Dated: May 27, 2009.

**Richard U. Rodriguez,**

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

[FR Doc. E9–12873 Filed 6–2–09; 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.

**Diagnostic Markers for Melanoma**

*Description of Technology:* This invention relates to diagnostic and prognostic markers for melanoma. It discloses the identification of somatic mutations in genes of the microphthalmia-associated transcription factor (MITF) pathway in patients with melanoma.

Melanoma is an aggressive and often fatal cancer with increasing incidence worldwide. Previous studies have linked the MITF pathway to the progression of melanoma. However, little is known about somatic mutations in genes of the MITF pathway that contribute to the development and progression of melanoma. To assess the role of the MITF pathway in melanoma, NIH investigators evaluated primary and metastatic melanoma samples for the presence of somatic mutations in two genes of the MITF pathway, MITF and SRY (sex determining region Y)—box 10 (SOX10). They identified 16 previously unidentified somatic mutations in these genes. These studies suggest that MITF and SOX10 genes be used as diagnostic markers in human metastatic melanoma.

*Applications*

• Diagnosis and prognosis of patients with melanoma by detecting any mutations in the MITF or SOX10 gene.

• Selection of therapy for melanoma patient; an MITF inhibitor can be selected for therapy if the patient has any of the disclosed mutations in MITF.

*Market:* Cancer is the second leading cause of death in the U.S. There is an acute need for cancer biomarkers that can be detected from clinically relevant samples and used for early diagnosis, therapeutic follow-up and prognosis of malignant diseases. Melanoma is the most serious type of cancer of the skin. The percentage of people who develop melanoma has more than doubled in the past 30 years. There are 68,720 estimated new cases and 8,650 estimated deaths from melanoma in the United States in 2009, according to the National Cancer Institute.

*Inventors:* Yardena R. Samuels *et al.* (NHGRI).

*Publication:* Cronin JC WJ, Loftus SK, Prickett TD, Wei X, Ridd, Vemula S, Burrell AS, Agrawal NS, Lin JC, Banister CE, Buckhaults P, Rosenberg SA, Bastian BC, Pavan WJ, Samuels Y: Frequent mutations in the MITF pathway in melanoma. Pigment Cell and Melanoma Research 2009, (In Press).

*Patent Status:* U.S. Provisional Application No. 61/214,415 filed 22 Apr 2009 (HHS Reference No. E–053–2009/0–US–01).

*Licensing Status:* Available for licensing.

*Licensing Contact:* Betty B. Tong, Ph.D; 301–594–6565; *tongb@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, or commercialize these newly identified candidate melanoma diagnostic and prognostic markers. Please contact NHGRI's Technology Development Coordinator (TDC) Claire T. Driscoll at *cdriscol@mail.nih.gov* for more information.

**T Cells Attacking Cancer: T Cell Receptors That Recognize the Tyrosinase Tumor Antigen**

*Description of Technology:* A problem with current chemotherapy-based cancer treatments is the harsh side-effects associated with many cancer drugs. Thus, there is an urgent need to develop new therapeutic strategies combining fewer side-effects and more specific anti-tumor activity. Adoptive cell transfer (ACT) is a promising new immunotherapeutic approach to treat cancer and other diseases by directing an individual's innate and adaptive immune system to recognize specific disease-associated antigens.

T cell receptors (TCRs) are proteins that recognize antigens in the context of infected or transformed cells and activate T cells to mediate an immune response and destroy abnormal cells. TCRs consist of two domains, one variable domain that recognizes the antigen and one constant region that helps the TCR anchor to the membrane and transmit recognition signals by interacting with other proteins.

Scientists at the National Institutes of Health (NIH) have isolated T cells that recognize the human tyrosinase tumor-associated antigen (TAA) from the tumor infiltrating lymphocytes (TIL) of a melanoma cancer patient. The human tyrosinase antigen is a tumor antigen expressed in a variety of cancers, including skin cancer (melanoma) and brain cancer (glioblastoma). Utilizing the tyrosinase specific T cells, these scientists developed human/mouse hybrid TCRs with enhanced affinity for the tyrosinase TAA. The TCR sequences were modified by making specific amino acid substitutions and replacing certain TCR regions with mouse homologues. These TCRs also showed CD8-independency and, thus, can be expressed in both CD8 and CD4 T cells. T cells expressing these engineered TCRs recognize skin and brain tumor cells in culture. These T cells also exhibit enhanced cytokine induction and better tumor reactivity compared to unmodified TCRs. Previous versions of gene-modified T cells developed by NIH researchers demonstrated objective clinical responses in some cancer patients, which have validated gene-modified T cell immunotherapy as a promising cancer treatment strategy. TCRs directed against the tyrosinase TAA could serve as valuable new immunotherapeutic tools for attacking tumors, especially in patients whose tumors do not express other common TAAs.

