NEWS RELEASE

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Lycera Achieves First Milestone in Merck Research Collaboration

Partnership Focused on Developing Drugs Targeting the Th17 Pathway through the Inhibition of RORγt to Treat Autoimmune Diseases

ANN ARBOR, Mich. – December 21, 2011 – Lycera Corp., a biopharmaceutical company pioneering innovative approaches to developing novel oral medicines to treat autoimmune diseases, today announced that it has reached the first milestone under its research collaboration with Merck, triggering a milestone payment. The collaboration is focused on developing drug candidates that have the potential to treat major autoimmune diseases such as rheumatoid arthritis, psoriasis, inflammatory bowel disease and multiple sclerosis.

In March, the companies announced an exclusive research collaboration to discover, develop and commercialize small molecules that target T-helper 17 (Th17) cells, key mediators of inflammation. Lycera received the first $12 million in upfront cash payments at the commencement of the collaboration. Additionally, Lycera is entitled to royalty payments and sales milestones on global sales, and has a profit share option in the U.S., to all products that are developed as a result of the collaboration.

"We are delighted to have met this important research milestone in our collaboration with Merck in less than a year from signing the deal," said Kathleen Metters, Ph.D., president and chief executive officer of Lycera. "This significant achievement reinforces the strength of our joint partnership and demonstrates the team's rapid progress in moving this program forward to develop novel, oral medicines for the treatment of autoimmune diseases."

Th17 cells are characterized by the production of interleukin-17 (IL-17), a highly inflammatory cytokine that plays an important role in the pathogenesis of immune-mediated diseases, including psoriasis, rheumatoid arthritis, multiple sclerosis, inflammatory bowel disease (colitis) and asthma. RORγt is the key transcription factor that orchestrates the differentiation of Th17 cells, inducing transcription of the genes encoding IL-17. Mice with RORγt deficient T cells have attenuated disease and lack tissue-infiltrating Th17 cells. Thus, RORγt is a key regulator of immune homeostasis and is a potential therapeutic target for immune diseases. Lycera has developed a proprietary program that targets Th17 cells and has identified novel, potent and specific inhibitors of RORγt that reduce IL-17 production in primary cells and *in vivo*.

About Autoimmune Diseases
Serious autoimmune diseases are a major and growing public health problem. An autoimmune disorder is a condition that occurs when the immune system mistakenly attacks and destroys healthy body tissue. There are more than 80 different types of autoimmune disorders[1], and approximately 50 million Americans, or one in five people, suffer from autoimmune diseases[2].

About Lycera
Lycera Corp. is focused on the discovery and development of selective, small-molecule immunomodulators for the treatment of patients with autoimmune diseases such as rheumatoid arthritis, psoriasis and inflammatory bowel disease. The company is developing drug candidates that target two novel therapeutic pathways and have the potential for first-in-class oral efficacy without the adverse effects of current standard-of-care antiproliferative and immunosuppressive agents. Lycera is focused on the emerging area of cellular bioenergetics to selectively target and silence pathologically activated cells. The company also has a program targeting the Th17 pathway through the inhibition of RORγt that is the basis of an exclusive research collaboration with Merck. Lycera's leadership team and advisors represent the core thought leaders in immunology, inflammation, organ transplantation and kinase biology and are responsible for key advances and discoveries in these fields. Visit www.lycera.com for more information.

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1 Medline Plus, http://www.nlm.nih.gov/medlineplus/ency/article/000816.htm.
2 American Autoimmune Related Diseases Association, http://www.aarda.org/q_and_a.php.