next-generation smallpox vaccines in humans; (3) discuss the most appropriate methods to bridge immunogenicity of next-generation smallpox vaccines to licensed smallpox vaccines in clinical trials; and (4) discuss viable methods of extrapolating clinical efficacy of next-generation smallpox vaccines from immunogenicity and efficacy data from relevant animal models.

Transcripts: Transcripts of the public workshop may be requested in writing from the Division of Freedom of Information Office (ELEM–1029), Food and Drug Administration, 12420 Parklawn Dr., Element Bldg., Rockville, MD 20857, approximately 15 working days after the public workshop at a cost of 10 cents per page. A transcript of the public workshop will be available on the Internet at *http://www.fda.gov/BiologicsBloodVaccines/NewsEvents/WorkshopsMeetingsConferences/TranscriptsMinutes/default.htm.*

Dated: August 4, 2011.

Leslie Kux,

Acting Assistant Commissioner for Policy.

[FR Doc. 2011–20367 Filed 8–10–11; 8:45 am]

BILLING CODE 4160–01–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.

Tumor Markers for Potentially Predicting Outcome of Anti-angiogenesis Therapy

Description of Technology: During the past decade, anti-angiogenesis therapy has evolved as a promising approach to the treatment of cancer. However, a significant fraction of patients do not benefit from anti-angiogenesis therapy, either by itself or in combination with chemotherapy. A significant need remains for a means of predicting clinical benefit from anti-angiogenesis therapy.

Researchers at the National Cancer Institute, NIH, have identified tumor cell apoptosis, p53, and HER2 as having potential predictive significance for treatment outcome in breast cancer patients who received anti-angiogenesis therapy in combination with chemotherapy. The researchers have developed a quantitative antibody-based testing method for correlating expression of p53 and HER2 and tumor apoptosis with clinical outcome. These markers can be potentially applied to predict which patients should receive anti-angiogenesis therapy plus chemotherapy.

Potential Commercial Applications:

- A diagnostic kit for predicting benefit of anti-angiogenesis therapy plus chemotherapy in breast cancer patients.
- A testing service for breast cancer patients.

Competitive Advantages:

- The clinical predictive markers p53, HER2 and tumor apoptosis indicators are easily and readily evaluated using the new assay.
- The new assay is potentially useful to determine which patients should or should not receive anti-angiogenesis therapy plus chemotherapy for longer survival and progression-free survival in patients with breast cancer.
- A study with a large sample size will be planned by the inventors and potential collaborators.

Development Stage:

- Pilot.
- In vivo data available (human).

Inventors: Sherry Yang (NCI), Seth Steinberg (NCI), *et al.*

Publication: Yang S, *et al.* p53, HER2 and tumor cell apoptosis correlate with clinical outcome after neoadjuvant bevacizumab plus chemotherapy in breast cancer. Int J Oncol. 2011 May; 38(5):1445–1452. [PMID 21399868]

Intellectual Property: HHS Reference No. E–096–2011/0—U.S. Patent Application No. 61/448,092 filed 01 March 2011

Licensing Contact: Patrick McCue, Ph.D.; 301–435–5560; mccuepat@mail.nih.gov

Collaborative Research Opportunity: The National Clinical Target Validation Laboratory, DCTD, NCI, NIH, is seeking statements of capability or interest from parties interested in collaborative research to further develop, evaluate, or commercialize p53, tumor apoptosis, and HER2 as markers for anti-angiogenesis therapy. For collaboration opportunities, please contact John Hewes, Ph.D. at *hewesj@mail.nih.gov.*

