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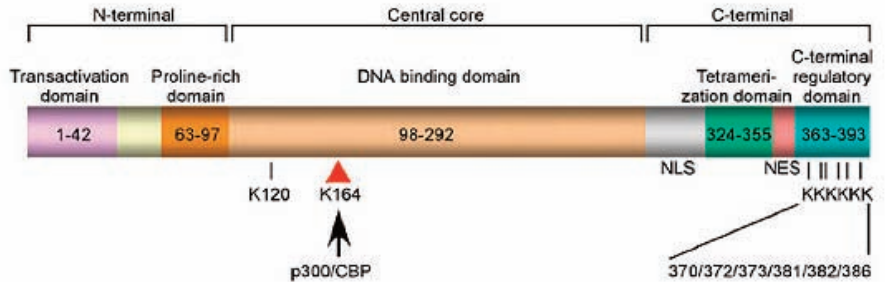
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INTELLIGENT INSIGHTS. SMART RESULTS.

In the Spotlight:

Acetylation is Indispensable for p53 Activation

Tumor suppressor p53 is a key regulator of various types of stress including DNA damage response. Increase in p53 protein level in response to various types of stress induces growth arrest/apoptosis by activating the [Read more...](#)



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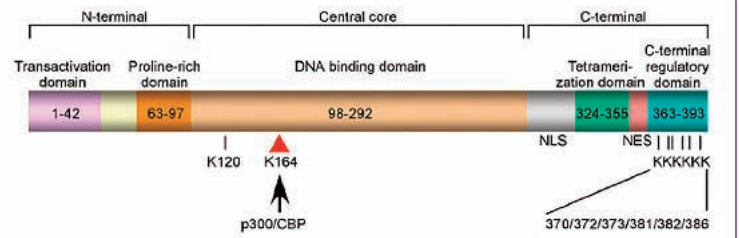


Spotlight Report

Acetylation is Indispensable for p53 Activation

Tumor suppressor p53 is a key regulator of various types of stress including DNA damage response. Increase in p53 protein level in response to various types of stress induces growth arrest/apoptosis by activating the transcription of a number of genes. p53 is regulated by a variety of post-translational modifications such as phosphorylation, acetylation, methylation, ubiquitination, etc. Ubiquitin-mediated proteosomal degradation plays a critical role in regulating p53 levels. However, the mechanism by which transcriptional function of p53 is activated is not understood and whether post-translational modifications are required for p53 activation is still under debate. The functions of p53 are inhibited by the mdm2 oncoprotein and p53 can only be activated if its interaction with mdm2 is abolished. One of the key transcriptional targets of p53 required for p53-mediated growth arrest is p21.

Identification of K164 within the Human p53 DNA-Binding Domain as a Novel Acetylation Site



Cell, 133, May 16, 2008

In this study, Tang Y and colleagues have explored the role of acetylation in the activation of p53. They have first shown that in addition to the 7 previously identified acetylation sites, p53 is also acetylated at lysine at position 164 (K164) within the core DNA binding domain. The authors established tet-inducible cell lines expressing wild type p53 or acetylation defective mutant (8KR) of p53 and demonstrated that loss of p53 acetylation abrogates p53-mediated growth arrest and apoptosis. Further, it was shown that p53 acetylation represses recruitment of mdm2 to the p21 promoter. Loss of acetylation at each individual site can be compensated by the modification of other sites. However, loss of acetylation at all sites completely abolished p53-mediated transactivation of p21. These studies clearly demonstrated that acetylation destabilizes mdm2-p53 interaction and is indispensable for p53-mediated activation of p21 and subsequent growth arrest.

Source: [Cell](#)



Business News

BMS to Acquire Kosan Biosciences

Bristol-Myers Squibb (BMS) has signed a deal to buy Kosan Biosciences, a cancer therapeutics company, for \$5.50 per share in cash. The transaction represents a net aggregate purchase price of approximately \$190 million after deducting Kosan's projected net cash. The acquisition of Kosan will enhance BMS's product pipeline with two important classes of anticancer agents: novel Hsp90 (heat shock protein 90) inhibitors and epothilones. The Hsp90 program includes a Phase III compound for the treatment of patients with multiple myeloma. Epothilones are microtubule stabilizers with multiple therapeutic applications in various cancers and potentially in neurodegenerative diseases.



