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Pfizer Oncology To Present New Clinical Data From Ten Molecules Across Multiple Tumor Types

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Data to be Featured from Pfizer's Lung Cancer Portfolio, Including Crizotinib (PF-02341066) and a Pan-HER Inhibitor (PF-00299804)

NEW YORK, N.Y., May 20 - Pfizer Oncology will present new data highlighting the company's focused approach to cancer drug development through the identification and validation of molecular targets. These results will be presented at the 46th Annual American Society of Clinical Oncology (ASCO) meeting in Chicago from June 4-8.

"Pfizer Oncology is committed to applying discoveries from cancer biology and genetics to the development of new drugs in a focused and rational way. This approach is being used to study crizotinib (PF-02341066), an ALK inhibitor, in lung cancer patients carrying the *ALK* fusion gene, and bosutinib, a dual Src and Bcr-Abl kinase inhibitor, in patients with chronic myelogenous leukemia who have progressed despite standard-dose imatinib," said Dr. Mace Rothenberg, senior vice president of clinical development and medical affairs for Pfizer's Oncology Business Unit. "We're also working to apply this personalized approach to better understand how already approved agents, such

as Sutent®, may work better in certain patient populations within approved indications."

Advancing Research and Understanding in NSCLC

Pfizer Oncology has multiple compounds in development to treat the various forms of non-small cell lung cancer (NSCLC).

At ASCO, Pfizer will present updated data from an expansion cohort of a Phase 1/2 study evaluating crizotinib (PF-02341066) in patients with NSCLC carrying the *ALK* (anaplastic lymphoma kinase) fusion gene.[1] Alteration of the *ALK* gene is thought to be a key driver of lung tumorigenesis in a subset of NSCLC patients.[2,3] Worldwide, it is estimated that approximately 45,000 newly diagnosed NSCLC patients per year are *ALK* positive.[4] These data will be featured in the ASCO Plenary Session on Sunday, June 6 (Abstract #3).

Crizotinib (PF-02341066), a first-in-class, oral ALK inhibitor,[5] is currently being evaluated in an open-label randomized Phase 3 trial, known as PROFILE 1007, which compares crizotinib (PF-02341066) with standard of care chemotherapy in the treatment of *ALK*-positive recurrent NSCLC.[6] Crizotinib is also being studied in a Phase 2 trial, known as PROFILE 1005, in similar patients who have received more than one line of prior chemotherapy. Pfizer has partnered with Abbott Molecular to develop a diagnostic to screen patients with NSCLC for the presence of alterations in the *ALK* gene.

PF-00299804 is an investigational, oral, pan-HER (pan-human epidermal growth factor receptor) inhibitor. It is an irreversible small molecule inhibitor of HER-1 (EGFR - epidermal growth factor receptor), -2 and -4 tyrosine kinase. Data from a global, randomized Phase 2 trial evaluating the anti-tumor activity and safety of PF-00299804 compared to erlotinib, in patients with NSCLC following progression on treatment with one

or more chemotherapy regimens, will be presented in a poster discussion session (Abstract #LBA7523, June 7).[7] PF-00299804 is currently being evaluated in BR.26, a Phase 3 double-blind, placebo-controlled, randomized study in patients with stage IIIB/IV NSCLC who have received standard chemotherapy and EGFR inhibitor therapy. This trial is being led by the National Cancer Institute of Canada (NCIC) - Clinical Trial Group (Kingston, Ontario).

Pfizer will present results from a Phase 3 trial (A4021016) of figitumumab (CP-751,871), a selective fully human IgG2 monoclonal antibody against the insulin like growth factor 1 receptor (IGF-1R) pathway, in combination with paclitaxel and carboplatin versus paclitaxel and carboplatin in patients with NSCLC (Abstract #7500, June 5).[8] This study was discontinued in December 2009 following the recommendation of an independent Data Safety Monitoring Committee who found that the addition of figitumumab to paclitaxel plus carboplatin would be unlikely to meet the primary endpoint of overall survival compared to paclitaxel plus carboplatin alone. Pfizer is continuing to analyze the clinical and biomarker data from this and other trials to identify a potential subset of NSCLC patients in whom further evaluation of figitumumab is warranted.

