than by recombination in mammalian cells.

2. It can be used to produce recombinant vaccinia viruses for gene expression.

3. It can be used for the production of modified vaccinia viruses that have improved safety or immunogenicity.

Advantages of the VAC–BAC shuttle system:

1. VAC–BACs are clonally purified from bacterial colonies before virus reconstitution in mammalian cells.

2. Manipulation of DNA is much simpler and faster in bacteria than in mammalian cells.

3. Modified genomes can be characterized prior to virus reconstitution.

4. Only virus with modified genomes will be produced so that virus plaque isolations are not needed.

5. Generation of a stock of virus from a VAC–BAC is accomplished within a week rather than many weeks.

6. Multiple viruses can be generated at the same time since plaque purification is unnecessary.

References:

1. Domi, A., and B. Moss. 2002. Cloning the vaccinia virus genome as a bacterial artificial chromosome in Escherichia coli and recovery of infectious virus in mammalian cells. Proc. Natl. Acad. Sci. USA 99:12415–12420.

2. Domi, A., and B. Moss. 2005. Engineering of a vaccinia virus bacterial artificial chromosome in Escherichia coli by bacteriophage lambda-based recombination. Nature Methods 2:95–97.

In addition to licensing, the technology is available for further development through collaborative research opportunities with the inventors.

Dated: May 12, 2005.

Steven M. Ferguson,

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

[FR Doc. 05–10064 Filed 5–19–05; 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, DHHS.

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.

DU145 Camptothecin (CPT)-Resistant Cell Line

Dr. Yves Pommier (NCI)
DHHS Reference No. E–159–2005/0—Research Tool
Licensing Contact: John Stansberry; 301/435–5236; *stansbej@mail.nih.gov*

Drug resistance is a major limitation of chemotherapy. Understanding how drug resistance develops may lead to more effective treatments. This invention describes the DU145 Camptothecin (CPT)-resistant prostate cancer cell line that can be used to study mechanisms of drug resistance. For more details see Pommier et al., Cancer Research 61, 1964–1969, March 1, 2001.

Mammary Gland Differentiation by 2-Methoxyestradiol

Jeffrey E. Green et al. (NCI)
DHHS Ref. No. E–069–2005/0–US–01
Licensing Contact: Thomas P. Clouse; 301/435–4076; *clouset@mail.nih.gov*

This invention is based on the discovery that administration of 2-Methoxyestradiol (2-ME2) to female mice at various developmental stages will result in the differentiation of mammary epithelial cells to form rudimentary alveolar structures and to produce milk proteins. This effect has also been demonstrated in an in vitro experimental system. Since 2-ME2 is highly expressed during late stages of human pregnancy and pregnancy is known to reduce the risk of human bresat cancer, possibly due to differentiating effects on the mammary gland, 2ME2 may be developed into a preventive agent against breast cancer in women. Additionally, 2-ME2 may be useful in augmenting mammary gland differentiation and milk production under circumstances where normal differentiation is compromised.

In addition to licensing, the technology is available for further development through collaborative research opportunities with the inventors.

Methods for Detecting Progression of Low Grade Cervical Dysplasia

Thomas Ried et al. (NCI)
DHHS Reference No. E–041–2005/0–US–01
Licensing Contact: Thomas P. Clouse; 301/435–4076; *clouset@mail.nih.gov*

This invention describes a test that can be applied to Pap smears to differentiate low-grade dysplastic lesions that are likely to progress to higher-grade dysplasia and cervical cancer from those that are likely to regress. The differentiating factor is the presence of genetic gain on the long arm of chromosome 3. The inventors have shown that low grade Pap smears that progress already exhibit extra copies of 3q, while those that do not show the 3q gain spontaneously regress.

Around 10–15% of the 3 million Pap smears with low-grade dysplasia each year in the United States progress to higher grade lesions. Currently, HPV testing is used to stratify these low grade disease Pap smears, but as the majority of these Pap smears are already HPV infected, the test has very low specificity. The instant 3q test, which targets the human telomerase gene, TERC, is a significant improvement in sensitivity and specificity over the current methods used for the detection of progressing versus regressing lesions.

