time, should be directed to the: Office of Management and Budget, Office of Regulatory Affairs, New Executive Office Building, Room 10235, Washington, DC 20503, Attention: Desk Officer for NIH. To request more information on the proposed project or to obtain a copy of the data collection plans and instruments, contact: Michele M. Doody, Radiation Epidemiology Branch, National Cancer Institute, Executive Plaza South, Room 7040, Bethesda, MD 20892–7238, or call non-toll-free at 301–594–7203 or e-mail your request, including your address to: *doodym@mail.nih.gov*.

Comments Due Date: Comments regarding this information collection are best assured of having their full effect if received within 30 days of the date of this publication.

Dated: February 16, 2007.

Rachelle Ragland-Greene,

NCI Project Clearance Liaison, National Institutes of Health.

[FR Doc. E7–3435 Filed 2–27–07; 8:45 am]

BILLING CODE 4104–01–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.

Methods of Determining the Prognosis of Hepatocellular Carcinoma

Description of Technology: Hepatocellular carcinoma (HCC) represents an extremely poor prognostic cancer that remains one of the most common and aggressive malignancies worldwide. A major hallmark of HCC is intrahepatic metastasis and post-surgical reoccurrence. With current diagnostic methods, HCC patients are often diagnosed with end-stage cancer and have poor survival. Thus, there is a need for an accurate method to identify HCC and its proclivity for metastases/relapse, particularly at early stages of this disease.

The inventors have discovered a unique set of microRNA (miRNA) biomarkers that are associated with HCC metastasis/recurrence. This miRNA signature was validated in an independent cohort of 110 HCC samples as an independent predictor of HCC prognosis and likelihood of metastasis and relapse. In particular, the inventors provide evidence that these miRNA markers can predict HCC metastasis in the early stages of cancer. This methodology may enable clinicians to effectively stratify patients for appropriate cancer treatment and prioritize liver transplantation candidates.

Applications: (1) Method to prognose HCC, patient survival and likelihood of HCC metastasis/relapse; (2) Diagnostic tool to aid clinicians in determining appropriate cancer treatment; (3) Compositions that inhibit miRNA HCC biomarkers such as siRNA; (4) Method to treatment HCC patients with inhibitory miRNA compositions.

Market: (1) Primary liver cancer accounts for about 2% of cancers in the U.S., but up to half of all cancers in some undeveloped countries; (2) Post-operative five year survival rate of HCC patients is 30–40%.

Development Status: This technology is currently in the pre-clinical stage of development.

Inventors: Xin Wei Wang et al. (NCI).

Publication: Budhu *et al.* A Unique Metastasis-related MicroRNA Expression Signature Predicts Survival and Recurrence in Hepatocellular Carcinoma, manuscript in preparation.

Patent Status: U.S. Provisional Application No. 60/884,052 filed 09 Jan 2007 (HHS Reference No. E–050–2007/0–US–01).

Licensing Availability: Available for exclusive or non-exclusive licensing.

Licensing Contact: Jennifer Wong; 301/435–4633; *wongje@mail.nih.gov.*

A Varicella-Zoster Virus Mutant that is Markedly Impaired for Latent Infection Available for the Development of Shingles Vaccines and Diagnostics

Description of Technology: Reactivation of latent Varicella-Zoster virus (VZV) infection is the cause of shingles, which is prominent in adults over the age of 60 and individuals who have compromised immune systems, due to HIV infection, cancer treatment and/or transplant. Shingles is a worldwide health concern that affects approximately 600,000 Americans each year. The incidence of shingles is also high in Europe, South America, and India; the latter having an estimated two million individuals affected, yearly. Recent research studies show that VZV vaccines have a significant effect on decreasing the incidence of shingles in elderly.