Additional Research in Identifying Molecular Targets

Bosutinib is an investigational oral dual Src and Bcr-Abl kinase inhibitor.[9] It is believed that bosutinib may inhibit activated Bcr-Abl in chronic myeloid leukemia (CML) cells that allow the cells to grow, survive and reproduce.[10]

- Phase 2 study of safety and efficacy of bosutinib in patients with Philadelphia chromosome positive chronic phase CML who are resistant to imatinib and other tyrosine kinases (Abstract #6502, June 7)[11]

Bosutinib is currently being evaluated in an ongoing, randomized, open-label, Phase 3 trial which compares bosutinib with standard dose imatinib in patients with previously untreated, chronic phase, Philadelphia chromosome (Ph+) CML.

As part of Pfizer Oncology's commitment to advancing the science of renal cell carcinoma (RCC), Pfizer has partnered with Genomic Health, Inc. a molecular diagnostic company, on the development of a genomic test to estimate the risk of recurrence following surgery for patients with Stage 1-3 clear cell RCC.

- Identification of prognostic genomic markers in patients with localized clear cell RCC (Abstract #4501, June 5)[12]

Ongoing Evaluation of Sutent (sunitinib malate)

Sutent has played a significant role in advancing treatment for patients with advanced RCC or gastrointestinal stromal tumor (GIST) after disease progression on or intolerance to imatinib mesylate and remains a standard of care in these indications. Pfizer is continuing to study the potential role of Sutent in the treatment of various solid tumors including advanced NSCLC,[13] advanced castration-resistant prostate cancer,[14] and as adjuvant therapy for RCC, in Phase 3 trials.[15]

- Phase 3 patient-reported outcomes study in patients with pancreatic neuroendocrine tumors (NET) receiving sunitinib (Abstract #4003, June 5)[16]
- Updated efficacy and safety results of the Phase 3 trial of sunitinib versus placebo for the treatment of pancreatic NET (Abstract #4000, June 5)[17]

Analyses of efficacy and safety data from two Phase 3 trials of sunitinib in metastatic/advanced breast cancer will be presented. Earlier this year Pfizer announced that these studies did not meet their primary endpoints.

- Sunitinib in combination with docetaxel (D) vs D alone for the first-line treatment of advanced breast cancer (ABC) (Abstract #LBA1010, June 8)[18]
- Phase 3 Study of Sunitinib plus Capecitabine in Previously Treated ABC (Abstract # LBA1011, June 8)[19]

Additional Data Presentations

Pfizer will also present data from its development program, including:

- Interim data for ACT III - Phase 2 trial of PF-04948568 (CDX-110) [an investigational immuno-oncology vaccine targeting the tumor specific EGFR mutant EGFRvIII] in combination with temozolomide in patients with glioblastoma (Abstract #2014, June 5)[20]
- Final report of a phase 1 clinical pharmacokinetic and pharmacodynamic study of PD-00562271 targeting focal adhesion kinase (FAK) in advanced solid tumors (Abstract #3028, June 6)[21]
- Pharmacodynamically guided dose selection of PF-00337210, a VEGFR2 tyrosine kinase inhibitor in a phase 1 study (Abstract #3033, June 6)[22]
- First-in-human dose-escalation safety and PK trial of a novel intravenous humanized monoclonal CovX body inhibiting angiopoietin (Abstract #2524, June 5)[23]

Data on the following compounds and investigational agents will also be presented: axitinib (RCC),[24] Torisel (RCC),[25] Camptosar® (colorectal cancer),[26] and tremelimumab (melanoma, pancreatic).[27,28]

For more news and information, follow Pfizer Oncology at ASCO through Twitter @pfizer_news (http://twitter.com/pfizer_news).

About Sutent® (sunitinib malate)

Sutent is an oral multi-kinase inhibitor approved for the treatment of GIST after disease progression on or intolerance to imatinib mesylate and advanced / metastatic RCC.

Sutent works by blocking multiple molecular targets implicated in the growth, proliferation and spread of cancer. Two important Sutent targets, vascular endothelial growth factor receptor (VEGFR) and platelet-derived growth factor receptor (PDGFR), are expressed by many types of solid tumors and are thought to play a crucial role in angiogenesis, the process by which tumors acquire blood vessels, oxygen and nutrients needed for growth. Sutent also inhibits other targets important to tumor growth, including KIT, FLT3 and RET.

