• Used as single agents or in combination with other anti-cancer treatments like chemotherapy, biological therapy, or radiation.

Advantages: Targeting the PH domain improves specificity against Akt kinase in comparison to inhibitors of the ATP domain which typically are unspecific.

Inventors: Phillip A. Dennis (NCI) *et al.*

Relevant Publications

1. Memmott RM, Gills JJ, Hollingshead M, Powers MC, Chen Z, Kemp B, Kozikowski A, Dennis PA. Phosphatidylinositol ether lipid analogues induce AMP-activated protein kinase-dependent death in LKB1-mutant non small cell lung cancer cells. Cancer Res. 2008 Jan 15;68(2):580–588. [*PubMed:* 18199555.]

2. Gills JJ, Castillo SS, Zhang C, Petukhov PA, Memmott RM, Hollingshead M, Warfel N, Han J, Kozikowski AP, Dennis PA. Phosphatidylinositol ether lipid analogues that inhibit AKT also independently activate the stress kinase, p38alpha, through MKK3/6-independent and -dependent mechanisms. J Biol Chem. 2007 Sep 14;282(37):27020–27029. [*PubMed:* 17631503.]

3. Gills JJ, Holbeck S, Hollingshead M, Hewitt SM, Kozikowski AP, Dennis PA. Spectrum of activity and molecular correlates of response to phosphatidylinositol ether lipid analogues, novel lipid-based inhibitors of Akt. Mol Cancer Ther. 2006 Mar;5(3):713–722. [*PubMed:* 16546986.]

4. Carón RW, Yacoub A, Li M, Zhu X, Mitchell C, Hong Y, Hawkins W, Sasazuki T, Shirasawa S, Kozikowski AP, Dennis PA, Hagan MP, Grant S, Dent P. Activated forms of H–RAS and K–RAS differentially regulate membrane association of PI3K, PDK–1, and AKT and the effect of therapeutic kinase inhibitors on cell survival. Mol Cancer Ther. 2005 Feb;4(2):257–270. [*PubMed:* 15713897.]

5. Castillo SS, Brognard J, Petukhov PA, Zhang C, Tsurutani J, Granville CA, Li M, Jung M, West KA, Gills JG, Kozikowski AP, Dennis PA. Preferential inhibition of Akt and killing of Akt-dependent cancer cells by rationally designed phosphatidylinositol ether lipid analogues. Cancer Res. 2004 Apr 15;64(8):2782–2792. [*PubMed:* 15087394.]

6. Kozikowski AP, Sun H, Brognard J, Dennis PA. Novel PI analogues selectively block activation of the pro-survival serine/threonine kinase Akt. J Am Chem Soc. 2003 Feb 5;125(5):1144–1145. [*PubMed:* 12553797.]

Patent Status: U.S. Patent No. 7,378,403 issued 27 May 2008 (HHS Reference No. E–245–2002/0–US–03), and related international filings.

Licensing Status: Available for licensing.

Licensing Contact: Surekha Vathyam, PhD; 301–435–4076; *vathyams@mail.nih.gov.*

Dated: March 10, 2010.

Richard U. Rodriguez,

Director, Division of Technology Development and Transfer, Office of Technology Transfer, National Institutes of Health.

[FR Doc. 2010–5764 Filed 3–16–10; 8:45 am]

BILLING CODE 4140–01–P

DEPARTMENT OF HEALTH AND HUMAN SERVICES

National Institutes of Health

Government-Owned Inventions; Availability for Licensing

AGENCY: National Institutes of Health, Public Health Service, HHS.

ACTION: Notice.

SUMMARY: The inventions listed below are owned by an agency of the U.S. Government and are available for licensing in the U.S. in accordance with 35 U.S.C. 207 to achieve expeditious commercialization of results of federally-funded research and development. Foreign patent applications are filed on selected inventions to extend market coverage for companies and may also be available for licensing.

