmune and inflammatory diseases is to suppress immune responses that are mediated through TLR7 or TLR9 and thereby block the induction of multiple cytokines, in contrast to blocking the activity of individual cytokines as do many current treatments of autoimmune diseases," commented Sudhir Agrawal, D.Phil., Chairman and Chief Executive Officer of Idera. "IMO-3100, as a dual antagonist of TLR7 and TLR9, has been shown in these Phase 1 clinical trials to block the induction of multiple cytokines and may provide a novel approach for the treatment of autoimmune and inflammatory diseases."

Phase 1 Multiple-Dose Clinical Trial of IMO-3100 in Healthy Subjects

Study Design

In this trial, 24 healthy subjects were recruited and randomized into three treatment groups each of which received two subcutaneous injections per week for four weeks. The three groups, each comprising eight subjects, were (a) IMO-3100 at 0.64 mg/kg once per week and placebo once per week, (b) IMO-3100 at 0.32 mg/kg twice per week; and (c) placebo twice per week. The primary objective of this study was the evaluation of safety and tolerability. Secondary objectives were to assess IMO-3100 pharmacokinetics and pharmacodynamic mechanism of action. Study visits were performed twice weekly during the four-week treatment period, at Day 29 (End-of-Treatment), at Day 36 (interim follow-up) and at Day 59 (End-of-Study). For pharmacokinetic evaluation, plasma levels of IMO-3100 were measured through 24 hours after the first dose and after the seventh of the twice-weekly doses. For pharmacodynamic evaluation, blood samples were collected pre-dose on Days 1, 4, 8, 15, 22, and 25, and at the visits on Days 29 and 36. To assess the effect of IMO-3100 treatment, peripheral blood mononuclear cells (PBMCs) were isolated from these samples, stimulated with TLR7- and TLR9-agonists and the resulting induced cytokines assayed.

Study Results

- **Safety.** IMO-3100 was well tolerated in both treatment regimens. There were no serious adverse events and no treatment discontinuations. Mild injection site reactions were the most common adverse events.
- **Pharmacokinetics.** The pharmacokinetics of IMO-3100 were found to be dose-proportional and showed no evidence of accumulation after repeated dosing.
- **Pharmacodynamics mechanism of action.** Suppression of multiple cytokines including IFN-a, IL-6, MIP-1B, and IL-1Ra, mediated through TLR7 and TLR9 was observed in both IMO-3100 groups, when post-dose responses were compared to pre-dose responses in each subject.

Suppression of multiple cytokines was maintained in IMO-3100-treated subjects throughout the four-week treatment period, based on responses measured from the third day after the first dose through four or more days after the last dose.

No consistent suppression of any cytokines was observed in placebo-treated subjects.

These results provide evidence that IMO-3100 suppressed TLR7- and TLR9-mediated immune responses in the trial.

Authors of the presentation are Tim Sullivan, Ph.D., Lakshmi Bhagat, Ph.D., Wayne Jiang, M.D., Ph.D., Jessica Murphy, Natasha Ostavnenko, Melissa Precopio, Ph.D., Mallikarjuna Putta, Ph.D., Ekambar Kandimalla, Ph.D., and Robert Arbeit, M.D., of Idera Pharmaceuticals and George Atiee, M.D., of ICON Development Solutions, San Antonio, TX.

About IMO-3100

IMO-3100, a dual antagonist of TLR7 and TLR9, is a lead drug candidate in development to treat autoimmune and inflammatory diseases. Independent research studies suggest that pro-inflammatory cytokines characteristic of autoimmune disease are induced through activation of TLR7 and TLR9. IMO-3100 is designed to block production of multiple pro-inflammatory cytokines induced through TLR7 and TLR9. In contrast, many current autoimmune disease treatments aim to block the activity of individual cytokines. IMO-3100 has demonstrated potent activity in reducing pathologic and immunologic manifestations in preclinical mouse models of diseases such as lupus, rheumatoid arthritis, psoriasis and hyperlipidemia. Phase 1 clinical trials of IMO-3100, including an escalating single-dose study and a multiple-dose study, have been conducted in healthy subjects.

About Idera Pharmaceuticals, Inc.

Idera Pharmaceuticals develops drug candidates to treat chronic hepatitis C virus infection, autoimmune and inflammatory diseases, cancer, and respiratory diseases, and for use as vaccine adjuvants. The company's proprietary drug candidates are designed to modulate specific Toll-like Receptors, which are a family of immune system receptors. Idera's pioneering DNA and RNA chemistry expertise enables us to create drug candidates for internal development and generates opportunities for multiple collaborative alliances. For more information, visit www.iderapharma.com.

Idera Forward-Looking Statements

This press release contains forward-looking statements concerning Idera Pharmaceuticals, Inc. 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 preclinical studies and early clinical trials such as the studies and trials referred to in this release will be indicative of results obtained in future clinical trials; 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 United States Food and Drug Administration or equivalent foreign regulatory agencies; whether, if the Company's products receive approval, they will be successfully distributed and marketed; whether the Company's collaborations will be successful; whether the patents and patent applications owned or licensed by the Company will protect the Company's technology and prevent others from infringing it; whether Idera's cash resources will be sufficient to fund the Company's operations; and such other important factors as are set forth under the caption "Risk Factors" in Idera's Annual Report on Form 10-K for the year ended December 31, 2010 which important factors are incorporated herein by reference. Idera disclaims any intention or obligation to update any forward-looking statements.

SOURCE: Idera Pharmaceuticals

Idera Pharmaceuticals, Inc.
Teri Dahlman, 617-679-5519
tdahlman@iderapharma.com
or
MacDougall Biomedical Communications
Chris Erdman, 781-235-3060
cerdman@macbiocom.com