Exelixis Reports Phase 1 Data for XL019 at ASH Annual Meeting

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Encouraging Clinical Activity Seen in Refractory Pre-leukemic Myelofibrosis Patients

SAN FRANCISCO--(BUSINESS WIRE)--Dec. 7, 2008--Exelixis, Inc. (Nasdaq:EXEL) today reported phase 1 data from a dose-escalation study of XL019 in patients with myelofibrosis (MF). XL019 is a potent small molecule inhibitor of JAK2, a protein kinase that is mutationally-activated in approximately 50% of patients in MF. Neil Shah, MD, Ph.D., Assistant Professor in the Division of Hematology/Oncology at the University of California, San Francisco, reported the data in an oral presentation (Abstract #98) at the 50th Annual Meeting of the American Society of Hematology, which is being held Dec. 6-8, 2008 in San Francisco.

The reported data are from a phase 1 trial of XL019 in patients with advanced primary myelofibrosis, or with advanced myelofibrosis occurring post-polycythemia vera or post-essential thrombocythemia. This study included patients with evidence of leukemic transformation, which has been reported to be uniformly fatal after a median of 2.6 months (Kroger and Mesa, Leukemia, vol. 22, p. 474, 2008). The dose-escalation trial enrolled a total of 30 patients. The presentation focused on 21 patients who received XL019 at doses of 25 mg and 50 mg. Data on 9 patients who received doses of greater or equal to 100 mg had been reported previously. Disease assessments included hematologic parameters, spleen size, and bone marrow and neurologic evaluation.

Evidence of clinical activity was seen in patients with JAK2 V617F or MPL W515L mutations, and included the following: 50% or greater reduction of spleen size in 50% of patients receiving 25 or 50 mg XL019 daily, and in 25% of patients receiving 25 mg XL019 qMWF; reduction in leukocytosis in 7 of 9 patients who presented with baseline leukocytosis of greater than 15,000/mm^3; improvement in anemia in 4 patients; and relief of constitutional symptoms in 6 of 8 patients with baseline pruritus or inappetence.

Three of four patients with pre-leukemic transformation showed a reduction in the levels of circulating blasts after the first cycle of XL019, and a duration on study of 2.5, 3+, and 7.5+ months. Two of these patients also showed normalization of blast counts in the bone marrow. Of the three patients who showed a reduction in circulating blasts, two had the JAK2 V617F mutation and one had wild type JAK2.

"With these newest data, XL019 continues to show evidence of clinical activity in patients with myeloproliferative disorders at tolerable doses," said Michael Morrissey, Ph.D., president of research and development at Exelixis. "Importantly, we have seen evidence of hematologic improvement in pre-leukemic patients for the first time, including reductions in circulating and bone marrow blasts. We view these results with particular interest as they point to a potential sign of disease modification with a selective JAK2 inhibitor, and will factor them into our future development plans for the compound."

XL019 was generally well-tolerated. No drug-related hematologic adverse events were observed at the 25 mg and 50 mg dose levels. Mild to moderate adverse events including disgeusia, fatigue, nausea, balance disorder, confusional state, paresthesia, and skin rash have been reported. Mild peripheral neuropathy was observed in 1 of 16 patients at the 25 mg dose, and mild to moderate neuropathy in 2 of 5 patients at the 50 mg dose.

About JAK2 and XL019

JAK kinases are activated by cytokines and growth factor receptors and phosphorylate members of the STAT family of inducible transcription factors. JAK2 plays a pivotal role in the cellular response to growth factors that drive blood cell expansion, including erythropoietin and thrombopoietin. Mutational activation of JAK2 is observed in the majority of patients with myeloproliferative diseases including polycythemia vera, essential thrombocythemia, and myelofibrosis, and is thought to drive the inappropriate expansion of blood cells observed in these conditions. Other members of the JAK family play critical roles in regulating immune responses, including the anti-viral and anti-parasitic responses.

XL019 is a potent inhibitor of JAK2 (IC50 = 2 nM), and is selective for JAK2 versus the other members of the JAK kinase family (JAK1 IC50 = 130 nM, JAK3 IC50 = 250 nM, TYK2 IC50 = 340 nM). It is active against both wild type and mutationally activated forms of JAK2, and showed good oral bioavailability and pharmacodynamic properties in preclinical studies.

About Exelixis

Exelixis, Inc. is a development-stage biotechnology company dedicated to the discovery and development of novel small molecule therapeutics for the treatment of cancer and other serious diseases. The company is leveraging its fully integrated drug discovery platform to fuel the growth of its development pipeline, which is primarily focused on cancer. Currently, Exelixis' broad product pipeline includes investigational compounds in phase 3, phase 2, and phase 1 clinical development. Exelixis has established strategic corporate alliances with major pharmaceutical and biotechnology companies, including GlaxoSmithKline, Bristol-Myers Squibb, Genentech, Wyeth Pharmaceuticals and Daiichi-Sankyo. For more information, please visit the company's website at http://www.exelixis.com.

Forward-Looking Statements

This press release contains forward-looking statements, including, without limitation, statements related to the therapeutic potential and development path for XL019. Words such as "potential" and similar expressions are intended to identify forward-looking statements. These forward-looking

statements are based upon Exelixis' current plans, assumptions, beliefs, and expectations. Forward-looking statements involve risks and uncertainties. Exelixis' actual results and the timing of events could differ materially from those anticipated in such forward-looking statements as a result of these risks and uncertainties, which include, without limitation, risks related to the potential failure of XL019 to demonstrate safety and efficacy in clinical testing; the therapeutic and commercial value of XL019; and the ability to conduct XL019 clinical trials sufficient to achieve a positive completion. These and other risk factors are discussed under "Risk Factors" and elsewhere in Exelixis' quarterly report on Form 10-Q for the quarter ended September 26, 2008, and other filings with the Securities and Exchange Commission. Exelixis expressly disclaims any duty, obligation, or undertaking to release publicly any updates or revisions to any forward-looking statements contained herein to reflect any change in Exelixis' expectations with regard thereto or any change in events, conditions, or circumstances on which any such statements are based.

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