



Pfizer Oncology To Present Over 30 Abstracts On Pre-Clinical And Clinical Research At The American Association For Cancer Research Annual Meeting

Tuesday, April 13, 2010 - 10:30pm

First-ever Data Presentation of PF-04691502, Pfizer's Dual PI3K/mTOR Inhibitor

"Seniors represent a rapidly growing population globally whose independence and complete health and wellness needs are not fully understood,"

(BUSINESS WIRE)--Pfizer Oncology announced today that it will present new data from the company's pipeline, including two novel, dual PI3K/mTOR (phosphatidylinositol 3-kinase / mammalian target of rapamycin) inhibitors (PF-04691502 and PKI-587, also known as PF-05212384);² crizotinib (PF-02341066), an ALK (anaplastic lymphoma kinase) inhibitor that also inhibits c-MET (c-mesenchymal-epithelial transition factor);³ and PF-04605412, an anti- $\alpha 5\beta 1$ inhibitor.⁴ Data from these, and other investigational compounds, including sunitinib,⁵ axitinib,⁶ and figitumumab,⁷ will be presented at the American Association for Cancer Research (AACR) 101st Annual Meeting 2010 in Washington, D.C., from April 17 through April 21.

"Pfizer's data at AACR demonstrate our strategic focus on the discovery and development of novel mechanisms that align complex biology with creative clinical strategies," said Dr. Neil Gibson, chief scientific officer of Pfizer's Oncology Research Unit. "Our research on the mutations and pathways that are genetic drivers of the segments of disease, defined by specific 'genetic signatures,' is designed to help us identify patient populations that will derive the most benefit from the novel therapeutic modalities we discover and

develop."

On Sunday, April 18, Pfizer Oncology researchers and leadership will host a media briefing to provide a high-level overview of Pfizer's novel research at AACR. The briefing, "An 'Inside Look' at Early Clinical Development at Pfizer," will be held at The Renaissance Hotel, Room 2, from 7:00 – 8:00 p.m.

Seven Abstracts Presented on New Agents in an Emerging Class

The PI3K pathway and key kinases within it, such as mTOR, are believed to play a central role in regulating cellular signaling that may influence cancer cell growth and survival.⁸ Genetic abnormalities in the pathway have been closely linked to the development and progression of cancer.⁸

Pfizer has two dual PI3K/mTOR inhibitors in development, one administered orally (PF-04691502)¹ and one intravenously (PKI-587/PF-05212384).² PF-04691502 will be featured during an oral presentation at AACR (Abstract #5779, April 21), providing details that led to the discovery of this selective compound.¹

An additional six abstracts on PF-04691502 and PKI-587/PF-05212384 will be presented at the meeting (Abstract #s 302,⁹ 4473,¹⁰ 4479,¹¹ 3224,¹² 349213 and 723)². Based on these preliminary studies, trials are underway to assess safety and tolerability of these agents in cancer patients with solid tumors.^{14,15}

Pfizer's First-In-Class Agents: Crizotinib (PF-02341066) and PF-04605412

Scientific advances in personalized medicine have led to the identification of ALK as a new therapeutic target in cancer. Crizotinib (PF-02341066) is a first-in-class investigational, oral ALK inhibitor which also inhibits c-MET.¹⁶ The fusion gene EML4-ALK is thought to be a key driver of lung tumorigenesis,¹⁷ and is estimated to be present in approximately 40,000 patients worldwide with newly diagnosed non-small cell lung cancer (NSCLC) annually.¹⁸ A pharmacokinetic and pharmacodynamic (QTc) population analysis from an open label, multi-center Phase 1 dose escalation study in patients with advanced cancer will be presented at AACR (Abstract #1673; April 19).³

In addition, various clinical studies suggest the adhesion molecule known as integrin $\alpha 5\beta 1$ plays an important role in cancer progression through the promotion of cancer cell migration, proliferation, survival and metastasis.¹⁹ In lung cancer patients, a five-year survival rate was found to be inversely correlated with the degree of tumor $\alpha 5$ expression, and similar associations have been noted in melanoma and ovarian cancer.¹⁹