Business (cont'd)

BMS and Kosan also announced that they have entered into a separate license agreement under which Kosan has granted BMS an exclusive worldwide license to its epothilone compounds and related intellectual property data. Kosan will assign to BMS its epothilone investigational new drug applications. Under the license agreement, Kosan will receive an initial payment of \$25 million and is entitled to milestone payments in connection with the development of epothilone product candidates.

Source: Bristol-Myers Squibb

Alnylam Joins Takeda in RNAi Deal

Alnylam Pharmaceuticals and Takeda have formed a strategic platform alliance in RNA Interference (RNAi) therapeutics in the fields of oncology and metabolic disease with the option to expand to additional therapeutic areas. This landmark alliance is the first major RNAi therapeutics partnership between a Japanese pharmaceutical company and a US biotechnology company, representing a new frontier in the advancement of RNAi therapeutics to patients on a global basis. The partnership includes \$100 million in upfront payments and \$50 million in near-term technology transfer payments for a non-exclusive license in two therapeutic fields. It is valued at potentially over \$1 billion in future research and development upon successful commercialization of multiple products.

With this collaboration, Takeda becomes Alnylam's strategic partner for RNAi therapeutics over a five-year period and the only Asian company to obtain a right of first negotiation to develop and commercialize Alnylam RNAi therapeutic development programs for the Asian market. In addition, Alnylam obtains options to co-develop and co-commercialize Takeda RNAi therapeutic programs in the US market on a 50-50 basis.

Source: Takeda, Alnylam Pharmaceuticals

Daiichi Sankyo Acquires U3 Pharma AG

Daiichi Sankyo has entered into an agreement to acquire the privately held firm, U3 Pharma AG, a German biotechnology company focusing on research into antibodies for the treatment of cancer. Daiichi Sankyo will purchase 100% of the stocks and make a one-time payment of €150 million (\$235 million) for the company.

U3 Pharma's pipeline of novel targeted therapeutics includes programs focusing on fully-human antibodies as potential therapies for breast, lung and colorectal cancers, among others. The company's lead product is U3-1287 (AMG 888), the first fully-human anti-HER3 monoclonal antibody to inhibit oncogenic signaling and tumor proliferation. Daiichi Sankyo's current novel therapeutics portfolio includes CS-1008, an oncologic agent in Phase II for malignant neoplasms. For commercialization only in Japan, Daiichi Sankyo has the rights to market denosumab (AMG 162), which is currently in Phase III for osteoporosis as well as for bone metastases in patients with advanced breast cancer. Daiichi Sankyo also has the exclusive rights in Japan to develop and market nimotuzumab (DE 766), an oncologic agent in Phase I to treat advanced solid malignancies.

Source: Daiichi Sankyo

Cancer Research UK and AstraZeneca Announce Innovative Deal

Cancer Research UK announced an innovative arrangement to progress into clinical development of AZD0424, a potential anti-cancer compound from AstraZeneca. AZD0424, a tyrosine kinase inhibitor, is the first drug to enter the Clinical Development Partnerships (CDP) programme. CDP was launched in 2006 to support the continuing development of promising anti-cancer agents. The compound is expected to enter Phase I trials within the next 18 months. Under the terms of the partnership deal, Cancer Research UK's highly experienced Drug Development Office will conduct the clinical trials at no cost to AstraZeneca. In addition, AstraZeneca retains the option to assume further development and marketing of the drug, with the Cancer Research UK receiving a share of any revenues.

Source: Cancer Research UK



Business (cont'd)

Five Prime and Pfizer Collaborate in Cancer Research

Five Prime Therapeutics and Pfizer have announced the initiation of a worldwide collaborative research and license agreement focusing on the discovery of antibody targets and novel therapeutic protein products to treat certain areas of cancer and diabetes. Under the collaboration, Five Prime will screen its comprehensive protein library in both cell-based assays and primary *in vivo* screens directed toward finding potential therapeutic protein products and antibody targets. Upon the commencement of the collaboration, Five Prime will receive an up-front payment and an equity investment from Pfizer and three years of committed research funding. Pfizer will have exclusive worldwide rights to develop and commercialize certain products and targets discovered during the research term, in exchange for future milestones and royalties.