The current technology describes compositions, cells and methods related to the production and use of a mutant VZV and the development of vaccines against the infectious agent. Latent VZV expresses a limited repertoire of viral genes including the following six open reading frames (ORFs): 4, 21, 29, 62, 63, and 66. The present invention describes an ORF29 mutant VZV that demonstrates a weakened ability to establish latency in animal studies. The current technology provides methods for using the mutant in the development of live vaccines and diagnostic tools. A related invention is described in PCT/US05/021788 (publication number WO2006012092).

Applications: Development of vaccines and diagnostics for prevention of shingles.

Development Status: Pre-clinical studies have been performed to demonstrate the reduced latency of the ORF29 mutant VZV in animals.

Inventors: Jeffrey Cohen (NIAID) and Lesley Pesnicak (NIAID).

Patent Status: U.S. Provisional Application No. 60/857,766 filed 09 Nov 2006 (HHS Reference No. E–029–2007/0–US–01).

Licensing Availability: Available for licensing and commercial development.

Licensing Contact: Chekesha Clingman, Ph.D.; 301/435–5018; *clingmac@mail.nih.gov.*

Collaborative Research Opportunity: The NIAID Laboratory of Clinical Infectious Diseases is seeking statements of capability or interest from parties interested in collaborative research to further develop, evaluate, or commercialize vaccine strains of VZV vaccine with impaired latency. Please contact Kelly Murphy, J.D., M.S., at 301/451–3523 or *murphykt@niaid.nih.gov* for more information.

Highly Soluble Pyrimido-Dione-Quinoline Compounds: Small Molecules That Stabilize and Activate p53 in Transformed Cells

Description of Technology: The tumor-suppressor p53 protein plays a major role in tumor development. Most human cancers ail to normally activate p53, which is at least partly responsible for the unregulated growth of cancer cells and their failure to undergo apoptosis. While many chemotherapeutics enhance p53 levels, their non-specific DNA damage (genotoxicity) causes unfavorable side effects.

This invention reports the composition and function of a pyrimido-dione-quinoline that was found to inhibit HDM2's ubiquitin ligase (E3) activity without the accompanying genotoxicity of current therapeutic drugs. Like the HLI98 family of compounds reported previously (see reference below), the subject of the current invention stabilizes p53 in cells, inhibiting its ubiquitin-mediated proteasomal degradation. Unlike the HLI98 compound, the pyrimido-dione-quinoline reported here induces a robust p53 response, and is highly water-soluble. Thus, these pyrimido-dione-quinoline compounds have the potential to stabilize p53 and activate a p53 response in tumors.

Applications and Modality: Water-soluble with improved potency in stabilizing p53 and activating a p53 response; Inhibits unregulated growth of cancer cells; Reduced genotoxicity compared to many chemotherapeutics.

Market: Small molecule-based cancer therapeutics for tumors expressing wild type p53, which comprises approximately 50% of cancers.

Development Status: The technology is currently in the pre-clinical stage of development.

Inventors: Allan M. Weissman and Yili Yang (NCI).

Related Publication: Y Yang et al. Small molecule inhibitors of HDM2 ubiquitin ligase activity stabilize and activate p53 in cells. Cancer Cell 2005 Jun;7(6):547–559.

Patent Status: U.S. Provisional Application No. 60/813,946 filed 14 Jun 2006 (HHS Reference No. E–138–2006/0–US–01).

Availability: Available for exclusive and non-exclusive licensing.

Licensing Contact: Thomas P. Clouse, J.D.; 301/435–4076; clousetp@*mail.nih.gov.*

Collaborative Research Opportunity: The Laboratory of Protein Dynamics and Signaling (LPDS) at the National Cancer Institute, NIH, is seeking a collaborative partner under a Cooperative Research and Development Agreement (CRADA) to develop therapeutics approaches utilizing inhibitors of the ubiquitin system such as described in this invention. Please contact John D. Hewes, Ph.D. at 301–435–3121 or *hewesj@mail.nih.gov* for more information.

