

allow it to be utilized to treat a broader population of patients.

- **Versatile antigen recognition**—These TCRs are CD8 and CD4 independent meaning that cells expressing these TCRs are capable of eliciting an immune response in the absence of CD8 or CD4 molecule expression on the T cell. When utilized for immunotherapy, this versatility allows engineered T cells expressing this TCR to recognize and eliminate tumors expressing SSX-2 regardless of how the antigen is presented to the T cell.

**Development Status:** This technology is in a preclinical stage of development.

**Inventors:** Richard A. Morgan *et al.* (NCI).

#### Publications

1. N Chinnasamy, *et al.* Development of HLA-A2 Restricted TCR Against Cancer Testis Antigen SSX-2 for Adoptive Immunotherapy of Cancer. Abstracts for the 25th Annual Meeting of the International Society for Biological Therapy of Cancer, J Immunother. 2010 Oct;33(8):860, DOI 10.1097/CJI.0b013e3181f1e08d.

2. D Valmori, *et al.* Expression of synovial sarcoma X (SSX) antigens in epithelial ovarian cancer and identification of SSX-4 epitopes recognized by CD4+ T cells. Clin Cancer Res. 2006 Jan 15;12(2):398–404. [PubMed: 16428478]

3. G Bricard, *et al.* Naturally acquired MAGE-A10- and SSX-2-specific CD8+ T cell responses in patients with hepatocellular carcinoma. J Immunol. 2005 Feb 1;174(3):1709–1716. [PubMed: 15661935]

**Patent Status:** U.S. Provisional Application No. 61/384,931 filed 21 Sept 2010 (HHS Reference No. E-269–2010/0–US-01).

**Related Technologies:** T cell receptor technologies developed against other CTAs: E-304–2006/0 and E-312–2007/1 (anti-NY-ESO-1) and E-236–2010/0 (anti-MAGE-A3).

**Licensing Status:** Available for licensing.

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

**Collaborative Research Opportunity:** The National Cancer Institute, Surgery Branch, is seeking statements of capability or interest from parties interested in collaborative research to further develop, evaluate, or commercialize the use of T cell receptor gene therapy for the treatment of cancer. Please contact John Hewes, Ph.D. at 301–435–3121 or hewesj@mail.nih.gov for more information.

Dated: November 24, 2010.

**Richard U. Rodriguez,**

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

[FR Doc. 2010–30279 Filed 12–1–10; 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.

#### Mouse Monoclonal Antibody for CEACAM

**Abstract:** The following biological material is a hybridoma cell line generated from mice lymphocytes immunized with human mammary carcinomas and fused to a myeloma cell line. The resulting mouse monoclonal antibody (MAb, clone B1.1) is directed against carcinoembryonic antigen (CEA). CEA are glyco-proteins whose expression levels are increased on the surface of metastatic cancer cells. Therefore, antibodies generated from the hybridoma clone B1.1 can be used to detect cancer cells. MAb B1.1 binds to the surface of human breast and melanoma cell lines and cells associated with colon carcinomas and adenomas. The antibody has been tested to work effectively in several techniques such as Immunofluorescence, Western Blot, Fluorescent Activated Cell Sorting

(FACS), and Immunohistochemistry (IHC).

#### Commercial Applications

- Developing cancer biomarker.
- Developing cell sorting assays (*e.g.* FACS).
- Immunofluorescence, Western Blotting, and Immunohistochemistry for CEA.
- Developing prognostic assays for cancer.

**Competitive Advantages:** Tested to bind CEA and can be used in different Immunological Techniques such as Immunofluorescence, Western Blot, Fluorescent Activated Cell Sorting (FACS), and Immunohistochemistry (IHC).

**Materials Available:** 1 vial of Hybridoma cell line (B1.1).

**Inventors:** Jeffrey Schlom and David Colcher (NCI).

#### Related Publications

1. D. Colcher *et al.* (1983) [PubMed: 6365268].

2. D. Stramignoni *et al.* (1983) [PubMed: 6852972].

**Patent Status:** “The Generation of Monoclonal Antibody (MAb) B1.1 and Its Reactivity to Human Tumors,” HHS Reference No. E-272–2010/0—Research Material. Patent protection is not being pursued for this technology.