Source: [Pfizer](#)



Research Highlights

Superoxidase Dismutase is a Regulator of Growth Factor Signaling

Superoxidase dismutase 1 (SOD1) is a copper/zinc enzyme that converts toxic superoxide into hydrogen peroxide and molecular oxygen. SOD1 knock-out mice exhibit increased susceptibility to liver tumors and SOD1 knock-down by siRNA has been shown to induce senescence in fibroblasts. Tetrathiomolybdate (ATN-224) is an inhibitor of SOD1 that attenuates growth factor (GF) – mediated phosphorylation of ERK1/2 in endothelial cells and is currently being evaluated in Phase II trials in cancer patients. The precise mechanism by which inhibition of SOD1 abolishes ERK phosphorylation is not understood. In a study published in *PNAS*, Juarez Jose and colleagues have shown that GF signaling leads to an increase in reactive oxygen species (ROS) which inactivates protein tyrosine phosphatases (PTPs) by oxidizing an essential cysteine residue in the active site. Inhibition of SOD1 thus protects PTPs from oxidation by preventing the formation of H₂O₂. This in turn inhibits ERK phosphorylation in cells stimulated with growth factors. These studies support the hypothesis that SOD1 is a master regulator of GF signaling and a therapeutic target for cancer since inhibition of SOD1 results in the down-regulation of multiple signaling pathways important for endothelial and tumor cell functions.

Source: [PNAS](#)



Research Highlights (cont'd)

Screening for Antigens Overexpressed in Carcinomas

Several humanized murine monoclonal antibodies (mAbs) became useful therapeutic agents against a few malignancies. These include alemtuzumab against CD52, trastuzumab against HER2, and rituximab against CD20 for treatment of CLL, breast cancer, and non-Hodgkins lymphoma, respectively. However, for majority of the human cancers useful therapeutic Abs are not yet available because of lack of knowledge of which antigens (Ags) are likely to become useful targets. In the study published in *PNAS*, Kurosawaa and colleagues used a phage-display antibody library and isolated a large number of human mAbs that bind to the surface of tumor cells. They were individually screened by immunostaining, and clones that preferentially and strongly stained the malignant cells were chosen. The Ags recognized by those clones were isolated by immunoprecipitation. Finally mAbs were converted to complete human IgG1 and the antitumor activity was examined. This procedure enabled the group to succeed in comprehensive identification of tumor-associated Ags (TAAs) and simultaneous isolation of mAbs against them. Investigators isolated 2,114 mAbs with unique sequences and identified 21 distinct Ags highly expressed on several carcinomas. The mAbs converted to IgG1 revealed effective Ab-dependent cell-mediated cytotoxicity as well as anti tumor activity *in vivo*. As half of the 21 Ags showed distinct tumor-specific expression pattern and the mAbs isolated showed various characteristics with strong affinity to the Ag, it is likely that some of the Ags detected will become useful targets for the corresponding carcinoma therapy and that several mAbs will become therapeutic agents.

Source: *PNAS*

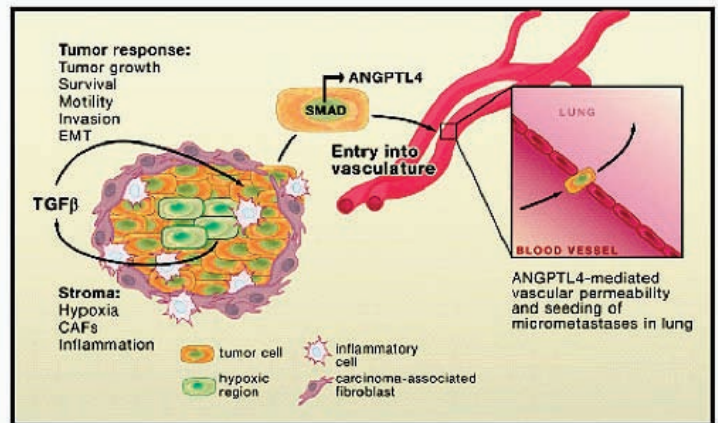
TGFβ Primes Breast Tumor Cells for Metastasis

Metastasis is a leading cause of mortality for cancer patients, yet the molecular mechanisms underlying metastasis are poorly understood. Particular tumor types have a propensity to metastasize to certain organs; for example, breast tumor cells tend to metastasize to lungs, bones, brain, and liver. In a study published in *Cell*, Padua et al. have shown that cytokine TGFβ in the breast tumor microenvironment primes tumor cells for lung metastasis by driving expression of the adipokine angiopoietin-like 4 (*ANGPTL4*) via Smad signaling pathway. Investigators observed that TGFβ activity in primary breast tumors is associated with an increased propensity of these patients to develop lung metastasis but not bone metastasis. This phenomenon

implies a biologically selective TGFβ-dependent mechanism that favors tumor targeting of the lungs. Tumor cell-derived Angptl4 disrupts the integrity of vascular endothelial cell layers both *in vitro* and in the lungs, increasing the permeability of lung capillaries, thus facilitating the passage of breast cancer cells. This evidence suggests that Angptl4 is a clinically relevant mediator of lung metastasis in breast cancer. Thus, the TGFβ-Angptl4 cytokine relay system provides an example of how stimuli in the primary tumor can affect distant metastases.