Antibodies to Rheb, a Ras-Related Protein

Geoffrey J. Clark and Michele Vos (NCI)
DHHS Reference No. E–351–2004—Research Tool.
Licensing Contact: Mojdeh Bahar; 301/435–2950; *baharm@mail.nih.gov*

The invention relates to polyclonal antibodies that recognize the protein Rheb, a key player in protein biosynthesis. Rheb is a small GTP-binding protein that is structurally related to the oncoprotein Ras, but Rheb does not activate the same pathways as Ras. Instead, Rheb binds to the tumor suppressor TSC2 (Tuberin) and causes activation of the S6 kinase in a TOR (Target of Rapamycin) dependent manner. Rheb likely plays roles in the response to insulin and the development of human tumors. Thus, the antibodies could provide useful reagents to investigate the functions of Rheb in these and other biological processes.

In addition to licensing, the technology is available for further development through collaborative research opportunities with the inventors.

Methods of Reducing the Activity and Concentration of an Eph Receptor Tyrosine Kinase

Jennifer Isaacs and Leonard Neckers (NCI)

U.S. Provisional Application No. 60/591,986 filed 29 Jul 2004 (DHHS Reference No. E–245–2004/0–US–01)

Licensing Contact: George Pipia; 301/435–5560; *pipiag@mail.nih.gov*

The Eph receptors comprise a family of 14 members and as such, they carry out diverse functions, including embryonic patterning, migration, and the formation of neural networks. Recently, it was discovered that a subset of these proteins play an integral role in the formation of blood vessels, or angiogenesis, which is a process essential to tumor development. In fact, several of these proteins have the capacity to transform normal cells, when overexpressed. We have discovered that the HSP90 inhibitor 17-Allylamino-17-demethoxygeldanamycin (17–AAG) effectively downregulates the level of several angiogenic Eph receptors and impairs their oncogenic signaling. This suggests that it maybe possible treat cancers overexpressing these oncogenes, by selectively inhibiting HSP90 with 17–AAG and its derivatives.

Retinal Pigment Epithelial Cells Immortalized with TERT and Expressing the Adenoviral E1A Oncoprotein

Karen Vousden *et al.* (NCI)

DHHS Reference No. E–135–2004/0—Research Tool

Licensing Contact: Thomas P. Clouse; 301/435–4076; *clouset@mail.nih.gov*

This invention describes human retinal pigment epithelial cells immortalized with telomerase reverse transcriptase (TERT). Some of these cells express the adenoviral E1A oncoprotein, while others do not. The E1A expressing cells serve as a model for cancerous cells. Those that do not express E1A behave like normal cells. As such these immortalized cells can be used to compare the behavior of normal and cancer cells in vitro.

Analogs of Thalidomide as Potential Angiogenesis Inhibitors

William D. Figg, Erin Lepper (NCI)

U.S. Provisional Application No. 60/486,515 filed 11 Jul 2003 (DHHS Reference No. E–272–2003/0–US–01); PCT Application No. PCT/US04/22242 filed 09 Jul 2004 (DHHS Reference No. E–272–2003/0–PCT–02)

Licensing Contact: Jesse Kindra; 301/435–5559; *kindraj@mail.nih.gov*

The present disclosure relates to anti-angiogenesis compositions and methods, and particularly thalidomide analogs that actively inhibit angiogenesis in humans and animals.

Angiogenesis is the formation of new blood vessels from pre-existing vessels. Angiogenesis is prominent in solid tumor formation and metastasis. A tumor requires formation of a network of blood vessels to sustain the nutrient and oxygen supply for continued growth. Some tumors in which angiogenesis is important include most solid tumors and benign tumors, such as acoustic neuroma, neurofibroma, trachoma, and pyogenic granulomas. Prevention of angiogenesis could halt the growth of these tumors and the resultant damage due to the presence of the tumor.

The subject application discloses active thalidomide analogs that exhibit enhanced potency in the inhibition of undesirable angiogenesis, and methods for using these compounds to treat angiogenesis and solid tumors. In particular, the presently disclosed method provides for inhibiting unwanted angiogenesis in a human or animal by administering to the human or animal with the undesired angiogenesis a composition comprising an effective amount of the active thalidomide analogs. According to a more specific aspect, the method involves inhibiting angiogenesis by exposing a mass having the undesirable angiogenesis to an angiogenesis inhibiting amount of one or more compounds, or pharmaceutically acceptable salts of such compounds.