Important Sutent® (sunitinib malate) Safety Information

Hepatic failure was observed in <1% of solid tumor patients treated with Sutent, sometimes with a fatal outcome. Monitor liver function tests and if symptoms of hepatic failure are present, patients should have Sutent discontinued.

Women of childbearing age who are (or become) pregnant during therapy should be informed of the potential for fetal harm while on Sutent.

Decreases in left ventricular ejection fraction (LVEF) to below the lower limit of normal (LLN) have been observed. Patients with concomitant cardiac conditions should be carefully monitored for clinical signs and symptoms of congestive heart failure.

Patients should be monitored for hypertension and treated as needed with standard antihypertensive therapy. Complete blood counts (CBCs) with platelet count and serum chemistries should be performed at the beginning of each treatment cycle for patients receiving treatment with Sutent.

The most common adverse reactions in GIST and RCC clinical trials were fatigue, asthenia, diarrhea, nausea, mucositis/stomatitis, vomiting, dyspepsia, abdominal pain, constipation, hypertension, rash, hand-foot syndrome, skin discoloration, altered taste, anorexia and bleeding.

For more information on Sutent and Pfizer Oncology, including full prescribing information for Sutent (sunitinib malate), please visit www.pfizer.com.

About Torisel® (temsirolimus)

Torisel is the only intravenous mammalian target of rapamycin (mTOR) inhibitor approved for the treatment of advanced renal cell carcinoma (RCC) in the first-line setting.

Based on preclinical studies, Torisel inhibits the activity of mTOR, an intracellular protein implicated in multiple growth-related cellular functions including proliferation, growth and survival. The inhibition of mTOR also reduces levels of certain growth factors, such as vascular endothelial growth factor (VEGF), which are overexpressed in solid tumors like kidney cancer and are thought to play a crucial role in angiogenesis, the process by which tumors acquire blood vessels, nutrients and oxygen needed for growth.

Important Torisel® (temsirolimus) Safety Information

Hypersensitivity reactions manifested by symptoms, including, but not limited to anaphylaxis, dyspnea, flushing, and chest pain have been observed with Torisel. Serum glucose, serum cholesterol, and triglycerides should be tested before and during treatment with Torisel. The use of Torisel is likely to result in hyperglycemia and hyperlipemia. This may result in the need for an increase in the dose of, or initiation of, insulin and/or oral hypoglycemic agent therapy and/or lipid-lowering agents, respectively.

The use of Torisel may result in immunosuppression. Patients should be carefully observed for the occurrence of infections, including opportunistic infections. Cases of interstitial lung disease, some resulting in death, have occurred. Some patients were asymptomatic and others presented with symptoms. Some patients required discontinuation of Torisel and/or treatment with corticosteroids and/or antibiotics. Cases of fatal bowel perforation occurred with Torisel. These patients presented with fever, abdominal pain, metabolic acidosis, bloody stools, diarrhea, and/or acute abdomen. Cases of rapidly progressive and sometimes fatal acute renal failure not clearly related to disease progression occurred in patients who received Torisel.

Due to abnormal wound healing, use Torisel with caution in the perioperative period. Patients with central nervous system tumors (primary CNS tumor or metastases) and/or receiving anticoagulation therapy may be at an increased risk of developing intracerebral bleeding (including fatal outcomes) while receiving Torisel. Live vaccinations and close contact with those who received live vaccines should be avoided. Patients and their partners should be advised to avoid pregnancy throughout treatment and for 3 months after Torisel therapy has stopped.

The most common (incidence ≥30 percent) adverse reactions observed with Torisel are: rash (47 percent), asthenia (51 percent), mucositis (41 percent), nausea (37 percent), edema (35 percent), and anorexia (32 percent). The most common laboratory abnormalities (incidence ≥30 percent) are anemia (94 percent), hyperglycemia (89 percent), hyperlipemia (87 percent, hypertriglyceridemia (83 percent), elevated alkaline phosphatase (68 percent), elevated serum creatinine (57 percent), lymphopenia (53 percent), hypophosphatemia (49 percent), thrombocytopenia (40 percent), elevated AST (38 percent), and leukopenia (32 percent). Most common grades 3/4 adverse events included asthenia (11 percent), dyspnea (9 percent), hemoglobin decreased (20 percent),

lymphocytes decreased (16 percent), glucose increased (16 percent), phosphorus decreased (18 percent), and triglycerides increased (44 percent).

