resolve the disagreement with reporting entity but was unsuccessful. The Department can only determine whether the report was legally required to be filed and whether the report accurately depicts the action taken and the reporter's basis for action. Additional detail on the process of dispute resolution and Secretarial Review process can be found at 45 CFR 60.14 of the NPDB regulations.

RECORD SOURCE CATEGORIES:

The records contained in the system are submitted by the following entities: (1) Insurance companies and others who have made payment as a result of a malpractice action or claim, (2) State Boards of Medical and Dental Examiners; (3) State Licensing Boards; (4) hospitals and other health care entities; (5) DEA; and (6) Federal entities which employ health practitioners or who have authority to sanction such practitioners covered by a Federal program. Section 1921 of the Social Security Act expands reporting of actions submitted by State health care practitioner licensing and certification authorities (including medical and dental boards), State entity licensing and certification authorities, peer review organizations and private accreditation organizations.

SYSTEMS EXEMPTED FROM CERTAIN PROVISIONS OF THE ACT:

The Secretary has exempted this system from certain provisions of the Act. In accordance with 5 U.S.C. 552(k)(2) and 45 CFR 5b.11(b)(ii)(L), this system is exempt from subsections 5 U.S.C. 552a(c)(3), (d)(1)–(4), (e)(4)(G) and (H), and (f).

[FR Doc. 2012–7612 Filed 3–29–12; 8:45 am]

BILLING CODE 4165–15–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.

MUC–1 Tumor Antigen Agonist Epitopes for Enhancing T-cell Responses to Human Tumors

Description of Technology: The MUC–1 tumor associated antigen has been shown to be overexpressed and/or underglycosylated in a wide range of human cancers. The C-terminus region of MUC–1 (MUC–1C) has been shown to be an oncogene and has been associated with a more aggressive phenotype in several different cancers.

Scientists at NIH have identified 7 new agonist epitopes of the MUC–1 tumor associated antigen. Compared to their native epitope counterparts, peptides reflecting these agonist epitopes have been shown to enhance the generation of human tumor cells, which in turn have a greater ability to kill human tumor cells endogenously expressing the native MUC–1 epitope. The agonist epitopes span both the VNTR region of MUC–1 and the C-terminus region. The epitopes encompass 2 major MHC alleles reflecting the majority of the population.

Along with the method of use, the technology encompasses the use of these agonist epitopes in peptide- and protein-based vaccines, with dendritic cells or other antigen presenting cells, or encoding sequences in DNA, viral, bacterial, yeast, or other types of vectors, or to stimulate T-cells *in vitro* for adoptive immunotherapy protocols.

Potential Commercial Applications:

- As a therapeutic vaccine to enhance patient's immune responses to a range of human cancers
- As a preventive vaccine for patients with preneoplastic conditions or a high risk of developing cancer
- As a preventive vaccine for cancers
- For *in vitro* stimulation of lymphocytes for adoptive transfer protocols for cancer

Competitive Advantages:

- The agonist epitopes have been shown to be much more potent than their natural counterparts in activating human T-cells to MUC–1.
- Compared to T-cells activated with the corresponding native epitopes, the T-cells activated by the agonist epitopes lyse tumor cells to a greater extent.
- The technology can be used in a wide range of cancer vaccine platforms and in adoptive immunotherapy protocols.
- The technology can be combined with existing vaccine platforms including those currently showing patient benefit, as well as with other therapeutic modalities.

Development Stage:

- Pre-clinical
- In vitro data available

Inventors: Jeffrey Schlom and Kwong-Yok Tsang (NCI).

Intellectual Property: HHS Reference No. E–001–2012/0—U.S. Patent Application No. 61/582,723 filed 03 Jan 2012.

Related Technologies:

- HHS Reference No. E–154–1998/0—PCT Application No. PCT/US98/03693
- HHS Reference No. E–321–2003/0—PCT Application No. PCT/US2004/41921

Licensing Contact: Sabarni Chatterjee, Ph.D., MBA; 301–435–5587; *chatterjeesa@mail.nih.gov.*

Collaborative Research Opportunity: The Laboratory of Tumor Immunology and Biology, National Cancer Institute, is seeking statements of capability or interest from parties interested in collaborative research to further develop, evaluate or commercialize the use of MUC–1 tumor antigen agonist epitopes for the treatment or prevention of cancer. For collaboration opportunities, please contact John Hewes, Ph.D. at *hewesj@mail.nih.gov.*

Novel Diagnostic, Prognostic and Therapeutic Biomarker for Hepatocellular Carcinoma

Description of Technology: Scientists at the National Cancer Institute have discovered that Stearol-CoA desaturase-1 (SCD–1) is associated with hepatocellular carcinoma (HCC). Utilizing a microarray to analyze HCC patient samples, the investigators found SCD–1 is elevated in liver tumor tissues and it is a marker for a highly aggressive form of HCC, hepatic stem cell-like HCC subtype (HpSC HCC), which retains stem-cell features capable of cellular plasticity and cell motility. The investigators found SCD–1 is significantly elevated in HpSC tumors in comparison to less aggressive HCC tumors and it is associated with poor patient survival. *In vitro* studies demonstrate SCD–1 inhibition and/or addition of saturated palmitic acid reduces HpSC HCC characteristics. In addition to diagnostic, prognostic, and treatment applications, this technology

may enable clinicians to effectively stratify patients for more aggressive cancer treatment and prioritize candidates for liver transplantation.