Accumulating evidence also suggests that antibody-dependent cellular cytotoxicity (ADCC) plays a significant role in anti-cancer therapy.¹⁹ At AACR, Pfizer will present data on PF-04605412, a first-in-class, selective, fully human anti- $\alpha 5\beta 1$ IgG1 antibody that has been specifically engineered to possess ADCC activity in preclinical models (Abstract #3811, April 20).⁴

Research Presentations on Causes of Drug Resistance and Novel Compounds in Animal Models

A novel SND1-BRAF fusion confers resistance to c-Met inhibitor PF-04217903 in GTL16 cells (Abstract #628; April 18)²⁰ Development of a model of acquired resistance to a multi-targeted VEGFR TKI and strategies to target resistance mechanisms through combination (Abstract #387; April 18)²¹ Establishing patient-derived colorectal cancer stem cell models with PI3CA mutation for the development of inhibitor drugs as targeted therapies (Abstract #4483; April 20)²² Inhibition of tumor malignancy by anti-angiogenic therapies in orthotopic mouse models of hepatocellular carcinoma (Abstract #4177; April 20)²³ Effects of a novel PI3 kinase/mTOR inhibitor on proliferation and pAKT signaling in canine lymphoma (Abstract #5043; April 21)²⁴

Additional Data Presentations

Sunitinib in advanced NSCLC: Correlation of circulating biomarkers with clinical outcome (Abstract #4682; April 20)⁵ Molecular predictors of sensitivity to an IGF-1R inhibitor (figitumumab) in pre-clinical models of lung and colon cancers (Abstract #LB-220; April 20)⁷ Pharmacogenomic analysis of a Phase 3 trial of gemcitabine plus axitinib versus gemcitabine plus placebo in patients with advanced pancreatic cancer (Abstract #78; April 18)⁶ An open-label Phase 1 study to evaluate the pharmacokinetics of axitinib (AG-013736) in healthy Chinese volunteers (Abstract #2762; April 19)²⁵

Follow Pfizer at AACR through Twitter @pfizer_news (http://twitter.com/pfizer_news).

About Pfizer Oncology

Pfizer Oncology is committed to the discovery, investigation and development of innovative treatment options to improve the outlook for cancer patients worldwide. Our strong pipeline, one of the most robust in the industry, is studied with precise focus on identifying and translating the best scientific breakthroughs into clinical application for patients across a wide range of cancers, including breast, lung, prostate, sarcoma, melanoma, and various hematologic cancers. Pfizer Oncology has biologics and small molecules in clinical development and more than 200 clinical trials underway.

By working collaboratively with academic institutions, individual researchers, cooperative research groups, governments, and licensing partners, Pfizer Oncology strives to cure or control cancer with breakthrough medicines, to deliver the right drug for each patient at the right time.

For more information please visit www.Pfizer.com.

Pfizer Inc: Working together for a healthier world™

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DISCLOSURE NOTICE: The information contained in this release is as of April 14, 2010. Pfizer assumes no obligation to update forward-looking statements contained in this release as the result of new information or future events or developments.

This release contains forward-looking information about various oncology products in development, including their potential benefits, that involves substantial risks and uncertainties. Such risks and uncertainties include, among other things, the uncertainties inherent in research and development; decisions by regulatory authorities regarding whether and when to approve any drug applications that may be filed for any such products in development as well as their decisions regarding labeling and other matters that could affect their availability or commercial potential; and competitive developments.

A further description of risks and uncertainties can be found in Pfizer's Annual Report on Form 10-K for the fiscal year ended December 31, 2009 and in its reports on Form 10-Q and Form 8-K.

1 AACR Accepted Abstract #5779. The Discovery of the Potent and Selective PI3K/mTOR Dual Inhibitor, PF-04691502, through Structure Based Drug Design. Oral Presentation, Wednesday, April 21, 2010: 9:30am-12:00pm. H. Cheng – Presenter. American Association for Cancer Research 101st Annual Meeting. Washington, DC. April 17-21, 2010.

2 AACR Accepted Abstract #723. Beyond Temsirolimus: Discovery of PKI-587 a highly efficacious dual PI3 kinase/mTOR inhibitor. General Poster Presentation, Sunday April 18, 2010, 2:00pm-5:00pm. A. Venkatesan – Presenter. American Association for Cancer Research 101st Annual Meeting. Washington, DC. April 17-21, 2010.