TRRAP and GRIN2A Mutations for the Diagnosis and Treatment of Melanoma

Description of Technology: Using whole-exome sequencing of matched normal and metastatic tumor DNAs, researchers at the NIH have identified several novel somatic (*e.g.,* tumor-specific) alterations, many of which have not previously been known to be genetically altered in tumors or linked to melanoma. In particular, the researchers identified a recurrent ''hotspot'' mutation in the transformation/transcription domain-associated protein (TRRAP) gene, found the glutamate receptor ionotropic N-methyl D-aspartate 2A (GRIN2A) gene as a highly mutated in melanoma, and have shown that the majority of melanoma tumors have alterations in genes encoding members of the glutamate signaling pathway. Therefore, this technology not only provides a comprehensive map of genetic alterations in melanoma, but has important diagnostic and therapeutic applications. Mutations in the TRRAP and GRIN2A genes can be used as diagnostic markers for melanoma and may serve as therapeutic targets in the treatment of melanoma. In addition, glutamate antagonists have previously been shown to inhibit proliferation of human tumor cells, and therefore further investigation of the pathway in melanoma could allow for the identification of new therapeutic proteins that target this pathway.

Potential Commercial Applications:

- Diagnostic array for the detection of TRRAP and GRIN2A mutations.
- Method of identifying TRRAP and GRIN2A inhibitors as therapeutic agents to treat malignant melanoma patients.
- Method of selecting a therapy based on the presence of TRRAP and GRIN2A mutations.

Competitive Advantages:

- Complete analysis of melanoma exome alterations.
- TRRAP, GRIN2A, and the other identified mutations are highly frequent and/or highly mutated in melanomas.
- Glutamate antagonists have already been shown to inhibit tumor growth. Thus, this technology may prove useful

for the development of novel diagnostic tests and therapeutics.

Development Stage: Pre-clinical.

Inventors: Yardena Samuels and Xiaomu Wei (NHGRI).

Publication: Wei X, et al. Exome sequencing identifies GRIN2A as frequently mutated in melanoma. Nat Genet. 2011 May;43(5):442–446. [PMID: 21499247].

Intellectual Property:

• HHS Reference No. E–013–2011/0—U.S. Provisional Application No. 61/462,471 filed 02 February 2011.

• Research Tool—Patent protection is not being pursued for the TRRAP and GRIN2A melanoma metastatic cell lines.

Related Technologies:

• HHS Reference No. E–272–2008/0—U.S. Patent Application No. 13/128,125 filed 06 May 2011, European and Australian applications filed; Mutations of the ERBB4 Gene in Melanoma.

• HHS Reference No. E–229–2010/0—Research Tool; ERBB4 Mutations Identified in Human Melanoma Metastasis Cell Lines (2690, 2379, 2197, 2183, 2535, 2645, 1770, 2359, 2238, 2319, 2190).

• HHS Reference No. E–232–2010/0—Research Tool; Isocitrate Dehydrogenase 1 (IDH1) R132 Mutation Human Melanoma Metastasis Cell Line.

Licensing Contact: Whitney Hastings; 301–451–7337; hastingw@mail.nih.gov.

Cells and Nanoparticles With Altered Protein Expression Patterns Useful for the Modulation of T Cell Activity for Immunotherapy

Description of Technology: NIH scientists have developed human cells and nanoparticles to enhance immunotherapy. Specifically, researchers have identified that cells or nanoparticles expressing a high temperature requirement serine peptidase 1 (HtrA1) activator and/or a cytokine-induced Src homology 2 protein (CIS) inhibitor are capable of increasing T cell activity. These compositions can be used primarily in T cell immunotherapy against various cancers and infectious diseases where enhanced T cell activity is beneficial. Conversely, cells or nanoparticles that express a HtrA1 inhibitor and/or a CIS activator can suppress T cell activity. These compositions can be utilized to treat various auto- or alloimmune diseases and can be used to prevent transplant rejections.

HtrA1 (also known as L56, ARMD7, ORF480, and PRSS11) is a serine protease that is known to inhibit the TGF-beta family proteins. CIS (also known as G18, SOCS, CIS–1, and CISH) is a member of the suppression of cytokine signaling (SOCS) family of proteins and inhibit the JAK/STAT signaling pathways. CIS acts to inhibit HtrA1 and repress cell activation targets. Immunotherapy, although an effective treatment strategy, sometimes fails when cells lose activity. T cells adoptively transferred into patients where CIS is inhibited and/or HtrA1 is activated should maintain their activity and lead to more successful adoptive T cell transfers.

