proposed information collections must be received by the OMB desk officer at the address below, no later than 5 p.m. on *May 11, 2009*.

OMB, Office of Information and Regulatory Affairs, Attention: CMS Desk Officer, Fax Number: (202) 395–6974, e-mail: *OIRA_submission@omb.eop.gov*.

Dated: April 3, 2009.

Michelle Shortt,

Director, Regulations Development Group, Office of Strategic Operations and Regulatory Affairs.

[FR Doc. E9–8223 Filed 4–9–09; 8:45 am]

BILLING CODE 4120–01–P

DEPARTMENT OF HEALTH AND HUMAN SERVICES

Administration for Children and Families

Proposed Information Collection Activity; Comment Request

Proposed Projects

Title: Request for Assistance for Child Victims of Human Trafficking.

OMB No.: New collection.

Description: The William Wilberforce Trafficking Victims Protection Reauthorization Act (TVPRA) of 2008, Public Law 110–457, directs the U.S. Secretary of Health and Human Services (HHS), upon receipt of credible information that a non-U.S. citizen, non-lawful permanent resident (alien) child may have been subjected to a severe form of trafficking in persons and is seeking Federal assistance available to victims of trafficking, to promptly determine if the child is eligible for interim assistance. The law further directs the Secretary of HHS to determine if the child is eligible for assistance as a victim of a severe forms of trafficking in persons after consultation with the Attorney General, the Secretary of Homeland Security, and nongovernmental organizations with expertise on victims of severe forms of trafficking.

In developing procedures for collecting the necessary information from potential child victims of trafficking, their case managers, attorneys, or other representatives to allow HHS to grant interim eligibility, HHS devised a form. HHS has determined that the use of a standard form to collect information is the best way to ensure requestors are notified of their option to request assistance for child victims of trafficking and to make prompt and consistent determinations about the child's eligibility for interim assistance.

Specifically, the form asks the requestor for his/her identifying information, for information on the child, information describing the type of trafficking and circumstances surrounding the situation, and the strengths and needs of the child. The form also asks the requestor to verify the information contained in the form because the information could be the basis for a determination of an alien child's eligibility for federally funded benefits.

Finally, the form takes into consideration the need to compile information regarding a child's circumstances and experiences in a non-directive, child-friendly way, and assists the potential requestor in assessing whether the child may have been subjected to trafficking in persons. The information provided through the completion of a Request for Assistance for Child Victims of Human Trafficking form will enable HHS to make prompt determinations regarding the eligibility of an alien child for interim assistance, inform HHS' determination regarding the child's eligibility for assistance as a victim of a severe form of trafficking in persons, facilitate the required consultation process, and enable HHS to assess and address potential child protection issues.

Respondents: Representatives of governmental and nongovernmental entities providing social, legal, or protective services to a non-U.S. citizen, non-lawful permanent resident (alien) individual under the age of 18 (child) in the United States who may have been subjected to a severe form of trafficking in persons.

ANNUAL BURDEN ESTIMATES

Instrument	Number of respondents	Number of responses per respondent	Average burden hours per response	Total burden hours
Request for Assistance for Child Victims of Human Trafficking	50	1	1	50
Estimated Total Annual Burden Hours				50

In compliance with the requirements of Section 506(c)(2)(A) of the Paperwork Reduction Act of 1995, the Administration for Children and Families is soliciting public comment on the specific aspects of the information collection described above. Copies of the proposed collection of information can be obtained and comments may be forwarded by writing to the Administration for Children and Families, Office of Administration, Office of Information Services, 370 L'Enfant Promenade, SW., Washington, DC 20447, Attn: ACF Reports Clearance Officer. E-mail address: *infocollection@acf.hhs.gov.* All requests should be identified by the title of the information collection.

The Department specifically requests comments on: (a) Whether the proposed collection of information is necessary for the proper performance of the functions of the agency, including whether the information shall have practical utility; (b) the accuracy of the agency's estimate of the burden of the proposed collection of information; (c) the quality, utility, and clarity of the information to be collected; and (d) ways to minimize the burden of the collection of information on respondents, including through the use of automated collection techniques or other forms of information technology. Consideration will be given to comments and suggestions submitted within 60 days of this publication.

Dated: April 6, 2009.

Janean Chambers,

Reports Clearance Officer.

