May 15, 2014

Xencor Presents Study Comparing XmAb7195 to Omalizumab at the American Thoracic Society 2014 International Conference

Rapid IgE clearance by XmAb7195 shows potential as a disease modifying treatment for allergy and asthma

MONROVIA, Calif., May 15, 2014 /PRNewswire/ -- Xencor, Inc. (NASDAQ: XNCR), a clinical-stage biopharmaceutical company developing engineered monoclonal antibodies for the treatment of autoimmune diseases, asthma and allergic diseases, and cancer, today announced it will present preclinical data comparing activity of XmAb®7195 to omalizumab in reducing IgE, a protein responsible for triggering disease activity in asthma and other allergic disorders. The study describes XmAb7195's three distinct mechanisms of action, including a novel mechanism for rapid clearance of IgE. The study supports future studies in human clinical trials. Data will be presented Tuesday May 20 at 8:15 a.m. PDT at the American Thoracic Society (ATS) 2014 International Conference (Poster A4261: Poster Board Number A97).

"Omalizumab has validated IgE inhibition as a strategy to reduce asthma symptoms and disability, however it only reaches a portion of its market potential due to suboptimal potency," said Bassil Dahiyat, Ph.D., President and CEO of Xencor. "The novel mechanisms by which XmAb7195 suppresses IgE levels represents a promising approach for overcoming the limitations of existing therapy."

XmAb7195 targets IgE with its variable domain, and uses Xencor's XmAb® immune inhibitor Fc domain to target FcγRIIb, resulting in three distinct mechanism of action for reducing IgE levels. First, XmAb7195 sequesters free IgE and prevents activation of mast cells and basophils, the mediators of allergic inflammation and pathology. Second, it prevents IgE production by suppressing IgE-positive B-cell activation and differentiation into IgE-secreting plasma cells. Third, Xencor has discovered a new mechanism of action whereby high FcγRIIb binding causes extremely rapid clearance of the complexes formed between XmAb7195 and IgE, resulting in rapid and marked reductions of the total IgE and free IgE in circulation.

The results of the study found that after a single 5 mg/kg intravenous dose of XmAb7195 or omalizumab in chimpanzees, XmAb7195 reduced free IgE to at least 10-fold lower levels than omalizumab. One hour after omalizumab treatment, free IgE dropped to ~50 ng/mL and then rebounded. XmAb7195 immediately reduced free IgE levels to below quantifiable levels, until day 10. Similar to its effects in humans, omalizumab increased total (sequestered plus free) IgE. In contrast, XmAb7195 rapidly depleted total IgE below quantifiable levels within 1 hour.

The full abstract by Moore, et al, titled, "*Accelerated Clearance Of IgE In Chimpanzees Is Mediated By XmAb7195, An Fc-Engineered Antibody With Enhanced Affinity For Inhibitory Receptor FcγRIIb*" [Poster Board # A97) **,** is available on the ATS 2014 website at: https://cms.psav.com/cPaper2012/myitinerary/publication-53966.html?congress=ats2014

The poster will also be available on Xencor's website following its presentation at ATS 2014.

About XmAb® Antibody Engineering Technology

In contrast to conventional approaches to antibody design that focus on the Fv domain responsible for binding to target cells, Xencor's XmAb® antibody engineering technology focuses on the Fc domain, the portion of the antibody that interacts with multiple segments of the immune system. Xencor's XmAb® Fc domains have shown an ability in preclinical and clinical studies to enhance antibody performance while typically maintaining over 99.5% identity in structure and sequence to natural antibodies. This design allows our engineered antibodies to retain the beneficial stability, pharmacokinetics and ease of discovery of natural antibodies, while utilizing validated methods for antibody manufacturing.

About Xencor, Inc.

Xencor is a clinical-stage biopharmaceutical company developing engineered monoclonal antibodies for the treatment of autoimmune diseases, asthma and allergic diseases, and cancer. Currently, six candidates are in clinical development internally and with partners that have been engineered with Xencor's XmAb® technology. Xencor's internally-discovered programs include XmAb5871, in Phase 1b/2a clinical trials for the treatment of Rheumatoid arthritis and lupus, XmAb7195 in preclinical development for the treatment of asthma, and XmAb5574/MOR208 which has been licensed to Morphosys AG and is in Phase 2 clinical trials for the treatment of acute lymphoblastic leukemia and non-Hodgkin lymphoma. Xencor's XmAb antibody engineering technology enables small changes to the structure of monoclonal antibodies resulting in new mechanisms of

therapeutic action. Xencor partners include Amgen, Merck, Janssen R&D LLC, Alexion and Boehringer Ingelheim.

For more information, please visit www.xencor.com.

Forward Looking Statements

Statements contained herein regarding matters that are not historical facts are "forward-looking statements" within the meaning of the U.S. securities laws, including statements associated with Xencor's research and its expectations regarding future therapeutic and commercial potential of Xencor's technologies, programs, drug candidates and intellectual property related to Xencor's XmAb technology. Because such statements are subject to risks and uncertainties, including risks associated with the process of discovering, developing and commercializing drugs that are safe and effective, actual results and the timing of events may differ materially from those expressed or implied by such forward-looking statements. These and other risks concerning Xencor's programs and technology are described in additional detail in Xencor's SEC filings. These forward-looking statements speak as of the date on which they were made, are based upon Xencor's current expectations and involve assumptions that may never materialize or may prove to be incorrect. Xencor disclaims any intention or obligation to update such statements to reflect events that occur or circumstances that exist after the date on which they were made.

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