Potential Commercial Applications:

- Method to diagnose HCC
- Method to prognose patient survival
- Method to stratify HCC for appropriate treatment
- Method to treat HCC

Competitive Advantages:

- Retrospective studies performed on human samples
- Modulation of SCD–1 reduces HpSC HCC characteristics

Development Stage:

- Early-stage
- In vitro data available
- In vivo data available (human)

Inventors: Anuradha Budhu and Xin W. Wang (NCI).

Intellectual Property: HHS Reference No. E–205–2011/0—U.S. Provisional Application No. 61/533,392 filed 12 Sep 2011.

Related Technology: HHS Reference No. E–139–2010/0—PCT Application No. PCT/US2011/032285 filed 13 Apr 2011.

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

Collaborative Research Opportunity: The National Cancer Institute is seeking statements of capability or interest from parties interested in collaborative research to further develop, evaluate or commercialize biomarkers for liver cancer. For collaboration opportunities, please contact John Hewes, Ph.D. at *hewesj@mail.nih.gov.*

Potential Use of Anti-IgE in the Treatment of Lupus Nephritis

Description of Technology: Systemic lupus erythematosus (SLE) is a multi-organ inflammatory disease characterized by a significant morbidity and mortality related to both disease evolution as well as therapeutic side effects. At least half of SLE patients develop lupus nephritis.

The inventors have used a *Lyn* -/- mouse model that develops an autoimmune disease exhibiting some features of human SLE. Using this model the inventors identified basophils and self-reactive IgEs as important components in the development of autoantibody-mediated kidney disease. The inventors found that depletion of basophils or the absence of IgE causes a considerable reduction in autoantibody production and preserves kidney function in the *Lyn* -/- mice. The inventors' work demonstrates that IgE immune complexes can activate basophils and that removal of self-reactive IgEs that form functional circulating immune complexes prevents kidney disease. Further, the inventors have shown that basophils are contributors to the production of the self-reactive antibodies that cause lupus-like nephritis in the *Lyn* -/- mice. Accordingly, reducing circulating IgE levels or reducing basophil activation may be of therapeutic benefit.

Potential Commercial Applications: Further research and development of therapeutic approach to treat lupus nephritis.

Competitive Advantages: Current treatment of lupus has not advanced for many years. This finding is of importance for its potential in advancing treatment of the disease.

Development Stage:

- Early-stage
- Pre-clinical

Inventors: Juan Rivera and Nicolas Charles (NIAMS).

Publications:

1. Charles N, *et al.* Basophils and the T helper 2 environment can promote the development of lupus nephritis. Nat Med. 2010 Jun;16(6):701–707. [PMID 20512127].

2. Brightbill HD, *et al.* Antibodies specific for a segment of human membrane IgE deplete IgE-producing B cells in humanized mice. J Clin Invest. 2010 Jun;120(6):2218–2229. [PMID 20458139].

3. Mack M, *et al.* Basophils and mast cells in renal injury. Kidney Int. 2009 Dec;76(11):1142–1147. [PMID 19692999].

4. Busse W, *et al.* Omalizumab, anti-IgE recombinant humanized monoclonal antibody for the treatment of severe allergic asthma. J Allergy Clin Immunol. 2001 Aug;108(2):184–90. [PMID: 11496232].

Intellectual Property: HHS Reference No. E–216–2010/0—PCT Application No. PCT/US2010/058077 filed 24 Nov 2010.

Licensing Contact: Jaime M. Greene; 301–435–5559; *greenejaime@mail.nih.gov.*

Collaborative Research Opportunity: The National Institute of Arthritis and Musculoskeletal and Skin Diseases is seeking statements of capability or interest from parties interested in collaborative research to further develop, evaluate or commercialize the technology for the use of anti-IgE in the treatment of Lupus Nephritis. For collaboration opportunities, please contact Cecilia Pazman at *pazmance@mail.nih.gov.*

Dated: March 27, 2012.

Richard U. Rodriguez,

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

[FR Doc. 2012–7709 Filed 3–29–12; 8:45 am]

BILLING CODE 4140–01–P

DEPARTMENT OF HOMELAND SECURITY

[Docket No. DHS–2012–0012]

National Infrastructure Advisory Council

AGENCY: National Protection and Programs Directorate, DHS.

ACTION: Committee Management; Notice of an open Federal Advisory Committee meeting.

SUMMARY: The National Infrastructure Advisory Council (NIAC) will meet on Tuesday, April 17, 2012, 1310 N. Courthouse Road, Suite 300, Virginia Room, Arlington, VA 22201. The meeting will be open to the public.

DATES: The NIAC will meet Tuesday, April 17, 2012, from 1:30 p.m. to 4:30 p.m. The meeting may close early if the committee has completed its business. For additional information, please consult the NIAC Web site, *www.dhs.gov/NIAC,* or contact the NIAC Secretariat by phone at (703) 235–2888 or by email at *NIAC@dhs.gov.*

ADDRESSES: 1310 N. Courthouse Road, Suite 300, Virginia Room, Arlington, VA 22201.

While this meeting is open to the public, participation in the NIAC deliberations is limited to committee members and appropriate Federal Government officials. Discussions may include committee members, appropriate Federal Government officials, and other invited persons attending the meeting to provide information that may be of interest to the Council.

Immediately following the committee member deliberation and discussion period, there will be a limited time period for public comment. Comments should be limited to meeting agenda items and previous NIAC studies. All previous NIAC studies can be located at *www.dhs.gov/NIAC.* Relevant public comments may be submitted in writing or presented in person for the Council to consider. Comments should be limited to the issues and topics addressed by the Council. In-person presentations will be limited to three minutes per speaker, with no more than 30 minutes for all speakers. Parties interested in making in-person