ADDRESSES: Licensing information and copies of the U.S. patent applications listed below may be obtained by writing to the indicated licensing contact at the Office of Technology Transfer, National Institutes of Health, 6011 Executive Boulevard, Suite 325, Rockville, Maryland 20852–3804; *telephone:* 301/496–7057; *fax:* 301/402–0220. A signed Confidential Disclosure Agreement will be required to receive copies of the patent applications.

Spontaneously Transformed Mouse Epithelial Cancer Cell Lines Serving as Mouse Models: A New Model for Cancer Research

Description of Invention: Investigators at the NIH have created a collection of 45 mouse epithelial cancer cell lines derived from six organs: Bladder, cervix, colon, lung, kidney, and mammary glands. These cells lines were obtained from spontaneously transformed primary cell cultures without genetic, viral or chemical manipulation so they can serve as mouse models for studying the natural process of oncogenesis.

The cell lines were characterized cytogenetically during their transformation from normal to spontaneously immortalization and were found to recapitulate many of the changes observed in human cancer cells such as the deregulation of oncogenes (Myc, Mdm2) and tumor suppressor genes (Cdnk4a/Ink4a/p16, Rb).

Carcinomas that arise from the epithelial cells lining organs lead to the most common cancers in humans. However, research on cellular transformation has largely relied on fibroblast cells which are not of epithelial origin and therefore, may not reflect the changes that lead to epithelial oncogenesis. The availability of these mouse epithelial cancer cell lines should allow for a more accurate analysis of this process.

Applications: These cell lines serve as "ideal" murine tumor models as they show evidence of progression, permitting analysis of the genetic and biological changes observed in the equivalent human carcinomas and associated with tumor progression. Their tumor histology is comparable to human cancers.

The cell lines have unique properties that make them suitable for study of the following:

• Unlimited replicative potential.

• Exhibit tumorigenic potential and EMT (Epithelial Mesencymal Transition).

• Exhibit high degree of chromosome instability (chromosome rearrangements, amplifications) in regions orthologous to those altered in human cancers.

• Use in mapping mouse genes homologous to human cancer genes and for the study of the effects of deregulation of cancer associated genes, through silencing or overexpression.

• For use in gene expression studies of tumor progression, comparing profiles to human cancers involving the same tissue types.

• Use as experimental controls in the analysis of oncogene signaling pathways.

• Use in the studying telomerase pathway regulation (200-fold expression difference between cell lines).

• Use of mouse as model of epithelial carcinomas and specifically cancers of the bladder, cervix, colon, lung, mammarys and kidney cancers.

• These mouse models serve as vehicles to test the efficacy of new therapies, targeting specific targets associated with the transformation of six different mouse epithelial tissues.

• Use for discovering drugs that alter the tumorigenic potential, invasiveness, and the Epithelial-Mesenchymal Transition state.

Advantages:

• Cytogenetically defined epithelial cell lines from mouse that model human carcinomas.

• Spontaneously transformed primary cell cultures were generated from isogenic mouse strain that has a low propensity for epithelial tumors in vivo therefore, not involving other mouse strains potentially influencing the genetic background.

• These cell lines were generated without viral, chemical or genetic manipulation and thus can serve as mouse models for studying the natural process of oncogenesis and as mouse models of human cancers.

• Genomically defined colon, bladder, and kidney cell lines showing oncogene deregulation (i.e. Mdm2 and Myc overexpression).

Development Status:

• Ready for use.

• Pre-clinical.

Market: Cancer is the second most common cause of death in the United States. More than half a million Americans are expected to die of cancer. The cell lines will serve as a valuable tool for cancer researchers.

Inventors: Hesed AM. Padilla-Nash *et al.* (NCI).

Publications: None currently available for this technology.

Patent Status: HHS Reference No. E–089–2010/0—Research Material. Patent protection is not being pursued for this technology.

Licensing Status: Available for licensing under a Biological Materials License Agreement.