Potential Commercial Applications:

• Immunotherapy for cancer or infectious diseases using human cells or nanoparticles expressing an HtrA1 activator and/or a CIS inhibitor

• Therapeutic for treating autoimmune diseases using human cells and or nanoparticles expressing an HtrA1 inhibitor and/or a CIS activator

• Agents expressing an HtrA1 inhibitor and/or a CIS activator to prevent organ, tissue, or cell transplant rejection and treat alloimmune diseases, such as graft-versus-host disease

• Components of a combination therapy to increase or suppress T cell activity in a patient

Competitive Advantages:

• Some patients do not respond to T cell immunotherapy due to lack of cell persistence, survival, or activity as well as for other poorly understood reasons. Modifying HtrA1 and CIS in currently existing T cell immunotherapies should increase the success rate of these therapies by increasing the persistence and survival of the infused cells.

• T cells can become ''exhausted'' as they mature following activation by target antigen. Cells with altered expression of HtrA1 and/or CIS may be able to avoid exhaustion after repeated activation.

Development Stage:

• Pre-clinical.

• In vitro data available.

• In vivo data available (animal).

Inventors: Douglas C. Palmer and Nicholas P. Restifo (NCI).

Publication: Palmer DC and Restifo NP. Suppressors of cytokine signaling (SOCS) in T cell differentiation, maturation, and function. Trends Immunol. 2009 Dec;30(12):592–602. [PMID 19879803].

Intellectual Property: HHS Reference No. E–069–2010/0—U.S. Patent Application No. 61/420,825 filed 08 December 2010.

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

Dated: August 5, 2011.

Richard U. Rodriguez,

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

[FR Doc. 2011–20447 Filed 8–10–11; 8:45 am]

BILLING CODE 4140–01–P

DEPARTMENT OF HEALTH AND HUMAN SERVICES

National Institutes of Health National Institute of Environmental Health Sciences; Notice of Meeting

Pursuant to section 10(a) of the Federal Advisory Committee Act, as amended (5 U.S.C. App.), notice is hereby given of a meeting of the Interagency Breast Cancer and Environmental Research Coordinating Committee.

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: Interagency Breast Cancer and Environmental Research Coordinating Committee.

Date: September 26–27, 2011.

Time: 8:30 a.m. to 5 p.m.

Agenda: The purpose of the meeting is to continue the work of the Committee, to share and coordinate information on existing research activities, & make recommendations to NIH & other Federal agencies on how to improve existing research programs related to breast cancer & the environment. The agenda will be posted on the web: *http://www.niehs.nih.gov/about/orgstructure/boards/ibcercc/.*

Place: Nat. Inst. of Environmental Health Sciences, Building 101, Rodbell Auditorium, 111 T. W. Alexander Drive, Research Triangle Park, NC 27709.

Contact Person: Gwen W. Collman, PhD, Director, Division of Extramural Research and Training (DERT), Nat. Inst. of Environmental Health Sciences, National Institutes of Health, 615 Davis Dr., KEY615/3112, Research Triangle Park, NC 27709, (919) 541–4980, *collman@niehs.nih.gov.*

Any member of the public interested in presenting oral comments to the Committee should submit their remarks in writing at least 10 days in advance of the meeting. Comments in document format (*i.e.* WORD, Rich Text, PDF) may be submitted via e-mail to *ibcercc@niehs.nih.gov.* You do not need to attend the meeting in order to submit comments.

Interested individuals and representatives of organizations may submit a letter of intent, a brief description of the organization represented, and a short description of the oral comments you wish to present. Only one representative per organization may be allowed to present oral comments and if