Source: *Cell*

TGFβ Influences Tumor Growth and Metastasis



Cell, 133, April 4, 2008



Research Highlights (cont'd)

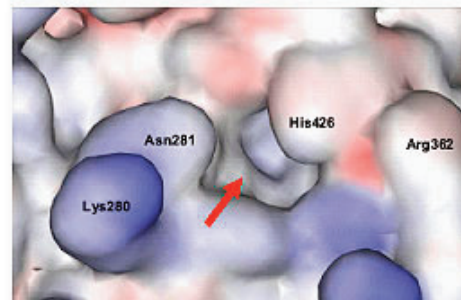
Specific Inhibitors of the Protein Tyrosine Phosphatase Shp2

The protein tyrosine phosphatase (PTP) Shp2 is a positive regulator of growth factor signaling. Distinct somatic gain-of-function mutations in several types of leukemia define Shp2 as a bonafide oncogene. The presence of activated or up-regulated Shp2 protein in human cancers and other disease makes Shp2 an excellent target for drug discovery.

In a recent study published in *PNAS*, Klaus Hellmuth and colleagues, used a high-throughput *in silico* screening procedure to identify phenylhydrazonopyrazolone sulfonate, PHP51, as a cell permeable inhibitor. PHP51 is highly specific for Shp2 over the closely related tyrosine phosphatases Shp1 and PTP1B. PHP51 inhibited hepatocyte growth factor/scatter factor (HGF/SF) induced scattering and branching morphogenesis. PHP51 also inhibited Shp2-dependent signaling of the Erk1/2 MAP kinases and dephosphorylation of paxillin. Hyperactivation of the Ras/MAP kinase pathway mediated by expression of the leukemic oncogene SHP2-E76K was also inhibited by PHP51. In addition, PHP51 efficiently blocked proliferation and anchorage-independent growth of various human tumor cells. It was not cytotoxic against normal epithelial cells. This specificity, together with its activity toward tumor cell line growth, its simple chemical structure, and excellent pharmacological properties, make this class of compounds suitable for further development for the treatment of hp2-dependent diseases.

Source: *PNAS*

Homology model of the active site of Shp2



PNAS, 105, May 20, 2008

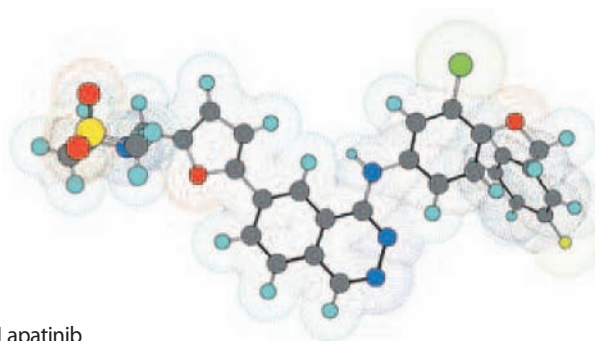


Clinical Development

Tykerb-Herceptin Combo Reduces Disease Progression

GlaxoSmithKline (GSK) announced positive data from the first-ever randomized, multi-centre, open label Phase III trial of the combination of two targeted agents, Tykerb (lapatinib) and Herceptin (trastuzumab), in women with HER2-positive metastatic breast cancer. GSK Oncology will present this data at the 2008 annual American Society of Clinical Oncology (ASCO) meeting in Chicago.

