commercialize this technology. Please contact John Hewes, Ph.D. at 301–435–3121 or *hewesj@mail.nih.gov* for more information.

Dated: October 21, 2010.

**Richard U. Rodriguez,**

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

[FR Doc. 2010–27181 Filed 10–26–10; 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.

**Photosensitizing Antibody-Fluorophore Conjugate for Photo-Immunotherapy**

*Description of Invention:* A major goal of targeted cancer therapy is to improve the sensitivity and specificity of the therapy so that cancer cells can be detected and targeted for elimination, while normal cells in the surrounding area remain largely intact. Photodynamic therapy (PDT) is a treatment for cancer and non-cancerous lesions involving light and a photosensitizer. The photosensitizer can be targeted to a specific cell using antibodies specific for proteins expressed on the target cell surface, the target cells will then be destroyed after being exposed to light at appropriate wavelength.

The NIH technology describes a method of photosensitizing cancerous cells by irradiating an antibody fluorophore conjugate. The NIH investigators have conducted in vitro studies using a proprietary IRDye 700DX NHS Ester. The IR700 dye was conjugated to a proprietary humanized anti-HER1 or anti-HER2 or anti-PSMA antibody, Panitumumab or Trastuzumab or huJ591. Subsequent irradiation of non-ionizing near infrared light showed rapid cell death of tumor cells, while normal cells were not noticeably killed. The studies were repeated in mice with similar results.

*Applications and Market:*

• Photodynamic therapy for cancer by selective targeting and killing of cells without suffering normal tissue side effects.

• Cancer was responsible for about 13% of all human deaths in 2007. There remains a need for therapies that effectively kill the tumor cells while not harming non-cancerous cells.

*Development Status:* Both *in vitro* and *in vivo* data available.

*Inventors:* Hisataka Kobayashi, Peter L. Choyke, Makoto Mitsunaga (NCI)

*Publications:* Manuscript in submission.

*Patent Status:* U.S. Provisional Patent Application No. 61/363,079, filed July 9, 2010 (HHS Reference No. E–205–2010/0–US–01)

*Licensing Status:* Available for licensing.

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

**Soluble Glypican-3 Protein for Treatment of Cancer**

*Description of Technology:* Hepatocellular carcinoma (HCC) is a form of liver cancer that is among the more deadly cancers in the world. HCC is typically only detected at the later stages of cancer development, which is always associated with poor prognosis. Because HCC is often associated with liver disease, traditional chemotherapy is not an option, making surgery the most common form of treatment. As a result, there is a need for new treatments.

Glypican-3 (GPC3) is a cell surface protein that is normally involved in cell growth and differentiation. GPC3 has been shown to act through the Wnt-signaling pathway, a pathway that is often activated in a number of different cancer cell types. Significantly, the ability of GPC3 to activate signaling through Wnt requires that GPC3 be bound to the cell membrane. GPC3 is also preferentially expressed on HCC cells, suggesting it could play a particularly important role in tumorigenesis in HCC.

This invention concerns a soluble form of GPC3 that lacks its cell membrane anchoring domain. This soluble form of GPC3 maintains its ability to interact with the Wnt signaling pathway, but cannot induce the activation of the pathway because it is not bound to the cell membrane. By competing with fully functional GPC3, the soluble GPC3 is able to inhibit the growth of HCC cells, thereby decreasing the ability of tumors to grow and metastasize. This suggests that soluble GPC3 represents a possible therapeutic for HCC.

*Applications:*

• Soluble GPC3 represents a potential therapeutic for patients with cancer with hyperactivated Wnt-signaling pathways.

• Specific cancers include hepatocellular cancer (HCC), melanoma, thyroid cancer, lung squamous cell carcinoma, Wilms' tumor, neuroblastoma, hepatoblastoma, and testicular germ-cell tumors.

*Advantages:*

• Removal of the glycosyl-phosphatidylinositol (GPI) anchor results in a soluble form of GPC3 that can interrupt Wnt-signaling.

• Soluble GPC3 maintains the ability to compete with fully functional GPC3 despite its inability to activate signaling.

