Dated: September 8, 2010.

Bernadette Dunham,

Director, Center for Veterinary Medicine.

[FR Doc. 2010–22811 Filed 9–13–10; 8:45 am]

BILLING CODE 4160–01–S

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.

Assay for Arf GTP-Binding Proteins

Description of Invention: The worldwide laboratory research reagents market is expected to surpass $13 billion in 2010, and the field of biotechnology appears key to maintaining the market's growth. Antibodies are becoming increasingly significant, especially for targeting the diseased cells and cell compounds.

Researchers at the National Cancer Institute (NCI), NIH, have developed an antibody-based assay that measures levels of Arf GTP-binding proteins, some of which have been linked to the invasive behavior of cancer cells. The assay is robust, can be performed both on cell lysates and fixed cells, and can distinguish among specific endogenous Arf-GTP isoforms.

Applications:

• Research on Arf function in physiology and cancer.

• Research on cancer invasion.

• Research on membrane traffic and actin reorganization.

Advantages:

• Ability to distinguish between the specific isoforms (i.e., Arf1, Arf3, Arf4, Arf5, and Arf6).

• Antibodies bind preferentially to the GTP-bound form of Arf.

Inventor: Paul A. Randazzo (NCI).

Relevant Publications:

1. Spang A *et al.* Arf GAPs: gatekeepers of vesicle generation. FEBS Lett. 2010 Jun 18;584(12):2646–2651. [PubMed: 20394747].

2. Campa F and Randazzo PA. Arf GTPase-activating proteins and their potential role in cell migration and invasion. Cell Adh Migr. 2008 Oct; 2(4):258–262. [PubMed: 19262159].

Patent Status: HHS Reference No. E–198–2010/0—Research Material. Patent protection is not being pursued for this technology.

Licensing Status: Available for licensing.

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

Collaborative Research Opportunity: The Center for Cancer Research, Laboratory of Cellular and Molecular Biology, is seeking statements of capability or interest from parties interested in collaborative research to further develop, evaluate, or commercialize this technology. Please contact John D. Hewes, PhD at 301–435–3121 or *hewesj@mail.nih.gov* for more information.

Sequences Encoding Two Novel Human Polyomaviruses

Description of Invention: Researchers at the National Cancer Institute, NIH, have discovered two species of a previously unknown polyomavirus genus.

Polyomaviruses are a diverse group of DNA-based viruses that infect humans and various animals. At least one human polyomavirus, the Merkel cell polyomavirus (MCV), plays a causal role in the development of an unusual form of skin cancer called Merkel cell carcinoma. The coat proteins of polyomaviruses can spontaneously assemble into virus-like particles (VLPs) similar to those that have been used in the recent vaccines against human papillomaviruses (HPVs).

Applications:

• Development of clinical diagnostic assays to detect linkages between the new polyomaviruses and human cancers.

• Development of a VLP-based prophylactic vaccine similar to the HPV vaccine.

Advantages: DNA sequences have broad applications in the studies of polyomavirus infection mechanisms and carcinogenesis. Notably, they are:

• Identification and purification of the normal and mutated polyomaviral proteins.

• Studies of antisense oligonucleotides in polyomavirus biology.

• Development of polyclonal and monoclonal antibodies against polyomaviruses.

Development Status: Pre-clinical.

Inventors: Christopher B. Buck and Diana V. Pastrana (NCI).

Relevant Publication: Schowalter RM *et al.* Merkel cell polyomavirus and two previously unknown polyomaviruses are chronically shed from human skin. Cell Host Microbe Jun 25;7(6):509–515. [PubMed: 20542254].

Patent Status: U.S. Provisional Application No. 61/318,080 filed 26 Mar 2010 (HHS Reference No. E–051–2010/0–US–01).

Licensing Status: Available for licensing.

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

Fenoterol and Fenoterol Analogues for Treatment of Glioma, Glioblastoma, and Astrocytoma

Description of Invention: To date there is no effective treatment for the brain tumors or brain cancers indentified as gliomas, glioblastomas, or astrocytomas.