Human Cancer Therapy Using Engineered Anthrax Lethal Toxin

Description of Technology: Anthrax lethal toxin (LeTx) consists of two components: The protective antigen (PrAg) and the lethal factor (LF). PrAg binds to the cell surface where it is activated by furin protease, followed by the formation of a PrAg heptamer. LF is then translocated into the cytosol of a cell via this heptamer, where it acts as a metalloprotease on all but one mitogen-activated protein kinase kinase (MAPKK). Approximately 70% of human melanomas contain a mutation (B–RAF V600E) that constitutively activates a MAPKK pathway, and LeTx has been shown to have significant toxicity towards cells which have this mutation. This suggested a potential use for LeTx in cancer therapy. Unfortunately, native LeTx is toxic to normal cells, detracting from its *in vivo* applicability.

PrAg has been engineered to be activated by a matrix metalloprotease (MMP), instead of by furin protease. Because MMPs are highly expressed in tumor cells, this modification increases selectivity towards cancer cells. Surprisingly, mouse data shows that the modified LeTx (denoted PrAg–L1/LF) is less cytotoxic to ''normal'' cells in vivo, when compared to wild-type LeTx. Significantly, PrAg–L1/LF maintained its high toxicity toward human tumors in mouse xenograft models of human tumors, including melanomas. However, this toxicity applied not only to tumors having mutations that constitutively activate MAPKKs, but also to other tumor types such as lung and colon carcinomas. The absence of toxicity to ''normal'' cells coupled to its effectiveness on a wide range of cancer cell types suggests that PrAg–L1/LF may represent a novel cancer therapeutic.

Applications: PrAg–L1/LF has applications as a human cancer therapeutic; Applicability extends beyond melanomas, including lung and colon carcinomas.

Market: The worldwide market for melanoma therapeutics is approximately $437M, and is predicted to reach $680M by the year 2009. Approximately 2.4 million people are afflicted with melanoma, with around 150,000 new cases each year. Demonstration of effectiveness *in vivo* for lung and colon carcinomas will increase the market for this technology.

Development Status: The technology is at the preclinical stage.

Inventors: Stephen H. Leppla (NIAID), Shi-hui Liu (NIAID), Thomas H. Bugge (NIDCR), John R. Basile (NIDCR), Brooke Currie (NIDCR).

Related Publications:

1. S Liu *et al.* Intermolecular complementation achieves high-specificity tumor targeting by anthrax toxin. Nat Biotechnol. 2005 Jun;23(6):725–730.

2. RJ Abi-Habib *et al.* A urokinase-activated recombinant anthrax toxin is selectively cytotoxic to many human tumor cell types. Mol Cancer Ther. 2006 Oct;5(10):2556–2562.

Patent Status: U.S. Provisional Application No. 60/870,050 filed 14 Dec 2006 (HHS Reference E–070–2007/0–US–01).

Licensing Status: Available for exclusive or non-exclusive licensing.

Licensing Contact: David A. Lambertson, Ph.D.; 301/435–4632; *lambertsond@od.nih.gov.*

Collaborative Research Opportunity: The NIAID Laboratory of Bacterial Diseases is seeking statements of capability or interest from parties interested in collaborative research to further develop, evaluate, or commercialize PrAg–L1/LF as a novel cancer therapeutic. Please contact Stephen H. Leppla, Ph.D. at 301/594–2865 and/or *sleppla@niaid.nih.gov* for more information.

This abstract was originally published in the **Federal Register** on Wednesday, February 7, 2007, 72 FR 5726, with an incorrect title of ''Extended Transgene Expression for a Non-Integrating Adenoviral Vector Containing Retroviral Elements.''

Dated: February 20, 2007.

Steven M. Ferguson,

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

[FR Doc. E7–3436 Filed 2–27–07; 8:45 am]

BILLING CODE 4140–01–P

DEPARTMENT OF HEALTH AND HUMAN SERVICES

Substance Abuse and Mental Health Services Administration

Agency Information Collection Activities: Proposed Collection; Comment Request

In compliance with Section 3506(c)(2)(A) of the Paperwork Reduction Act of 1995 concerning