Mycolactone and Related Compounds

Pamela L. Small and Kathleen M. George (NIAID)

U.S. Patent 6,680,055 issued 20 Jan 2004 (DHHS Reference No. E–199–1999/0–US–06)

Licensing Contact: John Stansberry; 301/435–5236; *stansbej@mail.nih.gov*

This application describes and claims novel pharmocoactive compounds which belong to the class of compounds known as polyketide macrolides. These compounds have been isolated from *M. ulcerans* the causative agent of buruli ulcers. Early work with these compounds suggests that the principle compound, mycolactone, or mixtures of mycolactone with other isolated polyketide macrolides or other agents may be useful in treating cancer or suppressing an inflammatory response.

In addition to the novel polyketide macrolide compounds the application also describes compositions derived from a non-virulent strain of *M. ulcerans.* These compositions may be useful in inducing an immune response (vaccines) which could be useful in providing subjects with resistance to the development of buruli ulcers. Antibodies against mycolactone are being developed. These antibodies could be used for diagnostic purposes.

Some early publications which describe this work are KM George et al. Science 283(5403): 854–7 (Feb. 5, 1999) and KM George et al. Infect. Immun. 66(2): 587–93 (Feb. 1998). More recently, novel mycolactones have been isolated and characterized from Australian isolates of *M. ulcerans* (Judd et al. Organic Lett. 6: 4901–4904 (2004)) as well as from the frog pathogen *M. liflandii* (Mve-Obiang, A. et al. Infect. Immun. (In Press)).

Spatial and Temporal Control of Gene Expression Using a Heat Shock Protein Promoter in Combination with Local Heat

Chrit T. Moonen (ORS)

U.S. Patent Application No. 10/864,102 filed 09 Jun 2004, claiming priority to 15 Aug 1996 (DHHS Reference No. E–235–1995/0–US–09); Foreign rights available

Licensing Contact: George Pipia; 301/435–5560; *pipiag@mail.nih.gov*

In many instances, it is desirable to express exogenous genes only in certain tissues, and/or at will at certain times, and/or only to a certain degree. However, current gene transfer and exogenous gene expression protocols do not provide adequate means of simultaneously controlling which cells in a heterogeneous population are transformed and when, where, and to what degree the transferred genes are expressed. The invention provides methods for using local heat to control gene expression. The heat shock protein (hsp) gene promoter is recombined with a selected therapeutic gene and expressed in selected cells. Local controlled heating is used to activate the hsp promoter, for example by using focused ultrasound controlled by MRI.

In addition to licensing, the technology is available for further development through collaborative research opportunities with the inventors.

Dated: May 11, 2005.

Steven M. Ferguson,

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

[FR Doc. 05–10065 Filed 5–19–05; 8:45 am]

BILLING CODE 4140–01–P

DEPARTMENT OF HEALTH AND HUMAN SERVICES

National Institutes of Health

National Cancer Institute; Notice of Meeting

Pursuant to section 10(a) of the Federal Advisory Committee Act, as amended (5 U.S.C. Appendix 2), notice is hereby given of a meeting of the National Cancer Institute Board of Scientific Advisors.

The meeting will be open to the public, with attendance limited to space available. Individuals who plan to attend and need special assistance, such as sign language interpretation or other reasonable accommodations, should notify the Contact Person listed below in advance of the meeting.

Name of Committee: National Cancer Institute Board of Scientific Advisors.

Date: June 27–28, 2005.

Time: June 27, 2005, 8 a.m. to 6 p.m.

Agenda: Director's Report; Ongoing and New Business; Reports of Program Review Group(s); and Budget Presentation; Reports of Special Initiatives; RFA and RFP Concept Review; and Scientific Presentations.

Place: National Institutes of Health, Building 31, C Wing, 6 Floor, Conference Rm. 10, 9000 Rockville Pike, Bethesda, MD 20892.

Time: June 28, 2005, 8:30 a.m. to 1 p.m.

Agenda: Ongoing and New Business; Reports of Program Review Group(s); and Budget Presentation; Reports of Special Initiatives; RFA and RFP Concept Review; and Scientific Presentations.

Place: National Institutes of Health, Building 31, C Wing, 6 Floor, Conference Rm. 10, 9000 Rockville Pike, Bethesda, MD 20892.

Contact Person: Paulette S. Gray, PhD, Executive Secretary, Division of Extramural Activities, National Cancer Institute, National Institutes of Health, 6116 Executive Boulevard, 8th Floor, Rm. 8001, Bethesda, MD 20892, (301) 496–5147.