[FR Doc. E9–8139 Filed 4–9–09; 8:45 am]

BILLING CODE 4184–01–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.

Treating Cancer Through Immunotherapy With Herceptin-Based Receptors Specific for ErbB2 (Her2/Neu)

Description of Technology: There is an urgent need to develop new therapeutic strategies for patients with cancer that combine fewer side-effects and more specific anti-tumor activity. Adoptive immunotherapy is a promising new approach to cancer treatment that engineers an individual's innate and adaptive immune system to fight against specific diseases, including cancer.

Chimeric antigen receptors (CARs) are hybrid proteins consisting of the portion of an antibody that recognizes a tumor-associated antigen (TAA) fused to protein domains that signal to activate the CAR-expressing cell. Human cells that express CARs, most notably T cells, can recognize specific tumor antigens with high reactivity to mediate an immune response that promotes tumor killing in targeted cancer cells.

Scientists at the National Institutes of Health (NIH) have developed CARs with high affinity for the ErbB2 (also known as Her2/Neu) antigen, which is overexpressed on a variety of cancer cells, including lung, breast, colorectal, ovary, prostate, and head and neck squamous cell cancer. These ErbB2-specific CARs are herceptin-based receptors composed of the part of a humanized herceptin antibody that recognizes ErbB2 and a portion of the T cell receptor (TCR)-related protein, CD3. The herceptin-CAR framework was selected since the herceptin monoclonal antibody has been proven to be an effective treatment for breast cancer. These ErbB2-specific CARs expressed in the context of T cells could prove to be powerful new immunotherapeutic tools for attacking ErbB2+ tumors after their infusion into patients.

Applications:

• Immunotherapeutics to treat and/or prevent the reoccurrence of a variety of human cancers that overexpress human ErbB2 by inserting herceptin-based CAR sequences into patient T cells.

• A drug component of a combination immunotherapy regimen aimed at targeting the specific tumor-associated antigens expressed by cancer cells within individual patients.

Advantages:

• This discovery is widely applicable to many different cancers: ErbB2 is overexpressed in many cancers, including lung, breast, colorectal, ovary, prostate, and head and neck squamous cell cancer. Anti-ErbB2 CAR immunotherapy could treat a variety of cancer types while reducing the side-effects of treatment.

• The technology is based on an already approved antibody: The herceptin monoclonal antibody is a U.S. Food and Drug Administration (FDA) approved treatment for ErbB2+ breast cancer patients. This current herceptin-based CAR immunotherapy treatment is predicted to draw favorable consideration from the FDA as it proceeds through clinical trials.

Development Status: This technology will soon enter the clinical stage of development since the inventors plan to initiate clinical trials using CAR engineered lymphocytes for adoptive immunotherapy of cancer. A clinical protocol is under review.

Market: Cancer continues to be a medical and financial burden on U.S. public health. Statistically, in the U.S. cancer is the second leading cause of death with over 565,000 deaths reported in 2008 and almost 1.5 million new cases were reported (excluding some skin cancers) in 2008. In 2007, the NIH estimated that the overall cost of cancer was $219.2 billion dollars and $89 billion went to direct medical costs. The fight against cancer will continue to benefit from the development of new therapeutics aimed at treating individual patients.

Inventors: Steven A. Rosenberg et al. (NCI).

Patent Status: U.S. Provisional Application No. 61/154,080 filed 20 Feb 2009 (HHS Reference No. E–045–2009/0–US–01).

Licensing Status: Available for licensing.

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

Collaborative Research Opportunity: Dr. Steven A. Rosenberg of the NCI Surgery Branch is seeking statements of capability or interest from parties interested in collaborative research to further develop, evaluate, or commercialize therapeutic T cell receptor technologies. Please contact John D. Hewes, PhD at 301–435–3121 or *hewesj@mail.nih.gov* for more information.

Peptides for Treating Mesothelin- and/or CA125–Expressing Cancers

Description of Technology: Mesothelin is a cell surface glycoprotein that is highly expressed in many cancers, including malignant mesothelioma and ovarian cancer. Mesothelin interacts with another cell surface protein that is also highly expressed on some cancer cells: CA125 (MUC16). Evidence indicates that this interaction mediates cell adhesion during tumor implantation and metastasis. This suggested that the disruption of the mesothelin-CA125 interaction may prevent the growth and spreading of tumors.