3 AACR Accepted Abstract #1673. A pharmacokinetics/pharmacodynamics evaluation of the concentration-QTc relationship of PF-02341066 (PF-1066), an ALK and c-MET/HGFR dual inhibitor administered orally to patients with advanced cancer. General Poster Presentation, Monday April 19, 2010: 9:00am-12:00pm. D. Nickens – Presenter. American Association for Cancer Research 101st Annual Meeting. Washington, DC. April 17-21, 2010.

4 AACR Accepted Abstract #3811. PF-04605412, a dual functional anti-alpha-5-beta-1 monoclonal antibody with enhanced ADCC demonstrates robust anti-tumor activity in nonclinical studies. General Poster Presentation, Tuesday April 20, 2010: 9:00am-12:00pm. D. Hu-Lowe – Presenter. American Association for Cancer Research 101st Annual Meeting. Washington, DC. April 17-21, 2010.

5 AACR Accepted Abstract #4682. Sunitinib (SU) in advanced NSCLC: Correlation of circulating biomarkers with clinical outcome. General Poster Presentation, Tuesday April 20, 2010: 2:00pm-5:00pm. C. Harmon – Presenter. American Association for Cancer Research 101st Annual Meeting. Washington, DC. April 17-21, 2010.

6 AACR Accepted Abstract #78. Pharmacogenomic analysis of a phase III trial of gemcitabine/axitinib vs gemcitabine/placebo in patients (pts) with advanced pancreatic cancer. General Poster Presentation, Sunday April 18, 2010: 2:00-5:00pm. K. Loomis – Presenter. American Association for Cancer Research 101st Annual Meeting. Washington, DC. April 17-21, 2010.

7 AACR Accepted Late Breaker Abstract #LB-220. Molecular predictors of sensitivity to an IGF1R inhibitor in pre-clinical models of lung and colon cancers. General Poster Presentation, Tuesday April 20, 2010: 9:00am-12:00pm. M. Lira – Presenter. American Association for Cancer Research 101st Annual Meeting. Washington, DC. April 17-21, 2010.

8 Gridelli C et al. The Potential Role of mTOR Inhibitors in Non-Small Cell Lung Cancer. *The Oncologist*. 2008; 13; 139-147.

9 AACR Accepted Abstract #302. Activity of PF-04691502, a dual PI3K/mTOR inhibitor in breast cancer cell lines and models discriminates between ER, PR and HER2 positive and negative segments. General Poster Presentation, Tuesday April 20, 2010: 2:00pm - 5:00pm. J. Yuan – Presenter. American Association for Cancer Research 101st Annual Meeting. Washington, DC. April 17-21, 2010.

10 AACR Accepted Abstract #4473. PF-04691502, a Potent and Selective PI3K/mTOR Dual Inhibitor, Demonstrates in vitro and in vivo Anti-tumor Activity in Non-Small-Cell-Lung Carcinoma Cells. General Poster Presentation, Tuesday April 20, 2010: 2:00pm-5:00pm. P.Mehta – Presenter. American Association for Cancer Research 101st Annual Meeting. Washington, DC. April 17-21, 2010.

11 AACR Accepted Abstract #4479. PF-04691502, a potent and selective PI3K/mTOR dual inhibitor with anti-tumor activity. General Poster Presentation, Tuesday April 20, 2010: 2:00pm-5:00pm. S. Bagrodia – Presenter. American Association for Cancer Research 101st Annual Meeting. Washington, DC. April 17-21, 2010.

12 AACR Accepted Abstract #3224. Preclinical PKPD modeling and human dose projection of PF-04691502, a PI3K/mTOR dual inhibitor. General Poster Presentation, Tuesday April 20, 2010: 9:00am-12:00pm. K. Luu – Presenter. American Association for Cancer Research 101st Annual Meeting. Washington, DC. April 17-21, 2010.