This technology relates to the discovery that fenoterol and related analogues block astrocytoma and glioblastoma cell division at low doses. In a xenograft model utilizing the 1321N1 astrocytoma tumor implanted in the flank of SKID mice, the (R,R)-4-methoxyfenoterol analogue significantly decreased tumor growth relative to a control group receiving vehicle and studies utilizing [^{3}H]-(R,R)-4-methoxyfenoterol have shown that the compound readily passes the blood-brain barrier. The anti-tumor effect is associated with the ability of fenoterol and related analogues to induce production of cyclic adenosine monophosphate (cAMP), which is normally decreased in glioblastomas and astrocytomas. Induced cAMP production inhibits brain tumor growth in vivo. Fenoterol and related analogues are beta-2 adrenergic receptor (β2 AR) agonists and the anti-tumor effect is associated with the expression of this receptor. Since there is a heterogeneous expression of β2 AR in human brain tumors, patients who will respond to fenoterol therapy can be predetermined leading to individualized treatment. In addition to use in the initial treatment of brain tumors, the systemic and CNS bioavailability of the drug after oral

administration and the minimal systemic toxicity suggest that fenoterol and it analogs can be used in the adjuvant treatment of patients with β2 AR-positive gliomas, glioblastomas or astrocytomas. Studies with a number of fenoterol analogs and CNS-implanted tumors are in progress.

The fenoterol analogues discussed in this technology are subject to HHS Ref. No. E–205–2006/3 (U.S. Patent Application No. 12/376,945 and PCT Publication No. WO/2008/022038).

Applications: Therapeutic in the front line and adjuvant treatment of glioma, glioblastoma and astrocytoma.

Advantages: Potential first-in-class therapeutic for multiple types of brain tumors.

Development Status: In vivo: tumor models in SKID mice. *In vitro:* cell-based assays using human glioblastoma and astrocytoma cell lines. Further *in vivo* studies in animal models are underway.

Market: Approximately 17,000 Americans are diagnosed with gliomas annually (*http://www.mayoclinic.org/glioma/*).

Inventors: Irving W. Wainer (NIA), *et al.*

Publication: Submitted.

Patent Status: U.S. Provisional Application No. 61/312,642 filed 10 Mar 2010 (HHS Reference No. E–013–2010/0).

Licensing Status: Available for licensing.

Licensing Contact: Fatima Sayyid, M.H.P.M.; 301–435–4521; *Fatima.Sayyid@nih.hhs.gov*.

Collaborative Research Opportunity: The National Institute on Aging, Laboratory of Clinical Investigation, is seeking statements of capability or interest from parties interested in collaborative research to further develop, evaluate, or commercialize the use of fenoterol and fenoterol analogs in the front line and adjuvant treatment of CNS tumors and other β2 AR expressing tumors. Please contact Nicole Darack, PhD at 301–435–3101 or *darackn@mail.nih.gov* for more information.

Chemical Attraction: Cell Lines Expressing the CXCR1 or CXCR2 Chemokine Receptors

Description of Invention: Chemoattractant receptors have been identified as important factors in regulating many innate and adaptive immune responses. Modulation of these receptors have implications for shifting immune responses to create either a dampening effect in fighting inflammatory diseases, such as autoimmune diseases or cardiovascular diseases, or a boosting effect to generate more effective responses to infectious agents, tumors, and promote wound healing. Chemokine receptors are expressed on a variety of tumor cells and play a role in helping cancer cells sense new microenvironments for metastatic growth.

Scientists at the National Institutes of Health (NIH) have developed human embryonic kidney (HEK) 293 cell lines that express the CXCR1 chemokine receptor or the CXCR2 chemokine receptor. These two receptors are also known as the IL-8 receptor-alpha and IL-8 receptor-beta, respectively. They both effectively bind IL-8, a potent neutrophil chemoattractant, as well as other chemokines with varying affinities. The collection of cell lines produced by these scientists includes HEK 293 cells that express the wild-type CXCR1, wild-type CXCR2, or mutant variants of each receptor. The cell lines were created by stably transfecting vectors containing the cDNA for each receptor into HEK 293 cells. HEK 293 cells transfected with the wild-type CXCR1 or CXCR2 display strong chemoattractant properties when placed in the presence of their corresponding CXC family chemokines, such as IL-8.

Application:

- Research tools for testing the activity of potential drugs and chemokine analogs in their ability to block cellular responses triggered by CXC chemokines, such as inflammatory responses induced by IL-8
- Cell lines expressing the wild-type CXCR1 or CXCR2 can serve as positive controls in chemokine receptor studies designed to identify novel chemoattractants or agents that inhibit chemokinetic functions.
- Research tool for screening compounds that block these receptors as a possible anti-cancer agent to inhibit angiogenesis and metastasis

Advantages:

- *Both wild-type and mutant cell lines available:* Wild-type CXCR1/CXCR2 receptors or mutant receptors with point and deletion mutations have been cloned into HEK 293 cells. These cell lines will have varying degrees of potency for their chemoattractant responses to provide a range of functional comparisons in chemokine studies.
- *Experimental verification of response to CXC family chemokines:* The scientists have compiled years of data over various publications indicating that these receptors respond appropriately to a profile of chemokines.