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

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

#### Novel Compounds That Specifically Kill Multi-Drug Resistant Cancer Cells

**Description of Technology:** One of the major hindrances to successful cancer chemotherapy is the development of multi-drug resistance (MDR) in cancer cells. MDR is frequently caused by the increased expression or activity of ABC transporter proteins in response to the toxic agents used in chemotherapy. The increased expression or activity of the ABC transporter proteins causes the toxic agents to be removed from cells before they can act to kill the cell. As a result, research has generally been directed to overcoming MDR by inhibiting the activity of ABC transporters, thus causing the chemotherapeutic agents to remain in the cell long enough to exert their effects. However, compounds that inhibit ABC transporter activity often elicit strong and undesirable side-effects due to the inhibition of ABC transporter function in normal cells, thereby restricting their usefulness as therapeutics.

Investigators at the NIH previously identified novel compounds with the ability to kill multi-drug resistant cancer cells while leaving normal cells relatively unharmed. These “MDR-selective compounds” were not inhibitors of ABC transporters because they killed multi-drug resistant cells without affecting the activity of ABC transporters. Furthermore, their activity was dependent directly on the level of expression of ABC transporters, thus increasing their selectivity for diseased cells. As a result, the undesirable side-effects that have prevented the use of inhibitors of ABC transporters as therapeutics should not affect the therapeutic application of the MDR-selective compounds.

The inventors have now generated third generation MDR-selective compounds with further improved solubility, selectivity and killing activity toward MDR cells. The new MDR-selective compounds selectively kill MDR cancer cells, and their efficacy correlates directly with the level of ABC transporter expression. This suggests that the third generation MDR-selective compounds represent a powerful strategy for treating MDR cancers.

#### *Applications:*

- Treatment of cancers associated with MDR, either alone or in combination with other therapeutics.
- Development of a pharmacophore for improved MDR-selective compounds.

#### *Advantages:*

- MDR-selective compounds capitalize on one of the most common drawbacks to cancer therapies (MDR) by using it as an advantage for treating cancer.
- The compositions do not inhibit the activity of ABC transporters, thereby reducing the chance of undesired side-effects during treatment.
- The effects of MDR-selective compounds correlate with the level of ABC transporter expression, allowing healthy cells to better survive treatments.
- Increased specificity and solubility of the new MDR-inverse compounds allows greater access to MDR cells, thereby increasing therapeutic effectiveness.

*Development Status:* Preclinical stage of development, *in vitro* data.

*Inventors:* Hall (NCI) *et al.*

*U.S. Patent Status:* U.S. Provisional Application 61/375,672 (E-249-2010/0-US-01).

*For more information, see:*

- Hall, MD *et al.* (2009) “Synthesis, activity, and pharmacophore development for isatin-beta-thiosemicarbazones with selective

activity toward multidrug-resistant cells” *J Med Chem.* 52(10):3191–204.

- PCT Publication WO 2009/102433 (PCT Patent Application PCT/US2009/000861).

*Licensing Status:* Available for licensing.

*Licensing Contact:* David A. Lambertson, Ph.D.; 301-435-4632; [lambertsond@mail.nih.gov](mailto:lambertsond@mail.nih.gov).

*Collaborative Research Opportunity:* The Center for Cancer Research, Laboratory of Cell 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 Hewes, Ph.D. at 301-435-3121 or [hewesj@mail.nih.gov](mailto:hewesj@mail.nih.gov) for more information.

### **Isocitrate Dehydrogenase 1 (IDH1) R132 Mutation Human Melanoma Metastasis Cell Line**

*Description of Technology:* Isocitrate dehydrogenase 1 (IDH1) plays an important role in glucose metabolism in the cytoplasm, converting isocitrate to  $\alpha$ -ketoglutarate while reducing nicotinamide adenine dinucleotide phosphate (NADP+ to NADPH). However, when IDH1 harbors a R132 mutation it results in the accumulation of 2-hydroxyglutarate and has a corresponding association with cancer. This mutation in IDH1 has previously been identified in approximately 80% of progressive gliomas and 10% acute myeloid leukemias (AML). In contrast, this mutation is very rare in other cancers. Therefore, additional research on the IDH1 R132 mutation could be useful for diagnostic, prognostic, and therapeutic purposes.