*Applications*

• Immunotherapeutics to treat and/or prevent the recurrence of a variety of human cancers, including melanomas and glioblastomas, that express tyrosinase by transferring T cells engineered with tyrosinase-specific TCRs into cancer patients.

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

• Immunotherapeutic to treat and/or prevent tumors that do not express other common tumor-associated antigens, such as MART–1, gp100, and NY–ESO–1.

*Advantages*

• The parent tyrosinase-specific TCR was isolated from tumor infiltrating lymphocytes, so the genetically-modified versions should have an elevated affinity for tyrosinase.

• The tyrosinase-specific T cells recognize skin and brain cancer cells in culture. These T cells are predicted to have broad anti-cancer activity once developed to a clinical level.

• CD8 independency: The tyrosinase-specific TCRs can be expressed in both CD8 and CD4 T cells to maximize the cell-mediated immune response to the tumor.

• The tyrosinase-specific T cells should not be rejected by a patient's immune system since the mouse tyrosinase-recognition enhancing TCR sequences are incorporated into a human TCR backbone.

*Market:* Cancer continues to be a medical and financial burden on U.S. public health. According to U.S. estimates, 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. Despite our increasing knowledge of oncology and cancer treatment methods, 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).

*Development Status:* This technology is in the pre-clinical stage of development. The inventors plan to develop the technology into clinical grade reagent for a clinical trial if the pre-clinical data continues to show promising results.

*Patent Status:* U.S. Provisional Application No. 61/147,846 filed 28 Jan 2009 (HHS Reference No. E–043–2009/0–US–01).

*Licensing Status:* Available for licensing.

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

*Collaborative Research Opportunity:* The National Cancer Institute, Surgery Branch, Tumor Immunology Section, is seeking statements of capability or interest from parties interested in collaborative research to further develop, evaluate, or commercialize T Cells Attacking Cancer: T Cell Receptors that Recognize the Tyrosinase Tumor Antigen. Please contact John D. Hewes, PhD at 301–435–3121 or *hewesj@mail.nih.gov* for more information.

**Genomics-Based Diagnostic Assay for Cancer**

*Description of Technology:* Molecular profiling with high throughput assays has gained utility in the management of select cancer patients and several gene expression-based assays are now marketed for improved prognostic accuracy for patients with cancer.

This technology describes a genomics based diagnostic assay for the diagnosis and prognosis of cancer patients. Using a mouse model of breast cancer, the inventors identified a gene expression signature that can predict the outcome for human breast cancer patients with as few as six genes. The gene signature includes a total of 79 cancer survival factor-associated genes and was validated using available genomic test sets that were based on previously conducted human clinical trials. More recently, the six-gene-model was validated for cancers other than breast using multiple, independent, publicly-available human lung cancer datasets. In addition to predicting the outcome of cancer patients, this technology could also be used to stratify patients for further therapy and treat patients by administering therapeutic agents that alter the activity of one of the aforementioned cancer survival factor-associated genes.

*Applications*

• Methods for cancer diagnosis and prognosis by evaluating expression levels of certain cancer survival factor-associated molecules in patients.

• Treatment of cancer by administering therapeutic agents that alter biological activity of cancer survival factor-associated molecule.

*Advantages:* Prognostic outcome of breast and lung cancer patients can be identified in as few as six genes.

*Development Status:* Pre-clinical stage of development.

*Inventors:* Steven K. Libutti and Mei He (NCI).

*Patent Status:* U.S. Provisional Application No. 61/152,597 filed 13 Feb 2009 (HHS Reference No. E–023–2009/0–US–01).

*Licensing Status:* Available for licensing.

*Licensing Contact:* Whitney A. Hastings; 301–451–7337; *hastingsw@mail.nih.gov.*

**New Weapons To Attack Cancer: T Cell Receptors Designed To Recognize Tumors With Enhanced Affinity**

*Description of Technology:* Given the unpleasant side-effects associated with many cancer drugs, there is an urgent need to develop new therapeutic strategies combining 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.

