Dated: June 14, 2007.

Catina Conner,

Acting Assistant Reports Clearance Officer, Centers for Disease Control and Prevention.

[FR Doc. E7–11936 Filed 6–19–07; 8:45 am] BILLING CODE 4163–18–P

DEPARTMENT OF HEALTH AND HUMAN SERVICES

Health Resources and Services Administration

Privacy Act of 1974: New System of Records

AGENCY: Health Resources and Services Administration (HRSA), HHS.

ACTION: Notification of new system of records.

SUMMARY: In accordance with the requirements of the Privacy Act, the Health Resources and Services Administration (HRSA) is publishing notice of a proposal to add a new system of records titled, "Information Center (IC) Integrated Clearinghouse System (IC/ICS)," System No. 09-15-0067. The HRSA IC/ICS will facilitate the delivery of publications and requested information by members of the general public. The HRSA IC/ICS will also enable HRSA to deliver information efficiently through physical mailings or broadcast e-mail messages to HRSA Grantee organizations and other interested parties.

DATES: HRSA invites interested parties to submit comments on the proposed New System of Records on or before July 30, 2007. HRSA has sent a Report of New Systems of Records to Congress and to the Office of Management and Budget (OMB). The New System of Records will be effective 40 days from the date submitted to OMB unless HRSA receives comments which would result in a contrary determination.

ADDRESSES: Please address comments to Donn Taylor, Health Resources and Services Administration, Division of Management Services, 5600 Fishers Lane, Room 14A–20, Rockville, Maryland 20857; Telephone (301) 443–0204. Comments received will be available for inspection at this same address from 9 a.m. to 3 p.m., Monday through Friday. This is not a toll-free number.

FOR FURTHER INFORMATION CONTACT: Tina Cheatham, Acting Director, Office of Communications, Health Resources and Services Administration, 5600 Fishers Lane, Room 14–27, Rockville, Maryland 20857, Telephone: 301–443–3376.

Please note this is not a toll free telephone number.

SUPPLEMENTARY INFORMATION: The

Health Resources and Services Administration proposes to establish a new system of records: "The HRSA Information Center (IC) Integrated Clearinghouse System (ICS)," HHS/ HRSA/Office of Communications. The HRSA Information Center provides easy access to a diversity of resources and a broad range of health information from over 70 Agency programs. The HRSA Information Center makes this information available to the public, health care professionals, policy makers and researchers to enhance their access to vital knowledge generated by HRSA supported public health programs.

Dated: May 31, 2007.

Elizabeth M. Duke,

Administrator.

[FR Doc. 07-3052 Filed 6-19-07; 8:45 am]

BILLING CODE 4165-15-M

DEPARTMENT OF HEALTH AND HUMAN SERVICES

Indian Health Service

Reimbursement Rates for Calendar Year 2007

AGENCY: Indian Health Service, HHS.

ACTION: Notice.

SUMMARY: Notice is given that the Director of Indian Health Service (IHS), under the authority of sections 321(a) and 322(b) of the Public Health Service Act (42 U.S.C. 248 and 249(b)), Public Law 83-568 (42 U.S.C. 2001(a)), and the Indian Health Care Improvement Act (25 U.S.C. 1601 et seq.), has approved the following rates for inpatient and outpatient medical care provided by IHS facilities for Calendar Year 2007 for Medicare and Medicaid beneficiaries and beneficiaries of other Federal programs. The Medicare Part A inpatient rates are excluded from the table below as they are paid based on the prospective payment system. Since the inpatient rates set forth below do not include all physician services and practitioner services, additional payment may be available to the extent that those services meet applicable requirements. Public Law 106-554, section 432, dated December 21, 2000, authorized IHS facilities to file Medicare Part B claims with the carrier for payment for physician and certain other practitioner services provided on or after July 1, 2001.

Inpatient Hospital Per Diem Rate (Excludes Physician/Practitioner Services)

Calendar Year 2007

Lower 48 States: \$1,725. Alaska: \$2,208.

Outpatient Per Visit Rate (Excluding Medicare)

Calendar Year 2007

Lower 48 States: \$256.

Alaska: \$398.