The researchers at the NIH have developed a human melanoma cell line designated 2633, which harbors the IDH1 R132C mutation. The inventors used low passage cell lines derived from a panel of confirmed metastatic melanoma tumor resections, paired with apheresis-collected peripheral blood mononuclear cells to identify IDH1 mutations. Sequencing of IDH1 in this panel allowed them to discover a melanoma cell line with the IDH1 R132C mutation. Until now no such cell line has been found and this has hindered the understanding of the effects mutated IDH1 has on cancer progression as well as the development of drugs that would be specific for cells that harbor this mutation. Use of this cell line will allow researchers to decipher the biology of this gene as well as aid in the development of specific inhibitors of its mutated form.

*Applications:*

- *In vitro* and *in vivo* cell model for the IDH1 R132C mutation in melanoma. This would be an extremely useful research tool for investigating the underlying biology of IDH1 phenotypes, including effects on growth, motility, invasion, and metabolite production.

- Research tool for testing the activity of inhibitors to IDH1, where such inhibitors could be used as a therapeutic drug to treat particular cancers including potentially glioma, AML and melanoma.

- Research tool to generate cell lines where the R132C mutation is knocked out or the wild type gene is knocked in using an adeno-associated virus. These resulting cells can be used to understand the underlying biology of IDH1 phenotypes or to identify candidate small molecule and other therapeutic drugs.

#### *Advantages:*

- *Cell line is derived from a melanoma patient:* This cell line likely retains many features of primary melanoma samples. For example novel melanoma antigens identified from this cell would be expected to correlate with antigens expressed on human melanoma tumors. Studies performed using this cell line could be used to elucidate to the biological basis of the initiation and progression of melanoma in humans as well as aid in the identification and/or testing of IDH1 R132-targeted inhibitors.

- *Expresses the R132 IDH1 mutation in melanoma:* IDH1 R132 mutations frequently occur in advanced gliomas, however this is the first identification of an IDH1 mutation in melanoma. Therefore, the 2633 cell line represents a tool that can be utilized to study the impact of this IDH1 gene and the R132C mutation on melanoma and other cancers.

*Inventors:* Yardena Samuels (NHGRI) and Steven Rosenberg (NCI).

*Publication:* Lopez GY, Reitman ZJ, Solomon D, Waldman T, Bigner DD, McLendon RE, Rosenberg SA, Samuels Y, Yan H. IDH1(R132) mutation identified in one human melanoma metastasis, but not correlated with metastases to the brain. *Biochem Biophys Res Commun.* 2010 Jul 30;398(3):585–587. [PubMed: 20603105]

*Patent Status:* HHS Reference No. E-232-2010/0—Research Tool. Patent protection is not being pursued for this technology.

*Licensing Status:* Available for licensing.

*Licensing Contact:* Whitney Hastings; 301-451-7337; [hastingsw@mail.nih.gov](mailto:hastingsw@mail.nih.gov).

*Collaborative Research Opportunity:* The National Human Genome Research Institute’s Cancer Genetics Branch is seeking statements of capability or

interest from parties interested in collaborative research to further develop, evaluate and/or commercialize this newly identified melanoma-associated gene as a diagnostic marker as well as utilize the IDH1 R132 cell line to identify and test IDH1 inhibitors as possible therapeutic drug candidates to treat melanoma and other cancers. Please contact Dr. Yardena Samuels at [samuelsy@mail.nih.gov](mailto:samuelsy@mail.nih.gov) for more information.

**ERBB4 Mutations Mutation Identified in Human Melanoma Metastasis Cell Lines (2690, 2379, 2197, 2183, 2535, 2645, 1770, 2359, 2238, 2319, 2190)**

**Description of Technology:** Protein tyrosine kinases (PTKs) have been associated with a wide variety of cancers, including melanoma. Using high-throughput gene sequencing, the NIH has analyzed PTKs in melanoma and identified several novel somatic alterations, including alterations in ERBB4 (also called HER4). These mutations were found to increase the sensitivity of cells in which they reside to small molecule inhibitors, such as lapatinib.