13 AACR Accepted Abstract #3492. A novel PI3K/mTOR dual inhibitor provides correlations of pharmacokinetic pharmacodynamic and tumor growth inhibition in a prostate PC3 xenograft model for application in clinical trials. General Poster Presentation, Tuesday April 20, 2010: 9:00am-12:00pm. W. Carley – Presenter. American Association for Cancer Research 101st Annual Meeting. Washington, DC. April 17-21, 2010.

14 ClinicalTrials.gov. A Trial to Assess Safety and Tolerability of PF-04691502 in Cancer Patients. Available at: <http://www.clinicaltrials.gov/ct2/show/NCT00927823?term=PF-04691502&phase=0&rank=1>. Accessed March 30, 2010.

15 Clinicaltrials.gov. Study of PKI-587 Administered Intravenously to Subjects with Solid Tumors. Available at: <http://www.clinicaltrials.gov/ct2/show/NCT00940498?term=PKI-587&rank=1>. Accessed March 30, 2010.

16 ASCO Accepted Abstract #3509. Clinical Activity Observed in A Phase 1 Dose Escalation Trial of an Oral C-Met And ALK Inhibitor, PF-02341966. Poster presentation. May 30, 2009: 300PM. E. Kwak- Presenter. American Society of Clinical Oncology Annual Meeting. Orlando, FL. May 29 – Jun , 2009.

17 Chiarle R, et al. The Anaplastic Lymphoma Kinase in the Pathogenesis of Cancer. *Nat Rev Cancer*. 2008;8(1):11-23.

18 Palmer H et al. Anaplastic Lymphoma Kinase: Signaling in Development and Disease. *Biochem Journal*. 2009; 420: 345-361.

19 Hu-Lowe D et al. Robust Anti-tumor Activity Through Targeting Integrin $\alpha 5\beta 1$ and Eliciting ADCC With A Dual Functional Monoclonal Antibody PF-04605412. AACR Accepted Poster. Presented Tuesday April 20,2010: 9:00am-12:00pm. D. Hu-Lowe – Presenter. American Association for Cancer Research 101st Annual Meeting. Washington, DC. April 17-21, 2010.

20 AACR Accepted Abstract #628. A novel SND1-BRAF fusion confers resistance to c-Met inhibitor PF-04217903 in GTL16 cells. General Poster Presentation, Sunday April 18, 2010: 2:00pm-5:00pm. N. Lee – Presenter. American Association for Cancer Research 101st Annual Meeting. Washington, DC. April 17-21, 2010.

21 AACR Accepted Abstract #387. Development of a model of acquired resistance to a multi targeted VEGFR TKI and strategies to target resistance mechanisms through combination. General Poster Presentation, Sunday April 18, 2010: 2:00pm-5:00pm. S. Garza – Presenter. American Association for Cancer Research 101st Annual Meeting. Washington, DC. April 17-21, 2010.

22 AACR Accepted Abstract #4483. Establishing patient-derived colorectal cancer stem cell models with PI3CA mutation for the development of inhibitory drugs as targeted therapies . General Poster Presentation, Tuesday April 20, 2010: 2:00pm – 5:00pm. D. Fang- Presenter. American Association for Cancer Research 101st Annual Meeting. Washington, DC. April 17-21, 2010.

23 AACR Accepted Abstract #4177. Inhibition of tumor malignancy by anti-angiogenic therapies in orthotopic mouse models of hepatocellular carcinoma. General Poster Presentation, Tuesday April 20, 2010: 2:00pm-5:00pm. G. Li-Presenter. American Association for Cancer Research 101st Annual Meeting. Washington, DC. April 17-21, 2010.

24 AACR Accepted Abstract #5043. Effects of a novel PI3 kinase/mTOR inhibitor on proliferation and pAKT signaling in canine lymphoma. General Poster Presentation, Wednesday April 21, 2010: 8:00am- 11:00am. P. Berlinski – Presenter. American Association for Cancer Research 101st Annual Meeting. Washington, DC. April 17-21, 2010.

25 AACR Accepted Abstract #2762. An open-label phase 1 study to evaluate the pharmacokinetics of axitinib (AG-013736) in healthy Chinese volunteers. General Poster Presentation, Monday April 19, 2010: 2:00pm-5:00pm. Y. Chen – Presenter. American Association for Cancer Research 101st Annual Meeting. Washington, DC. April 17-21, 2010.

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