In this study, 296 patients with HER2 positive breast cancer who had documented progression on trastuzumab treatment in the metastatic setting were eligible to be randomized to receive lapatinib (1000 mg QD) plus trastuzumab (2 mg/kg weekly after 4 mg/kg loading dose) or lapatinib alone (1500 mg QD). Patients were heavily pre-treated and had received a median of six prior anti-cancer regimens. Patients had also received a



Lapatinib

Nature Reviews Drug Discovery, 6, June 2007



Clinical Development (cont'd)

median of three prior lines of trastuzumab. Despite receiving multiple prior lines of anti-cancer therapy, patients who received lapatinib plus trastuzumab experienced a statistically significant increase in median progression-free survival versus lapatinib alone (12 weeks vs. 8.1 weeks). A 27% reduction in the risk of disease progression was noted with combination therapy. In addition, the combo yielded a response rate of 10.3% vs. 6.9% with Tykerb alone. Using the two therapies together also doubled the overall clinical benefit rate vs. Tykerb alone (24.7% vs. 12.4%).

Source: *GSK*

Docetaxel-Based Chemotherapy has an Edge in Node Negative Breast Cancer

Sanofi-aventis and GEICAM announced that for women with high-risk node-negative early stage breast cancer, adjuvant treatment with Taxotere® (docetaxel) Injection Concentrate as part of the TAC regimen (Taxotere®, doxorubicin, cyclophosphamide) was associated with a significant improvement in disease free survival (DFS) compared to a standard FAC regimen (5-fluorouracil, doxorubicin, cyclophosphamide) in the GEICAM 9805/Target-0 study. The 1,059 women enrolled in this multicenter, Phase III study were randomized to receive either TAC (n=539) or FAC (n=520) after surgical resection of their tumors. The study showed a significant improvement in DFS in the TAC arm over the FAC arm, with 91% and 86% patients, respectively, alive and disease free at 5 years. However, the overall survival (OS) data was immature; estimated 5-year OS was 97% for TAC and 95% for FAC. Furthermore, TAC produced significantly more hematological adverse reactions than FAC.

Source: *Sanofi-aventis*

Sanofi, Regeneron Drug Misses Primary Endpoint

Sanofi-aventis and Regeneron Pharmaceuticals reported results of a randomized, double-blind, Phase II study of 215 women with advanced ovarian cancer who were treated with aflibercept at a dose of 2 or 4 mg/kg every two weeks. Clinical investigators as well as an independent review committee (IRC) assessed the response to treatment. As assessed by the investigators, RECIST (Response Evaluation Criteria in Solid Tumors) response rates were 7.3% with the 4 mg/kg dose and 3.8% with the 2 mg/kg dose. However, as assessed by the IRC, patients achieved a response rate according to RECIST criteria of 4.6% in the 4 mg/kg arm and 0.9% in the 2 mg/kg arm. Thus, the study did not achieve its primary endpoint of demonstrating a response rate significantly greater than 5%, as assessed by an IRC.

CA-125 response, a key secondary endpoint defined as at least a 50% reduction in CA-125 protein levels, were 11.6% in the evaluable patients treated with 4 mg/kg and 11.5% in the evaluable patients treated with 2 mg/kg. Eighteen (13.8%) of the 130 CA-125 response-evaluable patients from both dose groups had either a RECIST (as assessed by the IRC) or CA-125 response. In the entire study population, as assessed by the IRC, median progression-free survival was 13.3 and 13.0 weeks with the 4 mg/kg and 2 mg/kg doses, respectively. Sanofi and Regeneron said that the study has shown some promising signs of effectiveness and they were continuing to evaluate the data from the trial to determine the next steps for aflibercept in advanced ovarian cancer.

Source: *Sanofi-aventis*

IDM Pharma Updates Data on Mifamurtide

IDM Pharma presented the updated data from a compassionate access program evaluating mifamurtide (L-MTP-PE) at the 21st Annual Meeting of The American Society of Pediatric Hematology/Oncology. The compassionate access program provides L-MTP-PE treatment to patients who have either failed or cannot tolerate treatment with existing therapies compared to the Phase III pivotal study, which evaluated newly diagnosed patients.

The study enrolled 29 high-risk osteosarcoma patients (ages 10 - 21), out of which 27 have been treated with L-MTP-PE in the compassionate access program. L-MTP-PE (2 mg/m² IV over 1 hour) was administered twice a week for 12 weeks



Clinical Development (cont'd)

followed by once a week for 24 weeks. In addition, some patients in the program were also treated with other agents including aerosol recombinant granulocyte monocyte colony stimulating factor (GM-CSF) an immune stimulating agent (n=20), ifosfamide (n=4), and/or gemcitabine (n=2). Updated results show that nine patients are alive with disease, nine patients have no evidence of disease and nine patients have died. There are two patients for whom the status is unknown. Treatment with L-MTP-PE combined with other agents including aerosol GM-CSF was generally well tolerated. Patients treated with chemotherapy had no unexpected toxicities. The data showed that L-MTP-PE in combination with other therapies is safe, well-tolerated and exhibited signs of disease control.