NIH inventors have generated specific peptides, based on the CA125 binding domain of mesothelin, that block the interaction between mesothelin and CA125. Significantly, blocking the interaction disrupted cell adhesion in cancer cells expressing both mesothelin and CA125. Antibodies that recognize the specific mesothelin peptides were also capable of disrupting the mesothelin-CA125 interaction. The peptides bound CA125 on cancer cells. As a result, these peptides are excellent candidates for a new cancer therapeutic.

Applications:

• Treatment of cancers that express mesothelin and CA125 by disrupting the mesothelin-CA125 interaction

• Treatment of CA125-expressing cancers by binding cell surface CA125

• Direct inhibition of the mesothelin-CA125 interaction with specific peptides

• Inhibition of the mesothelin-CA125 interaction by using specific peptides as a vaccine

Advantages:

• The specific peptides are from a human protein and may not elicit a strong immunogenic response that would inhibit its effectiveness as a blocking agent

• Any immunogenic response to the specific peptides would have a potential beneficial effect by generating antibodies that also inhibit mesothelin-CA125 interaction and inhibit CA125-expressing tumor growth

Inventors: Mitchell Ho (NCI) *et al.*

Patent Status:

• PCT Application PCT/US2008/85743 (HHS Reference E–336–2008/0–PCT–01)

For more information, see:

• US Patent 6,083,502 entitled ''Mesothelium Antigen and Methods and Kits For Targeting It''

• PCT Application PCT/US97/0224 entitled ''Mesothelium Antigen and Methods and Kits For Targeting It''

• US Patent 6,809,184 entitled ''Antibodies, Including Fv Molecules, and Immunoconjugates Having High Binding Affinity for Mesothelin and Methods for Their Use''

• PCT Application PCT/US98/25270 entitled ''Antibodies, Including Fv Molecules, and Immunoconjugates Having High Binding Affinity for Mesothelin and Methods for Their Use''

• US Patent 7,081,518 entitled ''Anti-mesothelin antibodies having high binding affinity''

• PCT Application PCT/US00/14829 entitled ''Immunoconjugates Having High Binding Affinity Improvement of scFVsr Ab's with Higher Affinity for Mesothelin''

Licensing Contact: David A. Lambertson, PhD; 301–435–4632; *lambertsond@mail.nih.gov.*

Collaborative Research Opportunity: The National Cancer Institute, Laboratory of Molecular Biology, is seeking statements of capability or interest from parties interested in collaborative research to further develop, evaluate, or commercialize peptides for treating mesothelin- and/or CA125-expressing cancers. Please contact Mitchell Ho at 301–451–8727 and/or *homi@mail.nih.gov* for more information.

An Imaging Radiotracer for the Noninvasive Detection of HER2-Positive Tumors

Description of Technology: Investigators at the NIH have developed a novel imaging radiotracer composed of an Affibody® molecule uniquely labeled with 18F for noninvasively locating and measuring the expression of HER2 breast cancer biomarker in tumors anywhere in the body. The over expression of HER2 in cells is a diagnostic marker for a particularly aggressive form of breast cancer. Currently, localized biopsies are needed to diagnose HER2-positive breast cancer. Noninvasive detection of HER2-positive cells in whole body will help to identify patients that can benefit from HER2-targeted therapies such as the monoclonal antibody trastuzumab. This imaging compound will also be useful for monitoring the tumor response to HER2-targeted therapies. The use of Affibody® molecule is advantageous because it is recombinant protein of relatively small size (more than 20 times smaller than antibodies), readily producible and having a high binding affinity to HER2. This allows the imaging compound to permeate the body easily and bind to the HER2-positive cells selectively. Conjugating Affibody® molecule to the positron-emitting radionuclide 18F enables noninvasive imaging using positron emission tomography (PET). The utility of this targeted radiotracer for detecting HER2-positive tumors has already been validated in animals.

Applications:

• Diagnostic and prognostic for HER2-positive tumors in breast cancer patients.

• Monitoring effectiveness of HER2-targeted therapy.

• Research tool for the in vivo study of HER2-positive carcinomas.

Advantages:

• Noninvasive.

• Detection of metastasis of HER2-positive tumors.

• Timely monitoring of tumor response to therapy.

• Improved accuracy in prognosis of patient survival.

Development Status: Pre-clinical in vitro and in vivo data available

Market:

• Breast cancer is the second leading cause of cancer death in women.