Strong inducers of CYP3A4/5 (e.g., dexamethasone, rifampin) and strong inhibitors of CYP3A4 (e.g., ketoconazole, atazanavir) may decrease and increase concentrations of the major metabolite of TORISEL, respectively. If alternatives cannot be used, dose modifications of Torisel are recommended. St. John's Wort may decrease Torisel plasma concentrations, and grapefruit juice may increase plasma concentrations of the major metabolite of Torisel, and therefore both should be avoided. The combination of Torisel and sunitinib resulted in dose-limiting toxicity (Grade 3/4 erythematous maculopapular rash, and gout/cellulitis requiring hospitalization).

Please see TORISEL full Prescribing Information at http://www.Torisel.com.

About Camptosar® (irinotecan HCI injection)

Camptosar is indicated as a component of 1st line therapy in combination with 5-FU/LV for the treatment of metastatic colorectal cancer. Camptosar is also indicated for patients with mCRC whose disease has recurred or progressed following initial FU-based therapy.

Important Camptosar® (irinotecan HCI injection) Safety Information

With Camptosar, both early and late forms of diarrhea can occur and may be life threatening. Late diarrhea should be managed promptly with loperamide and supportive care including antibiotics as needed. Camptosar can induce severe myelosuppression. Depending on the severity of neutropenia, dose delay, dose reduction, or use of a colony-stimulating factor should be considered.

Particular caution should be exercised in monitoring the effects of Camptosar in the elderly (>65), in patients who have previously received pelvic/abdominal irradiation, in patients with performance status of 2 or higher, and in patients known to be homozygous for the UGT1A1*28 allele. Rare cases of ileus, complicated colitis, or renal impairment have been observed. It is recommended that patients receive premedication with antiemetic agents. Prophylactic or therapeutic administration of atropine should be considered in patients experiences cholingeric symptoms. Provided intolerable toxicity does not develop, treatment with additional courses may be continued indefinitely as long as patients continue to experience clinical benefits. Thromboembolic events have been observed but the specific cause has not been determined. Camptosar should not be used in patients with severe bone marrow failure. Vaccination with a live vaccine should be avoided in patients receiving irinotecan due to the potential for serious fatal infections. In addition, patients with hereditary fructose intolerance should not be given Camptosar, as this product contains sorbitol.

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.

DISCLOSURE NOTICE: The information contained in this release is as of May 20, 2010. Pfizer assumes no obligation to update any 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 product candidates and potential additional indications for various in-line oncology products, 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 or supplemental drug applications that have been or may be filed for any such oncology product candidates or any such additional indications for in-line oncology products 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.

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[1] ASCO Accepted Abstract #3. Clinical Activity of the Oral ALK Inhibitor, PF-02341066, in ALK Positive Patients with Non-Small Cell Lung Cancer (NSCLC). Plenary Session, Sunday, June 6: 2:30pm. Yung-Jue Bang – Presenter. 2010 American Society of Clinical Oncology Annual Meeting. Chicago, IL. June 4 - 8, 2010.

[2] Horn L and Pao W. EML4-ALK: Honing in on a new target in non-small cell lung cancer. *Journal of Clinical Oncology*. 2009; 27: 4232-4235.

[3] Soda M, Choi YL, Enomoto M et al. Identification of the transforming EML4–ALK fusion gene in non-small-cell lung cancer. *Nature*. 2007; 448: 561- 567.

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

[5] Kwak E et al. Clinical Activity Observed in a Phase 1 Dose-Escalation Trial of an Oral MET and ALK Inhibito, PF-02341066. Accepted Oral Presentation at the American Society of Hematology 2009 Meeting, December 5-8, 2009. OR

[6] ClinicalTrials.gov. An Investigational Drug, PF-02341066 Is Being Studied Versus Standard Of Care In Patients With Advanced Non-Small Cell Lung Cancer With A Specific Gene Profile Involving The Anaplastic Lymphoma Kinase (ALK) Gene. 2010. Available at: http://clinicaltrials.gov/ct2/show/NCT00932893. Accessed May 7, 2010.

