AMICUS THERAPEUTICS PRESENTS PRECLINICAL DATA FROM STUDIES OF PLICERA

Studies Show Increased Enzyme Levels in Two Most Common Mutations

Cranbury, NJ, March 20, 2007 – Amicus Therapeutics, a biopharmaceutical company developing small molecule, orally-administered pharmacological chaperones for the treatment of a range of human genetic diseases, announced today that it will present the results of preclinical studies of Plicera™ (isofagomine tartrate, AT2101) for Gaucher disease at the American College of Medical Genetics (ACMG) annual meeting March 21-25 in Nashville, TN. The data demonstrate the ability of Plicera to increase levels of the target enzyme in cells derived from a patient with the N370S mutation and in mice that express the L444P mutation. The N370S and the L444P are the two most common mutations associated with Gaucher disease. Additional data from both studies will also be presented.

Plicera is designed to selectively bind to and stabilize GCase, the enzyme deficient in Gaucher disease. This deficiency leads to lysosomal accumulation of glucocerebroside inside certain cells, which is believed to cause the various symptoms of Gaucher disease. Plicera facilitates proper trafficking of the enzyme to the lysosomes, the compartments in the cell where it is needed to break down glucocerebroside.

The following is a summary of the preclinical Plicera data being presented at ACMG.

- In vitro exposure to Plicera increased transport of GCase to the lysosomes in cells derived from a patient with the N370S mutation. Once in the lysosome, the enzyme was stable and active for more than 3 days after Plicera was removed. The N370S is the most common mutation associated with Gaucher disease in the Western world. These studies were published in the September 12, 2006 issue of the Proceedings of the National Academy of Sciences (PNAS).

- Oral administration of Plicera resulted in a dose-dependent increase of GCase levels in various tissues, including the brain, in mice genetically modified to produce the L444P form of the enzyme. In addition, liver and spleen weights were decreased as were plasma levels of chitin III and IgG, which are biomarkers related to Gaucher disease. The L444P is one of the most common mutations associated with Gaucher disease. Gaucher patients with two copies of this mutation typically have neurological symptoms in addition to the visceral symptoms seen in Type I Gaucher disease.

About Gaucher Disease
Gaucher disease, the most commonly diagnosed lysosomal storage disorder, is caused by inherited genetic mutations in the GBA gene, which result in deficient activity of the enzyme acid β-glucosidase, also known as glucocerebrosidase (GCase). Deficient GCase activity leads to lysosomal accumulation of glucocerebroside inside certain cells, which is believed to cause the various symptoms of Gaucher disease, including an enlarged liver and spleen, abnormally low levels of red blood cells and platelets and skeletal complications. In some cases there is significant impairment of the central nervous system. Gaucher disease affects an estimated 8,000 to 10,000 people worldwide. The U.S. Food and Drug Administration's Office of Orphan Products Development has granted orphan drug designation for the active ingredient in Plicera in the United States.

About Amicus Therapeutics
Amicus Therapeutics is a biopharmaceutical company developing novel, oral therapeutics known as pharmacological chaperones for the treatment of a range of human genetic diseases. Pharmacological chaperone technology involves the use of small molecules that selectively bind to and stabilize proteins in cells, leading to improved protein folding and trafficking, and increased activity. Amicus is initially targeting lysosomal storage disorders, which are severe, chronic genetic diseases with unmet medical needs. Amicus is currently conducting Phase 2 clinical trials for its two lead compounds, Amigal™ for Fabry disease, and Plicera™ for Gaucher disease. The company is currently conducting Phase 1 trials with AT2220 for the treatment of Pompe disease.