Outpatient Per Visit Rate (Medicare)

Calendar Year 2007

Lower 48 States: \$201. Alaska: \$356.

Medicare Part B Inpatient Ancillary Per

Calendar Year 2007

Diem Rate

Lower 48 States: \$353. Alaska: \$613.

Outpatient Surgery Rate (Medicare)

Established Medicare rates for freestanding Ambulatory Surgery Centers

Effective Date for Calendar Year 2007 Rates

Consistent with previous annual rate revisions, the Calendar Year 2007 rates will be effective for services provided on/or after January 1, 2007 to the extent consistent with payment authorities including the applicable Medicaid State plan.

Dated: January 4, 2007.

Charles W. Grim,

Assistant Surgeon General, Director, Indian Health Service.

[FR Doc. 07–3037 Filed 6–19–07; 8:45 am]

BILLING CODE 4165-16-M

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.

Synthetic Macrolides Inhibit Breast Cancer Migration

Description of Technology: This technology relates to the synthesis of several novel macrocylic compounds (macrolides), built upon a quinic acid-containing scaffold, which are potent inhibitors of tumor cell migration. Specifically, the new molecules have been shown to inhibit breast cancer cell migration in vitro.

Tumor metastasis or cell migration is a multi-step process in which primary tumor cells spread or migrate by invading adjacent tissues and/or metastasizing to distance sites. Thus, one approach to cancer treatment may be the inhibition of tumor migration. The initial observation that migrastatin, a macrolide natural product first isolated from a Streptomycete, inhibits tumor cell migration gave rise to the synthesis of the analogs with increased potency and tumor cell selectivity

reported here. *Applications:* These compounds may be the basis for new antimetastatic and antiangiogenic drugs. Some of the novel macrolides that have been designed and synthesized, inhibit tumor cell migration with low nanomolar to submicromolar IC₅₀ values via a mechanism that appears to be similar to that of migrastatin and its analogs. The synthetic protocol used is straight forward and relatively high yielding, and has the potential to be further simplified.

The new compounds and methods may be used to treat a pathologic condition that may be ameliorated by inhibiting or decreasing cell migration or metastasis, to decrease anchoragedependent growth of tumor cells, or to treat any pathologic condition characterized by neovascularization.

Advantages: The new molecules have been shown to inhibit breast cancer cell migration in vitro. Breast cancer is the most common female cancer in the United States, the second most common cause of death in women and the main cause of death in women ages 45 to 55.

Despite early diagnosis and treatment, recurrence of the cancer including distant tumor growth or metastases is common. Accordingly, there is a need for compounds, such as those described in this invention, that inhibit cell migration and angiogenesis.

Development Status: Synthesis of several analogs has been carried out; Migration of breast cancer cells has been demonstrated to be inhibited *in vitro* at sub-micromolar IC₅₀ values; The lead compound has been demonstrated not to be cytotoxic at levels up to 100 micromolar; Scaled up synthesis of the most potent macrolide is presently being scaled up to unable for future testing in a mouse model of breast cancer.

Inventors: Dr. Carole Bewley (NIDDK), Dr. Belhu B. Metaferia (NIDDK).

Publication: BB Metaferia, L Chen, HL Baker, XY Huang, CA Bewley. Synthetic macrolides that inhibit breast cancer cell migration in vitro. J Am Chem Soc. 2007 Mar 7;129(9):2434–2435. Epub 2007 Feb 13, doi 10.1021/ja068538d.

Patent Status: U.S. Provisional Application No. 60/900,151 filed 07 Feb 2007 (HHS Reference No. E-098-2007/ 0-US-01).

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

Licensing Contact: Michelle Booden,

Ph.D.; 301/451–7337;

boodenm@mail.nih.gov. Collaborative Research Opportunity: The National Institute of Diabetes and Digestive and Kidney Diseases, Laboratory of Bioorganic Chemistry, is seeking parties interested in collaborative research to develop larger scale syntheses of the most potent macrolides and/or analogs thereof, and the conduct toxicology and other efficacy studies related to these macrolides. Please contact Dr. Carole Bewley at caroleb@mail.nih.gov or Rochelle S. Blaustein at Rochelle.Blaustein@nih.gov for more information.