Inventors: Joost Oppenheim, Adit Ben-Baruch, Ji Ming Wang, David Kelvin (all NCI).

Publications:

1. E Cohen-Hillel, *et al.* Cell migration to the chemokine CXCL8: paxillin is activated and regulates adhesion and cell motility. Cell Mol Life Sci. 2009 Mar;66(5):884–899. [PubMed: 19151925].

2. H Attal, *et al.* Intracellular cross-talk between the GPCR CXCR1 and CXCR2: role of carboxyl terminus phosphorylation sites. Exp Cell Res. 2008 Jan 15;314(2):352–365. [PubMed: 17996233].

3. A Ben-Baruch, *et al.* The differential ability of IL–8 and neutrophil-activating peptide-2 to induce attenuation of chemotaxis is mediated by their divergent capabilities to phosphorylate CXCR2 (IL–8 receptor B). J Immunol. 1997 Jun 15;158(12):5927–5933. [PubMed: 9190946].

4. A Ben-Baruch, *et al.* IL–8 and NAP–2 differ in their capacities to bind and chemoattract 293 cells transfected with either IL–8 receptor type A or type B. Cytokine 1997 Jan;9(1):37–45. [PubMed: 9067094].

5. A Ben-Baruch, *et al.* Interleukin-8 receptor beta. The role of the carboxyl terminus in signal transduction. J Biol Chem. 1995 Apr 21;270(16):9121–9128. [PubMed: 7721826].

Patent Status: HHS Reference No. E–221–2009/0—Research Tool. Patent protection is not being pursued for this technology.

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

Licensing Contact: Samuel E. Bish, Ph.D.; 301–435–5282; *bishse@mail.nih.gov*.

DLC–1 Gene Deleted in Cancers

Description of Invention: Chromosomal regions that are frequently deleted in cancer cells are thought to be the loci of tumor suppressor genes, which restrict cell proliferation. Recurrent deletions on the short arm of human chromosome 8 in liver, breast, lung and prostate cancers have raised the possibility of the presence of tumor suppressor genes in this location.

The inventors have discovered the deletion of human DLC–1 gene in hepatocellular cancer (HCC) cells. They have performed in vitro experiments demonstrating the deletion in over 40% of human primary HCC and in 90% of HCC cell lines. The DLC–1 gene is located on human chromosome 8p21.3–22, a region frequently deleted in many types of human cancer. DLC–1 mRNA is

expressed in all normal tissues tested, but it has either no or low expression in a high percentage of several types of human cancer, such as liver, breast, lung, and prostate cancers. Through in vitro and in vivo tumor suppression experiments, the inventors further demonstrated that DLC–1 acts as a new tumor suppressor gene for different types of human cancer.

Applications:

- Method to diagnose HCC.
- Method to treat HCC patients with DLC–1 compositions.
- Transgenic model to study HCC and other types of human cancer.
- DLC–1 compositions.

Market:

- Primary liver cancer accounts for about 2% of cancers in the U.S., but up to half of all cancers in some undeveloped countries.
- 251,000 new cases are reported annually.
- Post-operative five year survival rate of HCC patients is 30–40%.

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

Inventors: Bao-Zhu Yuan, Snorri S. Thorgeirsson, Nicholas Popescu (NCI).

Publications:

1. BZ Yuan *et al.* DLC–1 operates as a tumor suppressor gene in human non-small cell lung carcinomas. Oncogene. 2004 Feb 19;23(7):1405–1411. [PubMed: 14661059].

2. BZ Yuan *et al.* DLC–1 gene inhibits human breast cancer cell growth and in vitro tumorigenicity. Oncogene. 2003 Jan 23;22(3):445–450. [PubMed: 12545165].

3. BZ Yuan *et al.* Promoter hypermethylation of DLC–1, a candidate tumor suppressor gene, in several common human cancers. Cancer Genet Cytogenet. 2003 Jan 15;140(2):113–117. [PubMed: 12645648].