T cell receptors (TCRs) are proteins that recognize antigens in the context of infected or transformed cells and activate T cells to mediate an immune response and destroy abnormal cells. TCRs consist of two domains, one variable domain that recognizes the antigen and one constant region that helps the TCR anchor to the membrane and transmit recognition signals by interacting with other proteins.

Scientists at the National Institutes of Health (NIH) have developed T cells with an enhanced ability to recognize the tumor associated antigens (TAAs) NY–ESO–1 and MART–1. These T cells were engineered to increase their ability to recognize these TAAs by making small genetic modifications to the TCRs that recognize these TAAs. NY–ESO–1 is a cancer-testis antigen found in normal testis and various tumors. MART–1 is a melanoma antigen found on normal melanocytes and overexpressed in malignant melanomas. Previous versions of gene-modified T cells developed by these researchers to attack tumors demonstrated objective clinical responses in some cancer patients, which validated gene-modified T cell adoptive immunotherapy as a promising cancer treatment strategy. These latest versions of the NY–ESO–1 and MART–1 specific TCRs, designated 1G4 NY–ESO–1 and DMF5 MART–1, were rationally engineered to enhance anti-tumor activity. These TCRs cause T cells to exhibit enhanced cytokine production and increased lysis of tumor cells when stimulated with NY–ESO–1 or MART–1. Infusing these T cells into patients via adoptive immunotherapy could prove to be powerful new tools for attacking tumors.

*Applications*

• Immunotherapeutics to treat and/or prevent the recurrence of a variety of

human cancers that overexpress the NY–ESO–1 or MART–1 TAA, including melanoma, lung, breast, ovarian, prostate, thyroid, and bladder cancer, by adoptively transferring gene-modified T cells into patients.

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

*Advantages*

• NY–ESO–1 and MART–1 are overexpressed on a variety of cancers. Thus, this gene-modified TCR immunotherapy has wide applicability to treat a host of cancer types while reducing the side-effects of treatment.

• These latest engineered TCRs have improved affinity for their corresponding TAA compared to previously developed TCRs with modified sequences.

*Development Status:* These technologies are in clinical development. A clinical protocol (08–C–0121) is being conducted with the enhanced 1G4 NY–ESO–1 TCR.

*Market:* Cancer continues to be a medical and financial burden on U.S. public health. According to U.S. estimates, 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. Despite our increasing knowledge of oncology and cancer treatment methods, the fight against cancer will continue to benefit from the development of new therapeutics aimed at treating individual patients.

*Inventors:* Paul F. Robbins *et al.* (NCI).

*Publications*

1. PF Robbins *et al.* Single and dual amino acid substitutions in TCR CDRs can enhance antigen-specific T cell functions. J Immunol. 2008 May 1;180(9):6116–6131.

2. Y Zhao *et al.* High-affinity TCRs generated by phage display provide CD4+ T cells with the ability to recognize and kill tumor cell lines. J Immunol. 2007 Nov 1;179(9):5845–5854.

*Patent Status*

• U.S. Provisional Patent Application No. 60/974,872 filed 25 Sep 2007 (HHS Reference No. E–312–2007/0–US–01).

• PCT Patent Application No. PCT/US2008/77333 filed 23 Sep 2008 (HHS Reference No. E–312–2007/1–PCT–01).

*Licensing Status:* Available for licensing.

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

*Collaborative Research Opportunity:* The National Cancer Institute, Center for Cancer Research, Surgery Branch, is seeking statements of capability or interest from parties interested in collaborative research to further develop, evaluate, or commercialize TCRs that enhance the function of gene-modified T cells. Please contact John D. Hewes, PhD at 301–435–3121 or *hewesj@mail.nih.gov* for more information.

**Steroid Derivatives as Inhibitors of Human Tyrosyl-DNA Phosphodiesterase (Tdp1)**

*Description of Technology:* Tyrosyl-DNA phosphodiesterase (Tdp1) is an enzyme that repairs topoisomerase I (Top1)-mediated DNA damage induced by chemotherapeutic agents and ubiquitous DNA lesions that interfere with transcription. The current technology are steroid derivatives that human inhibit Tdp1.

Currently, there are various types of Top1 inhibitors used in chemotherapy, *e.g.,* camptothecin. However, Tdp1 inhibitors are expected to be effective in combination therapy with Top1 inhibitors for the treatment of cancers. Combining Tdp1 inhibitors with Top1 inhibitors would allow Tdp1 to potentiate the antiproliferative activity of Top1 inhibitors. In addition to Tdp1's effect on Top1, Tdp1 inhibitors can also exhibit antitumor activity independently, as tumors are shown to have excess free radicals, and Tdp1 repairs DNA damage by oxygen radicals.

