July 5, 2007

Communications Office, Banyu Pharmaceutical Co., Ltd.,
Tel: +81-3-6272-1001

Public Relations, Ono Pharmaceutical Co., Ltd.
Tel: +81-6-6263-5670

New Data Released at ADA Showed that
Initial Combination Therapy with sitagliptin and Metformin Led to
Significant Improvement in Markers of Beta Cell Function
in Patients with Type 2 Diabetes (Second Report)

Merck & Co., Inc, Whitehouse Station, New Jersey, U.S.A. presented new data from an investigational study of initial combination therapy with the Company's oral medicine for type 2 diabetes, sitagliptin, and metformin on June 25, 2007 (US local time). The new data presented at the American Diabetes Association (ADA) 67th Annual Scientific Sessions held in Chicago (June 22 - 26), showed that initial combination therapy with both medicines demonstrated significant improvement in markers of beta cell function in patients with type 2 diabetes. Also, it is revealed that, when used in combination, the different mechanisms of action of sitagliptin and metformin had a complementary effect of increasing concentrations of glucagon-like peptide-1 (GLP-1) levels. Sitagliptin is a new oral medicine for type 2 diabetes, called DPP-4 inhibitor, which was launched in the United States last autumn.

Two main points of the data presented this time are as follows.

- Initial combination therapy with sitagliptin added to metformin demonstrated significant improvement in markers of beta cell function in patients with type 2 diabetes. Concretely speaking, the addition of JANUVIA to metformin resulted in a 49% increase in measured change in static beta cell responsiveness to above-basal glucose following a meal, compared with metformin alone. Further, the addition of JANUVIA to metformin resulted in a 114% increase in measured change in dynamic beta cell responsiveness to the rate of increase in above-basal glucose following a meal, compared with metformin alone.

- Additional new data from a study conducted in healthy adults suggested that, when used in combination, the different mechanisms of action of sitagliptin and metformin had a complementary effect on GLP-1 levels. This aspect of the mechanism of action of metformin used in combination with sitagliptin was previously unknown. GLP-1 is another hormone that is an important regulator of blood sugar levels, and acts, in part, by enhancing pancreatic beta cell insulin production and secretion. In this study, sitagliptin and metformin, when taken separately, increased overall, post-meal, active concentrations of GLP-1 levels by 1.95- and 1.76-fold, respectively, compared with placebo. When administered together, sitagliptin and metformin increased active concentrations of GLP-1 by 4.12-fold compared with placebo, which provides evidence that the two drugs may have complementary effects on GLP-1.

Sitagliptin enhances a natural body system, called the incretin (GLP-1 etc.) system, which helps to regulate glucose by affecting the beta cells and alpha cells in the pancreas. Pancreatic islet beta cell function determines the ability of the body to produce and secrete insulin, a hormone which plays a central role in the regulation of blood sugar levels. When food is consumed, GLP-1 is released by the gastrointestinal tract to stimulate the pancreatic beta cells to produce and secrete insulin. GLP-1 also suppresses the release of glucagon from the pancreatic alpha cells, which, in turn, tells the liver to reduce its production of sugar. Sitagliptin improves blood sugar control by increasing active concentrations of GLP-1 that has such effects, inhibiting its breakdown to inactive GLP-1.

In the study conducted in healthy adults, in contrast to active GLP-1 levels that were increased by both drugs when taken separately, the levels of total GLP-1 (which includes both active and inactive GLP-1) were increased by metformin only and not by sitagliptin. These observations are consistent with the effect of sitagliptin to raise active GLP-1 levels by reducing the clearance, but suggest that metformin acts in a different manner to increase active GLP-1 levels. “This is the first study in humans that showed that metformin increases the concentration of the incretin hormone, GLP-1; thus increasing the total GLP-1 available. When used together with sitagliptin, a DPP-4 inhibitor that blocks the degradation of GLP-1 by inhibiting its breakdown, there was an increase in GLP-1 levels greater than what either agent provided alone," said Nir Barzilai, M.D., professor of Medicine and Molecular Genetics and director of the Institute for Aging Research, Albert Einstein College of Medicine. "It appears as though metformin allows for more total GLP-1 to be available, and sitagliptin helps GLP-1 to remain active thus acting in a

complementary way."

Every day, more than 4,000 people in the United States are newly diagnosed with diabetes, yet 58 percent of patients will receive no formal education about their disease. Merck & Co., Inc, Whitehouse Station, New Jersey, U.S.A. is committed to contribute to diabetes community through corporate activities to provide patients with educational resources to support them by helping them make the necessary lifestyle modifications that lead to improved self-management as well as currently marketed diabetes products (sitagliptin, and combination drug of sitagliptin with metformin) and its pipeline of investigational diabetes compounds.

This press release is a short and recompiled version of the original second press release issued by Merck & Co., Inc, Whitehouse Station, New Jersey, U.S.A. on June 25, 2007 (US local time) in conjunction with the American Diabetes Association (ADA) 67th Scientific Sessions. The original press release in English can be accessed on the Company's web site (http://www.merck.com/newsroom/). The other short Japanese version of the first original press release issued on June 23, 2007 (US local time) was issued on July 2, 2007(Japan Time).

In Japan, sitagliptin is in Phase III by Banyu and Ono in accordance with the licensing agreement between Merck & Co., Inc, Whitehouse Station, New Jersey, U.S.A. and Ono executed in November 2004.

This press release is a short Japanese version into which is translated and redigested of the first press release issued by Merck & Co., Inc, Whitehouse Station, New Jersey, U.S.A. on June 25, 2007 (US local time), and thus the content and interpretation of the original English version have a priority over this Japanese version.