4. BZ Yuan *et al.* Cloning, characterization, and chromosomal localization of a gene frequently deleted in human liver cancer (DLC–1) homologous to rat RhoGAP. Cancer Res. 1998 May 15;58(10):2196–2199. [PubMed: 9605766].

Patent Status:

- U.S. Patent No. 6,897,018 issued 24 May 2005 (HHS Reference No. E–042–1998/0–US–03).
- U.S. Patent No. 7,534,565 issued 19 May 2009 (HHS Reference No. E–042–1998/0–US–05).

Licensing Status: Available for licensing.

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

Collaborative Research Opportunity: The National Cancer Institute, Laboratory of Experimental Carcinogenesis, is seeking statements of capability or interest from parties interested in collaborative research to further develop, evaluate, or commercialize diagnostics based on tumor suppressor genes. Please contact John D. Hewes, PhD, at 301–435–3121 or *hewesj@mail.nih.gov* for more information.

Dated: September 7, 2010.

Richard U. Rodriguez,

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

[FR Doc. 2010–22834 Filed 9–13–10; 8:45 am]

BILLING CODE 4140–01–P

DEPARTMENT OF HEALTH AND HUMAN SERVICES

Centers for Medicare & Medicaid Services

[CMS–1338–CN]

RIN 0938–AP87

Medicare Program; Prospective Payment System and Consolidated Billing for Skilled Nursing Facilities for FY 2011; Correction

AGENCY: Centers for Medicare & Medicaid Services (CMS), HHS.

ACTION: Correction notice.

SUMMARY: This document corrects technical errors that appeared in the notice with comment period published in the **Federal Register** on July 22, 2010 entitled, "Medicare Program; Prospective Payment System and Consolidated Billing for Skilled Nursing Facilities for FY 2011."

DATES: *Effective Date:* This correction is effective October 1, 2010.

FOR FURTHER INFORMATION CONTACT: Bill Ullman, (410) 786–5667.

SUPPLEMENTARY INFORMATION:

I. Background

In FR Doc. 2010–17628 of July 22, 2010 (75 FR 42886), there were several technical errors that are identified and corrected in the "Correction of Errors" section below. The corrections described below are effective as if they had been included in the document published on July 22, 2010. Accordingly, the corrections are effective October 1, 2010.

II. Summary of Errors

We are correcting the titles and wage index columns (along with the resulting values) of Tables 8A and 8B, which appeared on page 42911 of the July 22, 2010 notice with comment period. These two tables illustrate the skilled nursing facility (SNF) prospective payment system (PPS) payment rate computations for a hypothetical "XYZ" SNF located in Cedar Rapids, IA (Urban CBSA 16300) under the RUG–IV and Hybrid RUG–III (HR–III) systems, respectively. In the title of the tables as well as in the third column ("Wage Index"), the wage index value for Cedar Rapids, IA is incorrectly displayed as 0.8858. Accordingly, in section III of this document ("Correction of Errors"), we are revising the entries in Tables 8A and 8B to reflect the correct wage index value of 0.8844. We are similarly revising the immediately preceding portion of the preamble text, which references the total PPS payment amounts displayed in these two tables. However, we note that the corresponding entry for CBSA 16300, as it appears in an addendum to the July 22, 2010 notice with comment period (Table A, "FY 2011 Wage Index for Urban Areas Based on CBSA Labor Market Areas"), already reflects the correct wage index value of 0.8844 (75 FR 42923). We are also revising the footnote that appears in Tables 8A and 8B to clarify that in these examples, all 10 of the Medicare days listed under the "CC2" RUG group would involve a resident with AIDS and, thus, would qualify for the special 128 percent adjustment under section 511 of the Medicare Prescription Drug, Improvement, and Modernization Act of 2003 (MMA) (Pub. L. 108–173, enacted on December 8, 2003).

III. Correction of Errors

In FR Doc. 2010–17628 (75 FR 42886), make the following corrections:

1. On page 42910, third column, in line five from the bottom of the page, the phrase "$41,979 for RUG–IV and $36,517 for HR–III, respectively" is revised to read "$41,935 for RUG–IV and $36,479 for HR–III, respectively".

2. On page 42911, Tables 8A and 8B are revised to read as follows:

3. On page 42911, underneath Table 8A and Table 8B, we removed the asterisk statement "*Reflects a 128 percent adjustment from section 511 of the MMA" and replaced it with "*Reflects a 128 percent adjustment from section 511 of the MMA. All CC2 days should be considered to be for a resident with AIDS."