Immunotoxin With In-Vivo T Cell Suppressant Activity

Description of Invention: The invention concerns immunotoxins and methods of using the immunotoxins for the treatment of autoimmune diseases and T cell malignancies. The immunotoxins are targeted via an antibody that is specific to T cells. This allows the specific ablation of malignant T cells and resting T cells. The transient ablation of resting T cells can "reset" the immune system by accentuating tolerizing responses. The toxin portion of the immunotoxin is genetically engineered to maintain bioactivity when recombinantly produced in *Pichia*

pastoris. Data are available in transgenic animals expressing human CD3 ϵ which supports the effects of the immunotoxin against T cells.

Applications: Treatment of autoimmune diseases such as multiple sclerosis, lupus, type I diabetes, aplastic anemia; Treatment of T cell leukemias and lymphomas such as cutaneous T cell leukemia/lymphoma (CTCL).

Advantages: Specificity of the immunotoxin avoids the killing of non-T cells, reducing side-effects associated with other mechanisms of treatment (e.g., radiation and cyclophosphamide) such as infection and induced malignancy; A GMP production process has already been successfully implemented, and patient doses are available; All testing required for an FDA issued IND has been completed, allowing faster evaluation of the efficacy of the invention.

Benefits: New methods and compositions with limited side-effects have the potential to revolutionize treatment of autoimmune disease; provides an opportunity to capture a significant market share for the millions of people who suffer from an autoimmune disease.

Inventors: David Neville *et al.* (NIMH).

Patent Status: U.S. Patent No. 5,167,956 issued 01 Dec 1992 (HHS Reference No. E-012-1991/0-US-01); U.S. Patent No. 5,725,857 issued 10 Mar 1998 (HHS Reference No. E-012-1991) 2-US-01); U.S. Patent No. 6,632,928 issued 14 Oct 2003 (HHS Reference No. E-044-1997/0-US-07); U.S. Patent Application No. 10/435,567 filed 09 May 2003, which published as 2003/ 0185825 on 02 Oct 2003 (HHS Reference No. E-044-1997/0-US-08); U.S. Patent Application No. 10/296,085 filed 18 Nov 2002, which published as 2004/ 0127682 on 01 Jul 2004 (HHS Reference No. E-044-1997/1-US-06); Foreign rights are also available

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

Licensing Contact: David A.

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

Collaborative Research Opportunity: The National Institute of Mental Health, Laboratory of Molecular Biology, is seeking statements of capability or interest from parties interested in collaborative research to further develop, evaluate, or commercialize methods of using the immunotoxins for the treatment of autoimmune diseases and T cell malignancies. Please contact David Neville at davidn@mail.nih.gov for more information.

Dated: June 13, 2007.

Steven M. Ferguson,

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

[FR Doc. E7–11824 Filed 6–19–07; 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.

NUP98-HOXD13 Transgenic Mice

Description of Technology: Myelodysplastic syndrome (MDS) is collection of closely related blood diseases that arise in the bone marrow characterized by anemia, neutropenia, and thrombocytopenia resulting from hematopoietic stem cell disorders. A variety of genetic aberrations have been associated with MDS, including chromosomal translocations of the NUP98 gene. The only current curative therapy for MDS is allogeneic bone marrow transplant. Without bone marrow transplant, patients either die of progressive pancytopenia or following transformation of MDS to acute myeloid leukemia. Progress in understanding and treating MDS has been hampered by a lack of an animal model that accurately recapitulates all of the features of human MDS. Utilizing a NUP98-HOXD13 (hereafter NHD13) fusion gene, a mouse model was

developed to elucidate the biology of MDS. Genetically engineered mice that express an NHD13 transgene display all of the phenotypic features of MDS including peripheral blood cytopenia, bone marrow dysplasia, and transformation to acute leukemia. These mice provide an accurate preclinical model for MDS.

Applications: Model to study MDS and evaluate MDS therapy.

Market: 15,000–20,000 new cases of MDS are diagnosed in the U.S.; 80–90% of patients are older than 60 years old.