*Applications:* It is anticipated that Tdp1 inhibitors in association with Top1 inhibitors can have selective activity toward tumor tissues. Tdp1 inhibitors may exhibit antitumor activity by themselves because tumors have excess free radicals.

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

*Inventors:* Yves Pommier *et al.* (NCI)

*Relevant Publication:* A manuscript directly related to the above technology will be available as soon as it is accepted for publication.

*Patent Status:* PCT Application No. PCT/US2008/004541 filed 5 Apr 2008, claiming priority to 5 Apr 2007 (HHS Reference No. E–130–2007/2–PCT–01).

*Licensing Status:* Available for licensing.

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

*Collaborative Research Opportunity:* The Center for Cancer Research, National Cancer Institute, Laboratory of Molecular Pharmacology, is seeking statements of capability or interest from parties interested in collaborative research to further develop, evaluate, or commercialize inhibitors of Tyrosyl-DNA phosphodiesterase (Tdp1). Please contact John D. Hewes, Ph.D. at 301–435–3121 or *hewesj@mail.nih.gov* for more information.

**Method for Spectroscopic Quantitation of HER2 in Biological Samples**

*Description of Technology:* An important clinical objective in certain cancer patients is the quantitation of HER2. The level of HER2 expression in some tumors correlates with disease stage and severity. For example, HER2 positive breast cancer is a more aggressive disease with a greater likelihood of recurrence, a poorer prognosis, and a decreased chance of survival compared with HER2-negative breast cancer.

This invention discloses a mass spectrometry method for quantitatively measuring HER2 in a variety of biological samples such as tissue, serum, or plasma. This invention is unlike traditional assays that use antibodies for detection of a HER2 and is superior to the current immunohistochemistry methods to stage tumor development. Consequently, a mass spectrometry-based clinical assay could be used to allow physicians to more effectively determine patient treatment. Furthermore, since this technology can also be used to assay formalin-fixed prostate tissue (FFPE) tissues, it could be a useful biomarker for pathology labs.

*Applications*

• Diagnostic assay for cancer that measures HER2 levels in clinical samples, such as tissues and biological fluids.

• Prognostic assay to determine the stage of cancer and the appropriate cancer treatment.

• Research tool that could be used to correlate HER2 expression with the expression of other proteins.

*Market*

• This novel *in vitro* diagnostic test for cancer has use in oncology and pathology laboratories of hospitals and commercial clinical laboratories.

• In the United States, almost 1.5 million new cancer cases are expected to be diagnosed in 2009.

*Development Status:* Pre-clinical stage of development.

*Inventors:* Thomas P. Conrads (NCI) *et al.*

*Relevant Publications*

1. BL Hood, MM Darfler, TG Guiel, B Furusato, DA Lucas, BR Ringeisen, IA Sesterhenn, TP Conrads, TD Veenstra, DB Krizman. Proteomic analysis of formalin-fixed prostate tissue. Mol Cell Proteomics 2005 Nov;4(11):1741–1753.

2. DA Prieto, BL Hood, MM Darfler, TG Guiel, DA Lucas, TP Conrads, TD Veenstra, DB Krizman. Liquid tissue™: proteomic profiling of formalin fixed tissues. Biotechniques 2005 Jun;38:S32–S35.

3. DS Kirkpatrick, SA Gerber, SP Gygi. The absolute quantification strategy: A general procedure for the quantification of proteins and post-translational modifications. Methods 2005 Mar;35(3):265–273.

4. AM Hawkridge *et al.* Quantitative mass spectral evidence for the absence of circulating brain natriuretic peptide (BNP–32) in severe human heart failure. Proc Natl Acad Sci USA 2005 Nov 29;102(48):17442–17447.

5. L Anderson and CL Hunter. Quantitative mass spectrometric MRM assays for major plasma proteins. Mol Cell Proteomics 2006 Apr;5(4):573–588.

*Patent Status:* PCT Application No. PCT/US2007/003478 filed 4 Sep 2008 (HHS Reference No. E–204–2006/0–PCT–01).

*Licensing Status:* Available for licensing.

