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Novartis Selects Drug Candidate for Clinical Development from Protein Kinase Research Collaboration with Vertex

--Novel compound, VX-322, targets key mechanisms implicated in leukemia and other cancers--

Cambridge, MA, November 10, 2004 -- Vertex Pharmaceuticals Incorporated (Nasdaq: VRTX) today announced that Novartis has selected a small molecule protein kinase inhibitor, VX-322, from Vertex for clinical development for the treatment of cancer. VX-322 is the first drug candidate to be transferred from Vertex to Novartis under an amended agreement to discover and develop drug candidates directed at the protein kinase gene family, and represents a novel, highly targeted approach to the treatment of cancer. In conjunction with the selection of VX-322, Vertex will receive $10 million from Novartis, a portion of which will be recognized by Vertex as revenue in the fourth quarter of 2004, with the remainder recognized through the term of the contract.

"Vertex's delivery of this innovative drug candidate highlights our progress in drug discovery on many promising molecular targets in the kinase gene family, as well as the continued strength of the research collaboration between Novartis and Vertex," said Peter Mueller, Ph.D., Senior Vice President of Drug Discovery and Innovation and Chief Scientific Officer at Vertex. "VX-322 represents a novel approach to address key molecular mechanisms that drive tumor growth, and we are pleased that it has been selected for development by Novartis, an industry leader in cancer drug development."

VX-322 is a potent, selective, dual inhibitor of the Flt-3 and c-kit kinases, which function as molecular switches that regulate the growth of certain cancers. Flt-3 inhibition has attracted significant attention among cancer researchers as a highly targeted approach to the treatment of certain leukemias as well as other hematological malignancies and solid tumors. Flt-3 is abnormally activated or upregulated in a wide range of leukemias, including more than 70% to 100% of patients with acute myelogenous leukemia (AML).

Current treatment for AML generally involves aggressive chemotherapy with "non-specific" agents that cannot discriminate between healthy and diseased cells, which results in significant toxicity and limited efficacy. The five-year survival rate for AML patients is 14%. New targeted approaches hold the potential to transform the treatment of AML: reducing side effects, improving tolerability and increasing the efficacy of chemotherapeutic regimens.

In May 2000, Vertex and Novartis entered an alliance to discover, develop and commercialize small molecule drugs directed at targets in the kinase protein family. The agreement was amended in February 2004, providing for a more rapid and earlier stage transfer of the drug candidates discovered by Vertex to Novartis for clinical development. Upon selection, Novartis holds worldwide development and commercialization rights to VX-322. Vertex will receive milestone payments based on clinical advancement of VX-322, and will receive royalties based on commercial sales.

Background Information: Flt-3 and c-kit Kinases, and Discovery of VX-322
VX-322 was discovered in a joint effort by scientists in Vertex's San Diego and Cambridge research sites, leveraging expertise in protein biochemistry, structural biology, high throughput cell assays and medicinal chemistry. Earlier in 2004, Vertex researchers published the three-dimensional atomic crystal structure of Flt-3 in the scientific journal Molecular Cell1, a key scientific advance that provided insight into the mechanism by which mutated forms of the Flt-3 receptor can activate themselves, triggering uncontrolled proliferation of immature blood cells characteristic in several types of leukemia.

Flt-3 kinase is abnormally activated or upregulated in the majority of patients with AML, as well as in patients with acute lymphoblastic leukemia (ALL) and chronic myelogenous leukemia (CML). Specific Flt-3 mutations that are believed to be key drivers of cell proliferation are present in up to 40% of AML patients. In addition, the protein c-kit has been found at high levels in 60% to 80% of AML patients. Preclinical studies conducted at Vertex using cells isolated from AML patients have suggested that dual Flt-3/c-kit inhibition provides more potent reduction in cell proliferation compared to inhibitors of Flt-3 or c-kit kinase alone. These data have enabled researchers to design molecules with optimized inhibitory profiles against Flt-3 and c-kit kinases.

Key Facts About Leukemia
Leukemia is a cancer of the blood characterized by uncontrolled growth and proliferation of cells in bone marrow, the tissue responsible for the production of all blood cells. According to the Leukemia and Lymphoma Society, an estimated 33,440

individuals will be diagnosed with leukemia in the U.S. in 2004, more than one third of whom will have acute myelogenous leukemia (AML), the most common form.

About Vertex
Vertex Pharmaceuticals Incorporated is a global biotechnology company committed to the discovery and development of breakthrough small molecule drugs for serious diseases. The Company's strategy is to commercialize its products both independently and in collaboration with major pharmaceutical partners. Vertex's product pipeline is principally focused on viral diseases, inflammation, autoimmune diseases and cancer. Vertex co-promotes the new HIV protease inhibitor, Lexiva(R), with GlaxoSmithKline.

This press release may contain forward-looking statements, including statements that (i) VX-322 could potentially provide therapeutic benefits for the treatment of leukemias as well as other hematological malignancies and solid tumors; and, (ii) new targeted approaches hold the potential to transform the treatment of AML. While management makes its best efforts to be accurate in making forward-looking statements, such statements are subject to risks and uncertainties that could cause Vertex's actual results to vary materially. These risks and uncertainties include, among other things, risks that initial nonclinical testing of VX-322 will not be predictive of results obtained in human clinical trials, that further nonclinical and clinical testing will result in the discovery of unfavorable information about VX-322 that will limit or eliminate its potential for successful completion of clinical trials, that targeted approaches to the treatment of AML will not prove to be effective, and other risks listed under Risk Factors in Vertex's form 10-K filed with the Securities and Exchange Commission on March 15, 2004 and amended on September 8, 2004.

Lexiva(R) is a registered trademark of the GlaxoSmithKline group of companies.

1Griffith, J, Black J, Faerman C, Swenson L, Wynn M, Lu F, Lippke J, and Saxena K. (2004) The structural basis for autoinhibition of Flt3 by the juxtamembrane domain. Molecular Cell 13: 169-178.

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