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

Inventors: Peter D. Aplan *et al.* (NCI). *Publications:*

- 1. YW Lin *et al.* Notch1 mutations are important for leukemic transformation in murine models of precursor-T leukemia/lymphoma. Blood. 2006 Mar 15;107(6):2540–2543.
- 2. YW Lin *et al.*, NUP98-HOXD13 transgenic mice develop a highly penetrant, severe myelodysplastic syndrome that progresses to acute leukemia. Blood. 2005 Jul 1;106(1):287–295.

Patent Status: HHS Reference No. E-071-2007/0—Research Tool.

Licensing Status: Available for non-exclusive licensing.

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

Collaborative Research Opportunity: The Leukemia Biology Section, Genetics Branch, National Cancer Institute is seeking statements of capability or interest from parties interested in collaborative research to further develop, evaluate, or commercialize the NHD13 mouse model. Please contact John D. Hewes, Ph.D. at 301–435–3121 or hewesj@mail.nih.gov for more information.

Identification of Ovarian Cancer Tumor Markers and Therapeutic Agents

Description of Technology: Germline mutations of BRCA1 and BRCA2 tumor suppressor genes are responsible for 5%-10% of all epithelial ovarian cancers. However, little is known about the molecular mechanisms involved in BRCA1 and/or BRCA2 mutationassociated (termed BRCA-linked) ovarian carcinogenesis. To elucidate their pathways, microarrays were used to compare gene expression patterns in ovarian cancers associated with BRCA1 or BRCA2 mutations with gene expression patterns in sporadic epithelial ovarian cancers to identify patterns common to both hereditary and sporadic tumors. As a result of this analysis, genes that are upregulated in ovarian cancer were identified.

Approximately two-thirds of the sequences identified were previously known genes, while approximately one-third were expressed sequence tags, representing sequences that are cloned and identified but not yet characterized. Eighty-three genes were over-expressed in 50% of all tumors and these over-expressed sequences may be used as markers for ovarian cancer and/or targets for therapy.

Applications: Method to diagnose ovarian cancer; Method to treat ovarian cancer with therapeutics that target ovarian biomarkers; Ovarian cancer therapeutics that inhibit ovarian cancer

markers such as siRNA.

Market: Estimated 180,000 new cases of breast cancer in the U.S. in 2007; Estimated 41,000 deaths due to breast cancer in the U.S. in 2007.

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

Inventors: Amir Jazaeri (NCI), Edison T. Liu (NCI), et al.

Publications:

- 1. AA Jazaeri *et al.* BRCA1-mediated repression of select X chromosome genes. J Transl Med. 2004 Sep 21;2(1):32.
- 2. AA Jazaeri *et al.* Molecular determinants of tumor differentiation in papillary serous ovarian carcinoma. Mol Carcinog. 2003 Feb;36(2):53–59.
- 3. AA Jazaeri *et al.* Gene expression profiles of BRCA1-linked, BRCA2-linked, and sporadic ovarian cancers. J Natl Cancer Inst. 2002 Jul 3;94(13):990–1000.

Patent Status: U.S. Provisional Application No. 60/357,031 filed 13 Feb 2002 (HHS Reference No. E-310-2001/0-US-01); PCT Patent Application No. PCT/US2003/046888 filed 13 Feb 2003 (HHS Reference No. E-310-2001/0-PCT-02); U.S. Patent Application No. 10/505,680 filed 12 Aug 2004 (HHS Reference No. E-310-2001/0-US-03).

Licensing Status: Available for exclusive or non-exclusive licensing. Licensing Contact: Jennifer Wong; 301/435–4633; wongje@mail.nih.gov.

Tumor Markers in Ovarian Cancer

Description of Technology: Ovarian cancer is one of the most common forms of neoplasia in women. Although advanced ovarian cancer has only a 20–30% survival rate, an estimated 90% of cases are effectively treated when detected early. However, few symptoms are associated with early ovarian cancer, and approximately 25% of ovarian cancer cases are diagnosed before it metastasizes. Utilizing SAGE analysis, a unique set of ovarian cancer biomarkers has been identified that are highly expressed in ovarian epithelial tumor