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

**Tools To Identify Candidates for Effective Cancer Therapy: Antibodies to Human Asparagine Synthetase**

*Description of Technology:* Scientists at the National Institutes of Health (NIH) have developed peptide-specific polyclonal antibodies against human asparagine synthetase (ASNS), the enzyme that forms asparagine from aspartate using ATP. ASNS serves as a key biomarker for acute lymphoblastic leukemia (ALL) and other malignancies because these cancer cells express little or no ASNS compared to normal cells. As a result, these leukemia cells must acquire asparagine from the bloodstream to survive and proliferate to form tumors. Patients with ALL can be treated with L-asparaginase (L–ASP) to break down asparagine in the body and starve leukemia cells by preventing them from acquiring asparagine. The anti-ASNS antibodies could be used to detect ASNS levels in patient samples to help select patients that could benefit from L–ASP therapy. Studies at the NIH have shown that L–ASP therapy may prove to be a useful treatment for other types of cancer besides leukemia.

*Applications*

- Diagnostic tool to detect levels of asparagine synthetase (ASNS) in human samples to identify cancer patients that can benefit from L-asparaginase (L–ASP) treatment.
- Screening tool to identify other cancer cell types treatable by L–ASP therapy, such as ovarian cancer cells, which show diminished ASNS levels.
- Research tool to quantitate levels of ASNS in laboratory procedures, including various immunoassays, flow cytometry, and tissue sample analysis.

*Advantages:* These antibodies have been validated in immunoassays that showed that ASNS expression in a strong predictor of L–ASP efficacy in NCI–60 ovarian cancer cell lines.

*Inventors:* Paul K. Goldsmith *et al.* (NCI).

*Relevant Publications*

1. PL Lorenzi *et al.* Asparagine synthetase as a causal, predictive biomarker for L-asparaginase activity in ovarian cancer cells. Mol Cancer Ther. 2006 Nov;5(11):2613–2623.

2. KJ Bussey *et al.* Integrating data on DNA copy number with gene expression levels and drug sensitivities in the NCI–60 cell line panel. Mol Cancer Ther. 2006 Apr;5(4):853–867.

3. PL Lorenzi *et al.* Asparagine synthetase as a predictive biomarker for L-asparaginase activity in ovarian cancer cells. Mol Cancer Ther. 2008 Oct;7(10):3123–3128.

*Patent Status:* HHS Reference No. E–101–2006/0—Research Tool. Patent protection is not being pursued for this technology.

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

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

**Mouse Model With Targeted Disruption of the Neurofibromatosis Type-1 (Nf1) Gene**

*Description of Technology:* This invention relates to a mouse model having a targeted disruption of the neurofibromatosis type-1 (NF1) gene.

The neurofibromatosis (NF1) gene shows significant homology to mammalian GAP and is an important regulator of the Ras signal transduction pathway. To study the function of NF1 in normal development and to develop a mouse model of NF1 disease, the inventors have used gene targeting in ES cells to generate mice carrying a null mutation at the mouse Nf1 locus. Although heterozygous mutant mice, aged up to 10 months, have not exhibited any obvious abnormalities, homozygous mutant embryos die in utero. Embryonic death is likely attributable to a severe malformation of the heart. Interestingly, mutant embryos also display hyperplasia of neural crest-derived sympathetic ganglia. These results identify new roles for NF1 in development and indicate that some of the abnormal growth phenomena observed in NF1 patients can be recapitulated in neurofibromin-deficient mice. In addition, lethally-irradiated wild type mice transplanted with fetal liver cells taken from NF1 null embryos develop a form of juvenile chronic myelomonocytic leukemia (JMML) that is very similar to what is seen in children with NF1 disease.

*Applications*

- Research tool in studying some forms of human neuron diseases/injuries in addition to juvenile chronic myelomonocytic leukemia (JMML).
- Testing various therapeutic treatments for this disease.

*Inventors:* Neal G. Copeland *et al.* (NCI).

*Patent Status:* HHS Reference No. E–162–2004/0—Research Tool. Patent protection is not being pursued for this technology.

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

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

Dated: May 27, 2009.

**Richard U. Rodriguez,**

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

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**BILLING CODE 4140–01–P**

## DEPARTMENT OF HEALTH AND HUMAN SERVICES

### National Institutes of Health

### National Institute of General Medical Sciences; Notice of Closed Meeting

Pursuant to section 10(d) of the Federal Advisory Committee Act, as amended (5 U.S.C. App.), notice is hereby given of the following meeting.

The meeting will be closed to the public in accordance with the provisions set forth in sections 552b(c)(4) and 552b(c)(6), Title 5 U.S.C., as amended. The grant applications and the discussions could disclose confidential trade secrets or commercial property such as patentable material,