Licensing Contact: Sabarni Chatterjee, PhD; 301–435–5587; *chatterjeesa@mail.nih.gov.*

Collaborative Research Opportunity: The National Cancer Institute, Cancer Genetics Branch, is seeking statements of capability or interest from parties interested in collaborative research to further develop, evaluate, or commercialize this technology. Please contact John Hewes, PhD at 301–435–3131 or *hewesj@mail.nih.gov* for more information.

Tumor Tissues Harboring Mutations in cAMP-Specific Phosphodiesterases Useful for the Study of Endocrine Tumors

Description of Invention: Researchers at the National Institute of Child Health and Human Development (NICHD), NIH, have made available samples of patient-derived adrenal and heart tumors that harbor genetic mutations that have been implicated in the predisposition of endocrine tumors. An endocrine tumor is a growth that affects the parts of the body that secrete hormones. Because an endocrine tumor arises from cells that produce hormones, the tumor itself can produce hormones and cause serious illness.

The tumor samples made available herein contain deletions in the cyclic nucleotide phosphodiesterase (PDE) PDE7A or PDE8B genes that impair PDE function and are characterized by high sensitivity to changes in cAMP levels. Commercially, phosphodiesterase inhibitors are widely used in the treatment of various disorders, including asthma, pulmonary hypertension, and erectile dysfunction, suggesting a potential utility for these tissues in a wide range of investigations.

Applications: Useful in the investigation of the mechanisms of phosphodiesterase inhibition.

Inventors: Constantine Stratakis et al. (NICHD).

Patent Status: HHS Reference No. E–059–2010/0—Research Tool. Patent protection is not being pursued for this technology.

Licensing Status: Available for licensing under a biological material license.

Licensing Contact: Patrick P. McCue, PhD; 301–435–5560; *mccuepat@mail.nih.gov.*

Collaborative Research Opportunity: The National Institute of Child Health and Human Development, Division of Intramural Research, is seeking statements of capability or interest from parties interested in collaborative research. Please contact Joseph Conrad, PhD at 301–435–3107 or *jmconrad@mail.nih.gov* for more information.

Akt-Ser473 Phosphorylation as a Marker for Predicting Taxane Chemotherapy Outcome

Description of Invention: Over the past decades, taxanes such as paclitaxel and docetaxel have emerged as effective chemotherapy agents for breast cancer and other malignancies. Taxanes are effective in many patients, however, not all patients benefit from this type of chemotherapy. A significant need remains for a means of predicting clinical outcome from taxane-based chemotherapy.

Akt, a serine/threonine kinase that can block apoptosis, has been implicated in the regulation of microtubule dynamics and organization. Akt phosphorylation and its transducing downstream events play a central role in cell survival and cell cycle progression at the G_2/M transition. Paclitaxel or docetaxel inhibits Akt-Ser473 phosphorylation (pAkt) and induces mitotic arrest. Therefore, taxanes may cause more damage to tumor cells that are dependent on pAkt for survival and cell cycle progression, significantly impacting treatment outcome.

Researchers at the National Cancer Institute, NIH, have identified pAkt as having predictive significance for paclitaxel chemotherapy outcome in patients with early stage breast cancer. The researchers have developed an immunohistochemistry method for determining pAkt status with appropriate controls for assay performance and cutoff for pAkt positivity. They also discovered methods of correlating pAkt expression with clinical outcome (disease-free survival and overall survival). pAkt is a novel predictive marker of taxane chemotherapy, and can be applied to indicate which patients should receive taxane-based chemotherapy.

Applications: A Kit for identifying pAkt-positive tumors in surgical tumor specimens or tumor biopsies prior to treatment (adjuvant, neoadjuvant therapy or therapy for metastatic disease); and methods for predicting clinical outcome from taxane chemotherapy.

Advantages: pAkt is a useful clinical predictive marker to determine which patients should or should not receive taxane-based chemotherapy for cancer. Determining pAkt status would allow patients with pAkt-positive tumors to elect taxane therapy for whom are likely to benefit, and allow patients with pAkt-negative tumors for whom are unlikely to benefit to be spared from taxane therapy as well as toxicity, and earlier use of other therapies that could be more effective. The application of this invention may potentially reduce the cost of cancer care.