Available for licensing are several melanoma cell lines that harbor ERBB4 mutations. These cell lines provide methods of identifying specific inhibitors to ERBB4 that could be used to treat patients with ERBB4 mutations as well as methods to further understand the role of ERBB4 mutations in melanoma. Given the recent success of small molecule protein kinase inhibitors and specifically inhibitors to epidermal growth factor receptor (EGFR) (such as gefitinib and erlotinib), these reagents could be used to further the development of specific inhibitors to ERBB4 and improve existing melanoma treatments for patients with these mutations.

**Applications:**

- *In vitro* and *in vivo* cell model for understanding the biology of ERBB4, including growth, motility, invasion, and metabolite production.
- High throughput drug screening to test for ERBB4 inhibitors that could be used to treat particular cancers, such as melanoma.
- Diagnostic array for the detection of ERBB4 mutations.
- Research tool to generate cell lines where the ERBB4 mutation is knocked out or the wild type gene is knocked in using an adeno-associated virus. These resulting cells can be used to understand the underlying biology of ERBB4 phenotypes or to identify candidate small molecule and other therapeutic drugs.

**Advantages:**

- *Cell lines are derived from melanoma patients:* These cell lines are likely to retain many features of primary melanoma samples. For example novel melanoma antigens identified from this cell line would be expected to correlate with antigens expressed on human melanoma tumors. Studies performed using these cell lines could be used to elucidate the biological basis of initiation and progression of melanoma in humans as well as aid in the identification and/or testing of ERBB4 inhibitors.

• *Expresses the ERBB4 mutation in melanoma:* ERBB4 is a highly mutated gene in melanoma, suggesting its important functional role in the disease. Therefore, these cell lines represent a tool that can be utilized to study the impact of the ERBB4 gene and the associated mutations on melanoma, and possible other cancers since mutations in ERBB family members such as EGRF and ERBB2 are prevalent in lung cancer, glioblastoma and gastric cancer.

**Inventors:** Yardena Samuels (NHGRI), Steven Rosenberg (NCI), and Todd Prickett (NHGRI).

**Publication:** Prickett TD, Agrawal NS, Wei X, Yates KE, Lin JC, Wunderlich JR, Cronin JC, Cruz P, Rosenberg SA, Samuels Y. Analysis of the tyrosine kinome in melanoma reveals recurrent mutations in ERBB4. *Nature Genet.* 2009 October; 41(10):1127–1132. [PubMed: 19718025]

**Patent Status:** HHS Reference No. E–229–2010/0—Research Tool. Patent protection is not being pursued for this technology.

**Licensing Status:** Available for licensing.

**Licensing Contact:** Whitney Hastings; 301–451–7337; [hastingw@mail.nih.gov](mailto:hastingw@mail.nih.gov).

**Collaborative Research Opportunity:** The National Human Genome Research Institute's Cancer Genetics Branch is seeking statements of capability or interest from parties interested in collaborative research to further develop, evaluate and/or commercialize these newly characterized ERBB4 mutant cell lines as well as to identify and test ERBB4 inhibitors as possible therapeutic drug candidates to treat melanoma and other cancers. Please contact Dr. Yardena Samuels at [samuelsy@mail.nih.gov](mailto:samuelsy@mail.nih.gov) for more information.

**Synthetic Analogs of RGD and NGR Cyclic Peptides**

**Description of Technology:** Cell surface biomolecules such as integrins ( $\alpha_v\beta_3$ ,  $\alpha_v\beta_5$ ,  $\alpha_v\beta_8$ ,  $\alpha_6\beta_4$ ), folate receptors, and CD13 are highly expressed in cancer cells and are involved in angiogenesis, invasion and

metastasis. Consequently, this has made these cellular biomolecules attractive targets for delivery of drugs that can bind to them selectively. The peptide motifs RGD (Arg-Gly-Asp) and NGR (Asn-Gly-Arg), in particular, are recognized by integrins  $\alpha_v\beta_3$  and  $\alpha_v\beta_5$  and CD13 with high affinity. Further, short peptide sequences of RGD and NGR are commercially useful because they are amenable to large scale synthesis, chemical modification and are non-immunogenic. Therefore, there is a need for cyclic compounds having the NGR peptide motif to target CD13 or having the RGD peptide motif to target  $\alpha_v\beta_3$  and  $\alpha_v\beta_5$  integrins.