[7] ASCO Accepted Abstract #LBA 7523. Efficacy and safety of PF299804 versus (v) erlotinib (E): a global, randomized phase (P) 2 trial in patients (pts) with advanced non-small cell lung cancer (NSCLC) after failure of chemotherapy (CT). Poster Discussion Session. Monday, June 7: 12:00pm. Michael J. Boyer – Presenter. 2010 American Society of Clinical Oncology Annual Meeting. Chicago, IL. June 4 – 8, 2010.

[8] ASCO Accepted Abstract # 7500. Randomized, Open Label, Phase III Trial of Figitumumab in Combination with Paclitaxel and Carboplatin versus Paclitaxel and Carboplatin in Patients with Non Small Cell Lung Cancer. Oral Presentation, Saturday, June 5: 8:00am. Jacek Jassem – Presenter. 2010 American Society of Clinical Oncology Annual Meeting. Chicago, IL. June 4 – 8, 2010.

[9]Cortes J et al. Efficacy and Safety of Bosutinib (SKI-606) in Patients with Chronic Phase (CP) Ph+ Chronic Myeloid Leukemia (CML) with Resistance or Intolerance to Imatinib. Poster Presented at the American Society of Hematology Meeting, December 6-9, 2008, San Francisco, CA.

[10] Konig H et al. Effects of Dasatinib on Src Kinase Activity and Downstream Intracellular Signaling in Primitive Chronic Myelogenous Leukemia Hematopoietic Cells. Cancer Research. 2008; 68: 9624-9633.

[11] ASCO Accepted Abstract # 6502. Safety and efficacy of bosutinib (SKI-606) in patients (pts) with chronic phase (CP) chronic myeloid leukemia (CML) following resistance or intolerance to imatinib (IM). Oral Presentation, Monday, June 7: 10:00am. Jorge Cortes - Presenter. 2010 American Society of Clinical Oncology Annual Meeting. Chicago, IL. June 4 – 8, 2010.

[12] ASCO Accepted Abstract # 4501. Gene Discovery Study: Identification of Prognostic Genomic Markers in Patients with Localized Clear Cell Renal Cell Carcinoma (ccRCC). Oral Presentation. Saturday, June 5: 8:15am. Brian Rini - Presenter. 2010 American Society of Clinical Oncology Annual Meeting. Chicago, IL. June 4 – 8, 2010.

[13] ClinicalTrials.gov. Sunitinib as Maintenance Therapy in Treating Patients With Stage III or Stage IV Non-Small Cell Lung Cancer Previously Treated With Combination Chemotherapy. Available at: http://clinicaltrials.gov/ct2/show/NCT00693992?term=sunitinib&cond=NSCLC&phase=2&rank=1. Accessed May 12, 2010.

[14] ClinicalTrials.gov. Sunitinib Plus Prednisone In Patients With Metastatic Castration-Resistant Prostate Cancer After Failure Of Docetaxel Chemotherapy (SUN 1120). Available at: http://clinicaltrials.gov/ct2/show/NCT00676650?term=Sunitinib&cond=prostate+cancer&lead=Pfizer&rank=2. Accessed on May 11, 2010.

[15] Sutent Clinical trials. A Clinical Trial Comparing Efficacy And Safety Of Sunitinib Versus Placebo For TheTreatment Of Patients At High Risk Of Recurrent Renal Cell Cancer (S-TRAC). Available at: http://clinicaltrials.gov/ct2/show/NCT00375674?term=Sunitinib&lead=Pfizer&phase=2&rank=6. Accessed May 11, 2010.

[16] ASCO Accepted Abstract # 4003. Patient-reported outcomes (PROs) in patients (pts) with pancreatic neuroendocrine tumors (NET) receiving sunitinib (SU) in a phase III trial. Clinical Science Symposium, Saturday, June 5: 2:15pm. Aaron Vinik - Presenter. 2010 American Society of Clinical Oncology Annual Meeting. Chicago, IL. June 4 – 8, 2010.

[17] ASCO Accepted Abstract # 4000. Updated safety and efficacy results of the phase III trial of sunitinib (SU) vs placebo (PBO) for treatment of pancreatic neuroendocrine tumors (NET). Clinical Science Symposium, Saturday, June 5: 1:15pm. Patricia Niccoli - Presenter. 2010 American Society of Clinical Oncology Annual Meeting. Chicago, IL. June 4 – 8, 2010.