Inventors: Sherry X. Yang *et al.* (NCI).

Related Publications:

1. Yang, SX, Costantino JP, Mamounas EP, Nguyen D, Jeong J–H, Wolmark N, Kim C, Kidwell K, Paik S, Swain SM. Correlation of levels of Akt phosphorylation at Ser473 with benefit from paclitaxel chemotherapy in NSABP B–28 patients with node-positive breast cancer. J Clin Oncol. 2009 (May 20 Supplement);27(15S):537.

2. Yang SX, Costantino JP, Mamounas EP, Nguyen D, Jeong J–H, Wolmark N, Kim C, Kidwell K, Paik S, Swain SM. Akt phosphorylation at Ser473 predicts benefit to paclitaxel chemotherapy in node-positive breast cancer. J Clin Oncol. 2010, In Press.

Patent Status: U.S. Provisional Application No. 61/180,558 filed 22

May 2009 (HHS Reference No. E–191–2009/0–US–01).

Licensing Status: Available for licensing.

Licensing Contact: Patrick P. McCue, PhD; 301–496–7057; *mccuepat@mail.nih.gov.*

Collaborative Research Opportunity: The National Cancer Institute is seeking statements of capability or interest from parties interested in collaborative research to further develop, evaluate, or commercialize the pAkt assay for use in a clinical setting. The National Cancer Institute would be particularly interested in discussing collaborations to provide additional clinical validation of pAkt as a primary biomarker. Please contact John Hewes, PhD at 301–435–3131 or *hewesj@mail.nih.gov* for more information.

Dated: March 10, 2010.

Richard U. Rodriguez,

Director, Division of Technology Development and Transfer, Office of Technology Transfer, National Institutes of Health.

[FR Doc. 2010–5765 Filed 3–16–10; 8:45 am]

BILLING CODE 4140–01–P

DEPARTMENT OF HEALTH AND HUMAN SERVICES

National Institutes of Health

National Institute of Diabetes and Digestive and Kidney Diseases; Notice of Closed Meetings

Pursuant to section 10(d) of the Federal Advisory Committee Act, as amended (5 U.S.C. App.), notice is hereby given of the following meetings.

The meetings will be closed to the public in accordance with the provisions set forth in sections 552b(c)(4) and 552b(c)(6), Title 5 U.S.C., as amended. The grant applications and the discussions could disclose confidential trade secrets or commercial property such as patentable material, and personal information concerning individuals associated with the grant applications, the disclosure of which would constitute a clearly unwarranted invasion of personal privacy.

Name of Committee: National Institute of Diabetes and Digestive and Kidney Diseases Special Emphasis Panel; RO1 Review.

Date: April 6, 2010.

Time: 2 p.m. to 4 p.m.

Agenda: To review and evaluate grant applications.

Place: National Institutes of Health, Two Democracy Plaza, 6707 Democracy Boulevard, Bethesda, MD 20892. (Telephone Conference Call)

Contact Person: D.G. Patel, PhD, Scientific Review Officer, Review Branch, DEA, NIDDK, National Institutes of Health, Room 756, 6707 Democracy Boulevard, Bethesda, MD 20892–5452, (301) 594–7682, *pateldg@niddk.nih.gov.*

This notice is being published less than 15 days prior to the meeting due to the timing limitations imposed by the review and funding cycle.

Name of Committee: National Institute of Diabetes and Digestive and Kidney Diseases Special Emphasis Panel; Translational Research.

Date: May 20, 2010.

Time: 8 a.m. to 5 p.m.

Agenda: To review and evaluate grant applications.

Place: Bethesda Marriott Suites, 6711 Democracy Boulevard, Bethesda, MD 20817.