• In 2008, an estimated 182,460 new cases of invasive breast cancer were expected among women in the United States.

Inventors: Jacek Capala (NCI) *et al.*

Publications:

1. D Kiesewetter *et al.* Radiolabeling of HER2-specific Affibody® molecule with F–18. J Fluor Chem. 2008 Sep;129(9):799–806, doi:10.1016/j.jfluchem.2008.06.021.

2. Kramer-Marek *et al.* [18F]FBEM–ZHER2:342–Affibody molecule—a new molecular tracer for in vivo monitoring of HER2 expression by positron emission tomography. Eur J Nucl Med Mol Imaging. 2008 May;35(5):100818. doi:10.1016/j.jphotobiol.2006.08.011.

Patent Status:

U.S. Provisional Application No. 60/891,875 filed 27 Feb 2007 (HHS Reference No. E–086–2007/0–US–01)

PCT Application No. PCT/US2008/055144 filed 27 Feb 2008, which published as WO 2008/118601 on 02 Oct 2008 (HHS Reference No. E–086–2007/0–PCT–02)

Licensing Status: Available for licensing.

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

Collaborative Research Opportunity: The National Cancer Institute, Center for Cancer Research, Radiation Oncology Branch, is seeking statements of capability or interest from parties interested in collaborative research to further develop, evaluate, or commercialize Affibody® molecules for diagnosis and molecular therapy of HER1- or HER2-positive tumors. Please contact John D. Hewes, PhD at 301–435–3121 or *hewesj@mail.nih.gov* for more information.

Small-Molecule TSH Receptor Modulators for Diagnosis and Treatment of Thyroid Disease and Cancer

Description of Technology: NIH investigators have discovered a series of low molecular weight thyroid-stimulating hormone (TSH) receptor modulators for use in evaluation and treatment of thyroid diseases, including thyroid cancer, hypothyroidism, and hyperthyroidism. Certain compounds encompassed by this technology are more potent and/or more specific TSH receptor activators than currently-available compounds; also, as small molecules, these compounds are orally available and are expected to be less costly and more straightforward to produce than recombinant protein counterparts currently on the market.

According to the National Cancer Institute, over 37,000 new cases of thyroid cancer were diagnosed in the United States in 2008, and over 1,500 people died of this disease. These numbers reflect a progressive increase in the incidence of thyroid cancer over the last several years. Because most cases of thyroid cancer are diagnosed in patients between the ages of 20 and 54, these patients will undergo decades of follow-up monitoring after cancer treatment. For the last decade, recombinant TSH protein has been used in this follow-up to increase detection sensitivity for recurrent or metastatic thyroid cancer, and to eliminate side effects associated with withdrawal of hormone replacement therapy. A small-molecule TSH receptor agonist encompassed by this technology would have utility similar to recombinant TSH, but would have several distinct advantages. For example, as a small molecule, rather than a recombinant protein, such a compound would be orally available, and would be less difficult and expensive to produce. These compounds are also more potent and/or specific for the TSH receptor than other known small-molecule TSH receptor agonists. In addition to use in thyroid cancer screening, these compounds may also be useful for adjunctive treatment (with radioactive iodide) of thyroid cancer, and certain forms of hypothyroidism.

Hyperthyroidism, or an overactive thyroid gland, affects about 1% of people in the United States and is often caused by autoimmune over-stimulation of the thyroid gland (Graves' disease), or by thyroid tumors. Drugs currently used for treatment of hyperthyroidism inhibit synthesis of thyroid hormones; the TSH receptor antagonist compounds encompassed by this technology have the advantage of directly inhibiting activity of the TSH receptor, rather than inhibiting thyroid hormone synthesis.

Applications:

• Diagnostic tools for evaluation and treatment of thyroid cancer.

• Therapeutics for thyroid cancer, hyperthyroidism, and hypothyroidism.

Market: Approximately 1 in 13 Americans suffers from a thyroid disorder, and 10 million have a thyroid-related condition that requires ongoing immunodiagnostic monitoring.

Development Status: Early stage.