[18] ASCO Accepted Abstract # LBA1010. Sunitinib in combination with docetaxel vs docetaxel alone for the first-line treatment of advanced breast cancer. Oral Presentation, Tuesday, June 8: 10:00am. Jonas Bergh - Presenter. 2010 American Society of Clinical Oncology Annual Meeting. Chicago, IL. June 4 – 8, 2010.

[19] ASCO Accepted Abstract # LBA1011. Phase III Evaluation of Sunitinib in Combination with Capecitabine vs Capecitabine in Previously Treated Advanced Breast Cancer. Oral Presentation, Tuesday, June 8: 10:15am. John Crown - Presenter. 2010 American Society of Clinical Oncology Annual Meeting. Chicago, IL. June 4 – 8, 2010.

[20] ASCO Accepted Abstract # 2014. Phase II Trial of PF-04948568 (CDX-110) in Combination with Temozolomide (TMZ) in Patients (pts) with Glioblastoma (GBM). Oral Presentation. Saturday, June 5: 12:00pm. Rose Lai - Presenter. 2010 American Society of Clinical Oncology Annual Meeting. Chicago, IL. June 4 – 8, 2010.

[21] ASCO Accepted Abstract # 3028. Final report of phase 1 clinical, pharmacokinetic (PK), pharmacodynamic (PD) study of PF-00562271 targeting focal adhesion kinase (FAK) in patients (pts) with solid tumors. Poster Discussion Session, Sunday, June 6: 5:00pm. Neesha C. Dhani - Presenter. 2010 American Society of Clinical Oncology Annual Meeting. Chicago, IL. June 4 – 8, 2010.

[22] ASCO Accepted Abstract # 3033. Pharmacodynamically guided dose selection of PF-00337210, a VEGFR2 tyrosine kinase (TK) inhibitor, in a phase 1 study. Poster Discussion Session, Sunday, June 6: 5:00pm. Glenn Liu - Presenter. 2010 American Society of Clinical Oncology Annual Meeting. Chicago, IL. June 4 – 8, 2010.
[23] ASCO Accepted Abstract # 2524. First-in-human dose-escalation safety and PK trial of a novel intravenous humanized monoclonal CovX body inhibiting angiopoietin 2. Poster Discussion Session, Saturday, June 5: 12:00pm. Lee Rosen - Presenter. 2010 American Society of Clinical Oncology Annual Meeting. Chicago, IL. June 4 – 8, 2010.
[24] ASCO Accepted Abstract # TPS235. Axitinib ± dose titration as first-line therapy for metastatic renal cell carcinoma (mRCC). Trials in Progress Poster, Monday, June 7: 8:00am. Eric Jonasch - Presenter. 2010 American Society of Clinical Oncology Annual Meeting. Chicago, IL. June 4 – 8, 2010.
[25] ASCO Accepted Abstract # 4631. Serum Lactate Dehydrogenase (LDH) is a Predictive Biomarker for mTOR Inhibition in Patients with Metastatic Renal Cell Carcinoma (RCC). General Poster Session, Monday, June 7: 1:00pm. Andrew J. Armstrong - Presenter. 2010 American Society of Clinical Oncology Annual Meeting. Chicago, IL. June 4 – 8, 2010.
[26] ASCO Accepted Abstract # 3504. Molecular and clinical determinants of survival following relapse after curative treatment of stage II-III colon cancer (CC). Results of the translational study on the PETACC 3 - EORTC 40993 -SAKK 60-00 trial. Oral Presentation. Sunday, June 6: 10:45am. Arnaud Roth - Presenter. 2010 American Society of Clinical Oncology Annual Meeting. Chicago, IL. June 4 – 8, 2010.
[27] ASCO Accepted Abstract # 2574. Clinical outcome of first-line melanoma patients who continue tremelimumab in spite of early disease progression. General Poster Session, Monday, June 7: 8:00am. Diane I. Healey- Presenter. 2010 American Society of Clinical Oncology Annual Meeting. Chicago, IL. June 4 – 8, 2010.
[28] ASCO Accepted Abstract # 4131. A phase I dose escalation trial of CP-675206 (tremelimumab) in combination with gemcitabine in patients with chemotherapy-naive metastatic pancreatic cancer. General Poster Session, Sunday, June 6: 2:00pm. Massimo Aglietta - Presenter. 2010 American Society of Clinical Oncology Annual Meeting. Chicago, IL. June 4 – 8, 2010.