Source: *IDM Pharma*



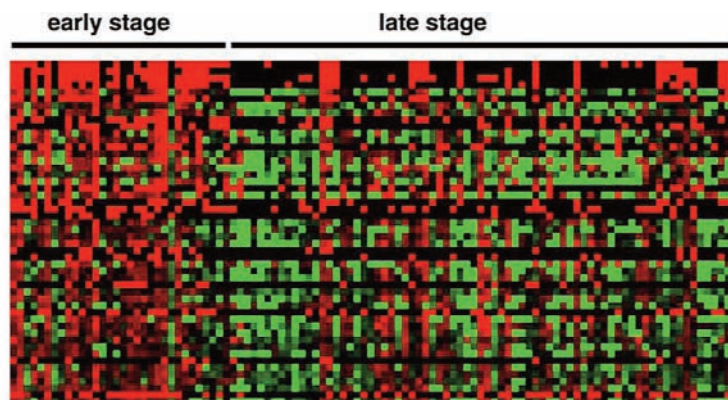
Biomarkers

MicroRNA Expression as Biomarker in EOC

MicroRNAs (miRNAs) are noncoding small RNAs, which negatively regulate gene expression. In human cancer, miRNAs might function as either oncogenes or tumor suppressor genes and their global expression is deregulated in most cancer types. Epithelial ovarian cancer (EOC) is the most common ovarian malignancy. In a study published in *PNAS*, Lin Zhanga and colleagues used integrative genomic approaches to perform a comprehensive analysis of miRNome alterations associated with malignant transformation of the ovarian surface epithelium (OSE) and/or ovarian tumor stage progression. They identified eight miRNAs located in the chromosome 14 miRNA cluster (*Dlk1-Gtl2* domain), as potential tumor suppressor genes in EOC cell lines and invasive breast and colon cancer. They were silenced by epigenetic mechanisms, and they were predicted to up-regulate a large number of mRNA transcripts in late stage EOC. Some of these miRNAs were also down-regulated in bladder cancer. Importantly, tumors with lower expression of these eight miRNAs were associated with higher proliferation index and significantly shorter survival. These results suggest that miRNAs may offer new biomarkers and therapeutic targets in EOC, but further work is required to understand their potential for cancer therapy.

Source: *PNAS*

Numerous miRNAs are down-regulated in late-stage or high-grade ovarian cancer



PNAS, 105, May 13, 2008



Biomarkers (cont'd)

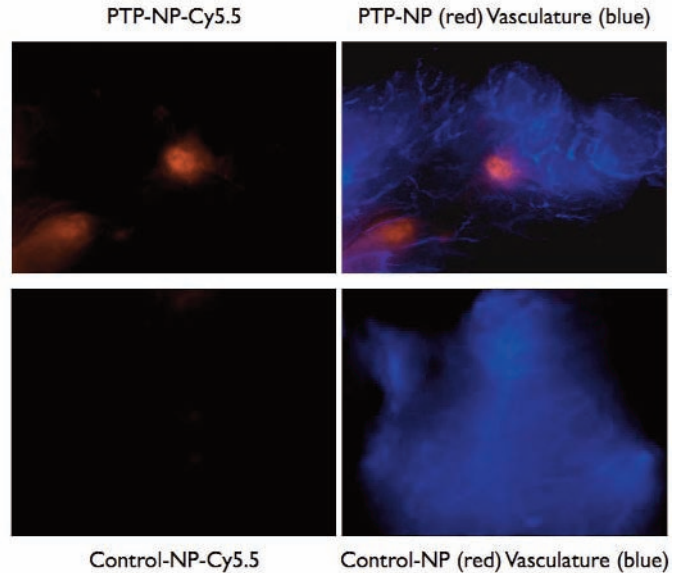
Early Detection of Pancreatic Ductal Adenocarcinoma

There is an urgent need for the early diagnosis of pancreatic ductal adenocarcinoma (PDAC), as most cases are currently diagnosed at a metastatic stage exhibiting profound resistance to existing therapies. In a recent study published in *PLoS Medicine*, Kelly et al. designed a study to identify novel molecular markers and to test their potential as targeted imaging agents.