Contact Person: Michele L. Barnard, PhD, Scientific Review Officer, Review Branch, DEA, NIDDK, National Institutes of Health, Room 753, 6707 Democracy Boulevard, Bethesda, MD 20892–2542, (301) 594–8898, *barnardm@extra.niddk.nih.gov.*

(Catalogue of Federal Domestic Assistance Program Nos. 93.847, Diabetes, Endocrinology and Metabolic Research; 93.848, Digestive Diseases and Nutrition Research; 93.849, Kidney Diseases, Urology and Hematology Research, National Institutes of Health, HHS)

Dated: March 10, 2010.

Jennifer Spaeth,

Director, Office of Federal Advisory Committee Policy.

[FR Doc. 2010–5768 Filed 3–16–10; 8:45 am]

BILLING CODE 4140–01–P

DEPARTMENT OF HEALTH AND HUMAN SERVICES

National Institutes of Health

Center for Scientific Review; Notice of Closed Meetings

Pursuant to section 10(d) of the Federal Advisory Committee Act, as amended (5 U.S.C. App.), notice is hereby given of the following meetings.

The meetings will be closed to the public in accordance with the provisions set forth in sections 552b(c)(4) and 552b(c)(6), Title 5 U.S.C., as amended. The grant applications and the discussions could disclose confidential trade secrets or commercial property such as patentable material, and personal information concerning individuals associated with the grant applications, the disclosure of which would constitute a clearly unwarranted invasion of personal privacy.

Name of Committee: Center for Scientific Review Special Emphasis Panel; Member Conflict: AOIC & VACC.

Date: April 7–8, 2010.

Time: 8 a.m. to 5 p.m.

Agenda: To review and evaluate grant applications.

Place: National Institutes of Health, 6701 Rockledge Drive, Bethesda, MD 20892. (Virtual Meeting)

Contact Person: Kenneth A. Roebuck, PhD, Scientific Review Officer, Center for Scientific Review, National Institutes of Health, 6701 Rockledge Drive, Room 5106, MSC 7852, Bethesda, MD 20892, (301) 435–1166, *roebuckk@csr.nih.gov.*

Name of Committee: Center for Scientific Review Special Emphasis Panel; Ethical, Legal and Societal Implications of Genetic Information.

Date: April 19, 2010.

Time: 12 p.m. to 1 p.m.

Agenda: To review and evaluate grant applications.

Place: National Institutes of Health, 6701 Rockledge Drive, Bethesda, MD 20892. (Telephone Conference Call)

Contact Person: Cheryl M. Corsaro, PhD, Scientific Review Officer, Center for Scientific Review, National Institutes of Health, 6701 Rockledge Drive, Room 2204, MSC 7890, Bethesda, MD 20892, (301) 435–1045, *corsaroc@csr.nih.gov.*

(Catalogue of Federal Domestic Assistance Program Nos. 93.306, Comparative Medicine; 93.333, Clinical Research, 93.306, 93.333, 93.337, 93.393–93.396, 93.837–93.844, 93.846–93.878, 93.892, 93.893, National Institutes of Health, HHS)

Dated: March 10, 2010.

Jennifer Spaeth,

Director, Office of Federal Advisory Committee Policy.

[FR Doc. 2010–5770 Filed 3–16–10; 8:45 am]

BILLING CODE 4140–01–P

DEPARTMENT OF HEALTH AND HUMAN SERVICES

National Institutes of Health

National Center on Minority Health and Health Disparities; Notice of Closed Meetings

Pursuant to section 10(d) of the Federal Advisory Committee Act, as amended (5 U.S.C. App.), notice is hereby given of the following meetings.

The meetings will be closed to the public in accordance with the provisions set forth in sections 552b(c)(4) and 552b(c)(6), Title 5 U.S.C., as amended. The grant applications and the discussions could disclose confidential trade secrets or commercial property such as patentable material, and personal information concerning individuals associated with the grant applications, the disclosure of which would constitute a clearly unwarranted invasion of personal privacy.

Name of Committee: National Center on Minority Health and Health Disparities Special Emphasis Panel Loan Repayment Program for Health Disparities Research—Panel 2.

Date: April 5, 2010.

Time: 8 a.m. to 5 p.m.

Agenda: To review and evaluate grant applications.