Accordingly, the researchers at the NIH have developed cyclic NGR and RGD pentapeptide analogs efficiently synthesized on resin via click chemistry. These cyclic peptides are potentially useful in targeted delivery of drugs, antibodies, or nanoparticles to the site of angiogenic blood vessels and tumors. By allowing for targeted drug delivery, these peptides can minimize general cytotoxicity and improve bioavailability. The cyclic peptides described are novel, synthetic analogs of RGD and NGR cyclic peptides. Therefore, their inherent cyclic structure and the cyclization strategy will make these compounds stable from hydrolytic degradation, thereby prolonging their half life in circulation.

**Applications:** Targeted drug delivery and medical imaging of cancer tissues expressing CD13 or  $\alpha_v\beta_3$  and  $\alpha_v\beta_5$  integrins.

**Advantages:**

- These cyclic peptides contain a triazole unit that would be less likely to be attacked by hydrolytic enzymes and esterases, thus making them ideal candidates for *in vivo* targeted delivery and imaging.

- The RGD and NGR cyclic peptides are amenable to large scale synthesis, chemical modification and are non-immunogenic, while the linear RGD peptide counterparts are prone to protease degradation making them much less stable and limiting their use in *in vivo* applications.

- Both linear and disulfide-bridged cyclic peptides containing the NGR motif have been used to deliver various anti-tumor compounds and viral particles to tumor vessels, with the cyclic versions showing more than a 10 fold higher binding affinity than their linear counterparts.

**Development Status:** Pre-clinical proof of principle.

**Inventors:** Belhu B. Metaferia and Javed Khan (NCI).

**Publications:** B Metaferia *et al.* Synthesis of novel cyclic NGR/RGD

peptide analogs via on resin click chemistry. In preparation.

**Patent Status:** U.S. Provisional Application No. 61/347,038 filed 21 May 2010 (HHS Reference No. E-130-2010/0-US-01).

**Licensing Status:** Available for licensing.

**Licensing Contact:** Whitney Hastings; 301-451-7337; [hastingsw@mail.nih.gov](mailto:hastingsw@mail.nih.gov).

### Novel Therapeutic Compounds for Treatment of Cancer and Immune Disorders

**Description of Invention:** The global market for cancer therapeutics is over \$40 billion and is anticipated to continue to rise in the future. There remains a significant unmet need for therapeutics for cancers that affect blood, bone marrow, and lymph nodes and the immune system, such as leukemia, multiple myeloma, and lymphoma. The proteasome inhibitor bortezomib, which may prevent degradation of pro-apoptotic factors permitting activation of programmed cell death in neoplastic cells dependent upon suppression of pro-apoptotic pathways, has been a successful mode of treatment for such cancers. However, some patient's cancers have been found to be resistant to the drug.

Researchers at the National Institutes of Health have developed novel hydrazone and diacyl hydrazine compounds that are inhibitors of the endoplasmic reticulum-associated protein degradation (ERAD) pathway. These compounds preferentially target the proteasome assistant ATPase p97/VCP at a site independent of nucleotide binding. The researchers have shown that these ERAD inhibitors can induce cancer cell death and can also synergize with bortezomib in cytotoxic activity. In addition to treating diseases or disorders in which inhibition of the ERAD pathway is an effective therapy, these novel compounds may also be useful in the study of protein degradation.

#### Advantages:

- Development of therapies against tumors that are resistant to bortezomib.
- Use in therapies in combination with proteasome inhibitors.
- Development of immunosuppressive therapies that target the ubiquitin proteasome system.
- Studies of the mechanism of protein degradation and other biological processes that involve the p97 ATPase.
- Bioprobes to detect endoplasmic reticulum (ER) structures in live cells.

#### Advantages:

- Potent anti-tumor activity.
- Simpler chemical structure makes synthesis easier and more cost-effective than previous ERAD inhibitors.

- Retain activity against bortezomib-resistant cells and can synergize with bortezomib.

- Fluorescent.

- High affinity for the ER.