Inventors: Marvin C. Gershengorn *et al.* (NIDDK)

Publications:

1. S Moore, H Jaeschke, G Kleinau, S Neumann, S Costanzi, JK Jiang, J Childress, BM Raaka, A Colson, R Paschke, G Krause, CJ Thomas, MC Gershengorn. Evaluation of small-molecule modulators of the luteinizing hormone/choriogonadotropin and thyroid stimulating hormone receptors: structure-activity relationships and selective binding patterns. J Med Chem. 2006 Jun 29;49(13):3888–3896.

2. S Neumann, G Kleinau, S Costanzi, S Moore, BM Raaka, CJ Thomas, G Krause, MC Gershengorn. A low molecular weight antagonist for the human thyrotropin receptor with therapeutic potential for hyperthyroidism. Endocrinology 2008 Dec;149(12):5945–5950.

3. Unpublished data are also available for review under a CDA.

Patent Status:

HHS Reference Nos. E–223–2006/0 and E–223–2006/1 —

• International Patent Application No. PCT/US2007/011951 filed 17 May 2007, which published as WO 2007/136776 on 29 Nov 2007.

• National Phase entered in Australia, Canada, Europe, Japan, and the United States.

HHS Reference No. E–284–2008/0 —

• International Patent Application No. PCT/US2008/011958 filed 20 Oct 2008.

Licensing Status: Available for licensing.

Licensing Contact: Tara L. Kirby, PhD; 301–435–4426; *tarak@mail.nih.gov.*

Collaborative Research Opportunity: The NIDDK, Clinical Endocrinology Branch is seeking statements of capability or interest from parties interested in collaborative research to further develop, evaluate, or commercialize small molecule TSH receptor modulators. Please contact Patricia Mello Lake; 301–451–3636; *lakep@mail.nih.gov* for more information.

April 3, 2009.

Richard U. Rodriguez,

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

[FR Doc. E9–8208 Filed 4–9–09; 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.

Selective Killing of Cancer Cells by Inhibition of Geminin

Description of Technology: The current strategy for developing cancer therapeutics is to identify unique differences between cancer cells and normal cells that can serve as specific targets for chemotherapeutic drugs, thereby allowing elimination of cancer cells with minimal toxicity to normal tissues. Geminin, an inhibitor of DNA replication, is typically undetectable in normal cells while rapidly proliferating cancer cells express geminin and hence could be targeted for cancer treatment. The NIH researchers have discovered that inhibition of geminin expression induced DNA re-replication in most of the tested cancer cell lines, but not in matched non-cancer cell lines from the same tissues. DNA re-replication occurs when DNA synthesis is initiated multiple times from the same replication origin during one cycle of cell division resulting in DNA damage which halts cell proliferation and induces apoptosis in a wide variety of cancer cells. The researchers also analyzed the effect of suppression of geminin expression on apoptosis and cell survival in cancer and non-cancer cell lines. They found that the geminin siRNA induced apoptosis in a colon carcinoma cell line, but not in a normal skin fibroblast cell line. Furthermore, suppression of geminin expression markedly reduced cell survival of several cancer cell lines, but not non-cancer cell lines. Therefore, suppressing the level of geminin expression can be potentially used to selectively kill cancer cells.

Applications: Therapeutic for treating breast, colon and rectal, kidney (renal cell), lung, brain, and bone cancers.

Advantages: Targeted therapeutic; No requirement for use of other cell cycle inhibitors

Market: Cancer continues to be a burden to the public health of Americans. After heart disease, cancer is the most common cause of death in the United States. For 2008, it was estimated that about 565,650 Americans were expected to die of cancer. The incidence of cancer has been dropping over the years but it is estimated that over 1.4 million Americans would be diagnosed with cancer in 2008. Therefore, there is a continued need for the development of new therapies to effectively treat this disease.

Inventors: Wenge Zhu and Melvin L. DePamphilis (NICHD)

Publications: Paper accepted for publication in Cancer Research.

Patent Status: U.S. Provisional Application No. 61/106,465 filed 17 Oct 2008 (HHS Reference No. E–324–2008/0–US–01)

Licensing Status: Available for licensing.

Licensing Contact: Betty Tong, PhD; 301–594–6565; *tongb@mail.nih.gov.*

Transgenic Mice in Which the Gene for MCP–1 Is Deleted

Description of Technology: Dr. Yoshimura has developed a transgenic mouse which does not express the chemokine MCP–1 due to a deletion of the gene for MCP–1. MCP–1 is a CC chemokine which is responsible for recruiting monocytes into sites of