Using phage display approach in primary cell lines derived from mouse model of PDAC, investigators screened for peptides that specifically bind to cell surface antigens on PDAC cells. These screens identified membrane-localized plectin-1 as a potential new biomarker for PDAC. Further studies confirmed that plectin-1 is present in the membrane of both murine and human pancreatic carcinoma cells, whereas in normal cells it is confined to the nucleus and cytoplasm.

Investigators then chemically synthesized the plectin-1 targeted peptides and attached it to a magnetofluorescent nanoparticle for tracking its location both optically and with MRI. When this nanoconstruct was injected into the mouse model, imaging revealed that it localized to the small, early lesions that were present at that stage. The approaches described here may be broadly applicable to the discovery of cancer biomarkers predictive of disease stage, prognosis, or presence of specific genetic alterations.

Source: *PLoS Medicine*



PLoS Medicine, 5, April 2008

deCODE Discovers Genetic Link Between Pigmentation Traits and Risk of Skin Cancer

deCODE scientists reported new findings in the genetics of pigmentation traits in people of European descent. In the first study published in online edition of *Nature Genetics*¹, Patrick Sulem et al. utilized genomic analysis of nearly 8,500 Icelandic and Dutch participants to identify two coding variants in *TPCN2* that were associated with hair color, and a variant at the *ASIP* locus which showed strong association with skin sensitivity to sun, freckling and red hair, phenotypic characteristics. In the second study published in online edition of *Nature Genetics*², Gudbjartsson et al. investigated whether the variants linked to pigmentation traits are also associated with risk of cutaneous melanoma (CM) and basal cell carcinoma (BCC). This study comprised 2,121 individuals with CM, 2,163 individuals with BCC and over 40,000 controls. A haplotype near *ASIP*, known to affect a similar spectrum of pigmentation traits as *MC1R* variants, conferred significant risk of CM and BCC. The variant in *TYR* encoding the R402Q amino acid substitution, previously shown to affect eye color and tanning response, conferred risk of CM and BCC. An eye color variant in *TYRP1* was also associated with risk of CM. "These findings may be useful for helping individuals to better gauge their susceptibility to skin cancer, and we are therefore very pleased to be including these variants in our deCODEme™ service," said Kari Stefansson, CEO of deCODE.

Source: *deCODE, Nature Genetics*¹, *Nature Genetics*²



Regulatory

Sanofi-aventis Gets FDA Approval for Oxaliplatin sNDA

Sanofi-aventis announced that the US FDA has approved the supplemental new drug application (sNDA) to include six-year overall survival analysis from the MOSAIC trial in the Eloxatin® (oxaliplatin injection) prescribing information (PI).

The new PI also reports five-year disease free survival (DFS) data in Stage III colon cancer patients treated following surgery to remove the primary tumor. The MOSAIC trial results showed that after a median follow-up of six years, Stage III colon cancer patients treated with FOLFOX4 (Eloxatin + 5-Fluorouracil/Leucovorin) had a 20% reduction in the risk of death compared to those treated with standard chemotherapy alone. In addition, stage III patients treated with the Eloxatin-based regimen at 5 years were 22% less likely to relapse or risk of disease recurrence after 77 month follow-up. "Inclusion of these survival results in the new US Eloxatin® PI marks an important milestone in the treatment of colon cancer," said principal investigator Dr. de Gramont.

Source: Sanofi-aventis

FDA Clears Calypso® Medical's GPS for Prostatectomy Patients

Calypso® Medical announced that the US FDA cleared a new indication for the use of implantable Beacon® electromagnetic transponders with the Calypso System in external beam radiation therapy. Known as GPS for the Body®, the Calypso System utilizes transponders to setup and continuously track the position of targeted tissue during radiation treatment. In the event that the tumor site moves outside of acceptable limits, the clinician adapts therapy during daily treatment to ensure the treatment is delivered as prescribed to the cancerous tissue while avoiding adjacent healthy organs. Calypso® Medical claims that this is the only technology platform designed to provide objective, accurate and continuous tracking information during external beam radiation therapy without adding ionizing radiation. Previously, GPS for the Body® technology was cleared solely for use in patients with an intact prostate.

Source: Calypso Medical

The E-Newsletter Team:

Dr. Chandra Kumar, Dr. Anuradha Dhingra, Ms. Meenu Grover, Dr. Neetu Singhal, Ms. Sarika Manchanda