**Development Status:** Pre-clinical.

**Inventors:** Adrian Wiestner (NHLBI), William Trenkle (NIDDK), Yihong Ye (NIDDK) *et al.*

**Relevant Publications:**

1. Qiuyan Wang *et al.* ERAD inhibitors integrate ER stress with an epigenetic mechanism to activate BH3-only protein NOXA in cancer cells. *Proc Natl Acad Sci USA* 2009 Feb 17;106(7):2200-2205. [PubMed: 19164757]
2. Qiuyan Wang *et al.* The ERAD inhibitor Eeyarestatin I is a bifunctional compound with a membrane-binding domain and a p97/VCP inhibitory group. *PloS ONE* 2010, in press.

**Patent Status:** U.S. Provisional Application No. 61/266,760 filed 04 Dec 2009 (HHS Reference No. E-291-2009/0-US-01).

**Licensing Status:** Available for licensing.

**Licensing Contact:** Surekha Vathyam, Ph.D.; 301-435-4076; [vathyams@mail.nih.gov](mailto:vathyams@mail.nih.gov).

### Targeted Anti-Cancer Compounds for Treating Chromosomal Instability Syndromes

**Description of Invention:** At \$47 billion, cancer is one of the largest, fastest growing markets in the pharmaceutical industry. There remains a significant unmet need for new therapeutics that target cancer cells while sparing normal cells. Cancer cells show higher levels of DNA damage than normal cells, and therefore rely more heavily than normal cells on DNA repair mechanisms for survival. There is a particular need for cancer therapies for cancer-prone chromosomal instability syndromes such as Ataxia Telangiectasia, Nijmegen Breakage, Bloom, and Fanconi's anemia, which result from dysfunctional DNA repair systems.

Researchers at Columbia University and the National Cancer Institute (NCI) have developed compositions and methods of use in the treatment of cancer and in the sensitization of cancer cells to cancer therapy. The compositions target the MRE11-RAD50-NBS1 (MRN) complex, a DNA repair complex essential for sensing and responding to DNA damage.

Given the dependency of cancer cells on DNA repair systems, they are susceptible to compositions that inhibit DNA damage repair. Thus, cancers that

already have one or more defects in DNA repair systems, such as those from patients with chromosomal instability syndromes, are effectively treated with the present compositions.

**Applications:** Development of treatments for cancer.

**Development Status:** Pre-clinical.

**Inventors:** Levy Kopelovich (NCI) *et al.*

**Relevant Publication:** A Dupré *et al.* A forward chemical genetic screen reveals an inhibitor of the Mre11-Rad50-Nbs1 complex. *Nat Chem Biol*. 2008;4(2):119-125. [PubMed: 18176557]

#### Patent Status:

- U.S. Provisional Application No. 61/203,377 filed 22 Dec 2008 (HHS Reference No. E-154-2009/0-US-01).
- International Application No. PCT/US09/69171 filed 22 Dec 2009, which published as WO 2010/075372 on 01 Jul 2010 (HHS Reference No. E-154-2009/0-PCT-02).

**Licensing Status:** Available for licensing.

**Licensing Contact:** Patrick P. McCue, Ph.D.; 301-435-5560; [mccuepat@mail.nih.gov](mailto:mccuepat@mail.nih.gov).

**Collaborative Research Opportunity:** The National Cancer Institute, Division of Cancer Prevention, Chemopreventive Agent Development Research Group, is seeking statements of capability or interest from parties interested in collaborative research to further develop, evaluate, or commercialize agents for the prevention and treatment of cancer. Please contact John Hewes, Ph.D. at 301-435-3121 or [hewesj@mail.nih.gov](mailto:hewesj@mail.nih.gov) for more information.

Dated: November 24, 2010.

**Richard U. Rodriguez,**

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

[FR Doc. 2010-30278 Filed 12-1-10; 8:45 am]

**BILLING CODE 4140-01-P**

## DEPARTMENT OF HOMELAND SECURITY

### U.S. Citizenship and Immigration Services

#### Agency Information Collection Activities: Form I-914, Extension of a Currently Approved Information Collection; Comment Request

**ACTION:** 30-Day Notice of Information Collection under Review: Form I-914 and Supplements A and B, Application for T Nonimmigrant Status; Application for Immediate Family Member of T-1 Recipient; and Declaration of Law Enforcement Officer for Victim of