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XOMA Presents Positive Phase 1 XOMA 358 Data at the ENDO Meeting

- Results show XOMA 358 reduces insulin receptor signaling and increased glucose production after exogenous insulin injection

- XOMA 358 shows potential for treatment of hyperinsulinemic hypoglycemia conditions

BERKELEY, Calif., March 7, 2015 (GLOBE NEWSWIRE) -- XOMA Corporation (Nasdaq:XOMA), a leader in the discovery and development of therapeutic antibodies, announced the presentation of data from XOMA 358, the lead antibody in the Company's XMet D program, today, at the Endocrine Society's Annual Meeting – ENDO 2015. Paul Rubin, M.D., Senior Vice President, Research and Development, and Chief Medical Officer at XOMA, presented the Company's data, titled "*XOMA 358, a Novel Treatment for Hyperinsulinemic Hypoglycemia: Safety and Clinical Pharmacology from the First in Human Trial*".

"The XOMA 358 Phase 1 data is the first to show that a monoclonal antibody can down-regulate the insulin receptor and its downstream signaling. Insulin is the primary hormone in blood glucose regulation, and there are several rare diseases where the body produces inappropriately high levels of insulin, causing dangerously low blood sugars. This condition, which can lead to brain damage or, in rare cases, death, often requires extensive therapy including continuous ingestion of glucose or even pancreatectomy," stated Dr. Rubin. "Our results suggest XOMA 358 is a first-in-class molecule active against the insulin receptor and shows potential for treatment of patients who experience this endogenous over-production of insulin."

The Phase 1 first-in-human study enrolled 19 healthy volunteer subjects; 14 received XOMA 358 and 5 received placebo. Individual cohorts were administered XOMA 358 doses of 0.1, 0.3, 1, and 3 mg/kg or placebo, with dose escalation based on review of safety and pharmacokinetic data from each cohort. Changes in insulin and glucose were monitored through the use of Mixed meal tests (MMTs) and insulin tolerance tests (ITT). Dose-related increases in post-prandial (after eating a meal) glucose levels, consistent with down-regulation of insulin signaling, were observed through Day 6 following drug infusion, with the Day 3 glucose AUC nearly 80% greater than placebo at the 1 mg/kg dose level. Fasting HOMA-IR values, a measure of reduced insulin signaling, were likewise elevated in a dose-dependent manner and, at peak time points, were several fold over baseline.

Subjects who were given a single administration of XOMA 358 at 3 mg/kg were infused with insulin before and 2, 3 and 5 days after dosing. Prior to administration of the drug, as expected, the infusion caused a significant lowering of blood sugar. This lowering of blood sugar was significantly blunted at all subsequent time points tested.

These preliminary data provide a strong rationale for continued study of XOMA 358 in adults and children with hypoglycemia due to overproduction of insulin. XOMA 358 appeared to be well-tolerated; there were no serious adverse events.

About XOMA 358

Insulin is the major hormone for lowering blood glucose levels. Abnormal increases in insulin secretion can lead to profound hypoglycemia (low blood sugar), a state that can result in significant morbidities including cerebral damage and epilepsy. In some instances, profound hypoglycemia can be fatal. XOMA 358 is a fully human allosteric modulating monoclonal antibody that binds to insulin receptors and attenuates insulin action. XOMA 358 is being investigated as a novel treatment for non-drug-induced, endogenous hyperinsulinemic hypoglycemia (low blood glucose caused by excessive insulin production) and other related disorders. A therapy that safely and effectively mitigates insulin-induced hypoglycemia has the potential to address a significant unmet therapeutic need for certain rare medical conditions associated with hyperinsulinism.

About XOMA Corporation

XOMA's innovative product candidates are the result of the Company's expertise in developing ground-breaking monoclonal antibodies, including allosteric modulating antibodies, which have created new opportunities to potentially treat a wide range of human diseases. XOMA is developing its lead product gevokizumab (IL-1 beta modulating antibody) with SERVIER through a global Phase 3 program for Behçet's disease uveitis, non-infectious uveitis and pyoderma gangrenosum. XOMA's scientific research also produced the XMet program, which consists of three classes Selective Insulin Receptor Modulators (SIRMs) antibodies. XOMA 358, the lead antibody in the XMetD program, is an allosteric modulating monoclonal antibody that reduces both the binding of insulin to its receptor and down-regulates insulin signaling, and could have a major effect on the treatment of abnormal metabolic states.

More detailed information can be found at www.xoma.com.

Forward-Looking Statements

Certain statements contained in this press release including, but not limited to, statements related to anticipated timing of initiation and completion of clinical trials, regulatory approval of unapproved product candidates, the anticipated success of any product launch, or statements that otherwise relate to future periods are forward-looking statements within the meaning of Section 27A of the Securities Act of 1933 and Section 21E of the Securities Exchange Act of 1934. These statements are based on assumptions that may not prove accurate, and actual results could differ materially from those anticipated due to certain risks inherent in the biotechnology industry and for companies engaged in the development of new products in a regulated market. Potential risks to XOMA meeting these expectations are described in more detail in XOMA's most recent filing on Form 10-K and in other SEC filings. Consider such risks carefully when considering XOMA's prospects. Any forward-looking statement in this press release represents XOMA's views only as of the date of this press release and should not be relied upon as representing its views as of any subsequent date. XOMA disclaims any obligation to update any forward-looking statement, except as required by applicable law.

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