Any interested person may file written comments with the committee by forwarding the statement to the Contact Person listed on this notice. The statement should include the name, address, telephone number and when applicable, the business or professional affiliation of the interested person.

In the interest of security, NIH has instituted stringent procedures for entrance into the building by non-government employees. Persons without a government I.D. will need to show a photo I.D. and sign-in at the security desk upon entering the building.

Information is also available on the Institute's/Center's home page: *http://deainfo.nci.nih.gov/advisory/bsa.htm,* where an agenda any any additional information for the meeting will be posted when available.

(Catalogue of Federal Domestic Assistance Program Nos. 93.392, Cancer Construction; 93.393, Cancer Cause and Prevention Research; 93.394, Cancer Detection and Diagnosis Research; 93.395, Cancer Treatment Research; 93.396, Cancer Biology Research; 93.397, Cancer Centers Support; 93.398, Cancer Research Manpower; 93.399, Cancer Control, National Institutes of Health, HHS.)

Dated: May 12, 2005.

LaVerne Y. Stringfield,

Director, Office of Federal Advisory Committee Policy.

[FR Doc. 05–10070 Filed 5–19–05; 8:45 am]

BILLING CODE 4140–01–M

DEPARTMENT OF HEALTH AND HUMAN SERVICES

National Institutes of Health

National Cancer Institute; Notice of Closed Meeting

Pursuant to section 10(d) of the Federal Advisory Committee Act, as amended (5 U.S.C. Appendix 2) notice is hereby given of the following meeting.

The meeting will be closed to the public in accordance with theprovisions 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 Cancer Institute Initial Review Group, Subcommittee F—Manpower & Training.

Date: June 14–15, 2005.

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

Agenda: To review and evaluate grant applications.

Place: Wyndham City Center Hotel, 1143 New Hampshire Ave., NW., Washington, DC 20037.

Contact Person: Lynn M. Amende, PhD, Scientific Review Administrator, Resources and Training Review Branch, Division of Extramural Activities, National Cancer Institute, 6116 Executive Blvd., Room 8105, Bethesda, MD 20892, 301–451–4759, *amendel@mail.nih.gov.*

(Catalogue of Federal Domestic Assistance Program Nos. 93.392, Cancer Construction; 93.393, Cancer Cause and Prevention Research; 93.394, Cancer Detection and Diagnosis Research; 93.395, Cancer Treatment Research; 93.396, Cancer Biology Research; 93.397, Cancer Centers Support; 93.398, Cancer Research Manpower; 93.399, Cancer Control, National Institutes of Health, HHS.)

Dated: May 12, 2005.

LaVerne Y. Stringfield,

Director, Office of Federal Advisory Committee Policy.

[FR Doc. 05–10071 Filed 5–19–05; 8:45 am]

BILLING CODE 4140–01–M

DEPARTMENT OF HEALTH AND HUMAN SERVICES

National Institutes of Health

National Cancer Institute; Notice of Meeting

Pursuant to section 10(d) of the Federal Advisory Committee Act, as amended (5 U.S.C. Appendix 2), notice is hereby given of the meeting of the National Cancer Advisory Board.

The meeting will be open to the public as indicated below, with attendance limited to space available. Individuals who plan to attend and need special assistance, such as sign language interpretation or other reasonable accommodations, should notify the Contact Person listed below in advance of the meeting.

A portion of the meeting will be closed to the public in accordance with the provisions set forth in sections 552b(c)(4), and 552b(c)(6), 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 Cancer Advisory Board Subcommittee on Planning and Budget.

Open: June 6, 2005, 6:30 p.m. to 9 p.m.

Agenda: To discuss activities related to the Subcommittee on Planning and Budget.

Place: Hyatt Regency Bethesda, One Bethesda Metro Center, Bethesda, MD 20814.

Contact Person: Ms. Cherie Nichols, Executive Secretary, National Cancer Institute, National Institutes of Health, 6116 Executive Boulevard, 2nd Floor, Room 205, Bethesda, MD 20892–2590, (301) 496–5515.

Name of Committee: National Cancer Advisory Board.

Open: June 7, 2005, 8:30 a.m. to 4:30 p.m.

Agenda: Program reports and presentations; Business of the Board.

Place: National Cancer Institute, 9000 Rockville Pike, Building 31, C Wing, 6th Floor, Conference Room 10, Bethesda, MD 20892.

Contact Person: Dr. Paulette S. Gray, Executive Secretary, National Cancer