• For treatment of HCC, offers a non-invasive, potentially non-liver toxic alternative to current strategies.

*Development Status:* Preclinical stage of development; cell culture data with HCC cells

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

*For more information, see:*

• "Recombinant soluble glypican 3 protein inhibits the growth of hepatocellular carcinoma in vitro" Feng *et al.* Int. J. Cancer: E-pub (8 July 2010).

• "Soluble Glypican 3 inhibits the growth of Hepatocellular Carcinoma in vitro and in vivo" Zitterman *et al.* Int. J. Cancer: 126, 1291–1301 (2010).

*Patent Status:* U.S. provisional applications 61/334,135 (E–176–2010/0–US–01) and 61/350,722 (E–176–2010/1–US–01).

*Licensing Status:* Available for licensing.

*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 this technology. Please contact John Hewes,

PhD, at 301–435–3121 or *hewesj@mail.nih.gov* for more information.

**Drug Combination of DNA Topoisomerase I (TOP1) Inhibitors and Extracellular ATP Produces a Significant Increase in Beneficial Anti-Carcinoma Cytotoxicity**

*Description of Invention:* DNA Topoisomerase inhibitors are a category of drugs used for cancer therapy. DNA topoisomerase 1 (TOP1) inhibitors, such as Camptothecin (CPT) and its structurally related analogues, bind to the TOP1 complex and prevent the religation of the single strand DNA molecules, ultimately leading to cell death. CPT and close analogues show anticancer activity in clinical trials treating ovarian, small-cell lung, and colorectal cancers, but also adverse drug reaction. By reducing the cytotoxic dose in the thousands of folds, the NIH scientists are able to target the tumor and reduce the cytotoxicity to normal cells. The instant invention discloses that the drug combination of DNA topoisomerase 1 (TOP1) inhibitors, such as the anti-cancer drug Camptothecin (CPT), and extracellular ATP produces a significant increase in beneficial anti-carcinoma cytotoxicity.

*Applications and Market:*

• This invention may provide a new combination of drug with extracellular ATP to target various cancers for treatment.

• Cancer is the second leading cause of death in the U.S. The National Cancer Institute estimate the overall annual costs for cancer in the U.S. at $107 billion; development of more effective therapies with less adverse drug reaction is always in high demand.

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

*Inventors:* Joseph Riss, Glenn Merlino, J. Carl Barrett (NCI).

*Publications:* Manuscript in preparation.

*Patent Status:* U.S. Provisional Application No. 61/350,660 filed 02 Jun 2010 (HHS Reference No. E–098–2010/0–US–01).

*Licensing Status:* Available for licensing.

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

**Novel Prognostic and Therapeutic Biomarker for Cancer and Inflammatory Diseases**

*Description of Invention:* There remains a significant unmet need for diagnostics, prognostics, and therapeutics for conditions that involve inflammation and the formation of blood clots such as bleeding disorders, trauma, and diseases such as sepsis, cardiovascular disease, stroke, and cancer. The global market for such products is varied and competitive, and is forecast to be over $40 billion by 2010.

Researchers at the National Cancer Institute (NCI) have identified that levels of a novel soluble protein involved in the repair mechanism for damaged blood vessels correlate with outcome in sepsis and with the diagnosis of disseminated intravascular coagulation, a contributing factor to the morbidity and mortality associated with sepsis.

Further, the NCI researchers have demonstrated that a recombinant version of this novel protein facilitates the clotting of blood, suggesting a potentially significant therapeutic benefit for the treatment of bleeding disorders or trauma.

*Applications:*

• Diagnostic and prognostic biomarker for diseases that involve inflammation and blood clot formation (*i.e.,* sepsis, cardiovascular disease, stroke, cancer).

• Treatment of bleeding disorders or trauma.

• Treatment of cerebral bleeding associated with aneurism or stroke.

• Therapy for patients with low platelet counts.

• Therapy for women suffering from preeclampsia or thrombotic episodes.

*Advantages:*

• High specificity.

• Protein levels correlate with disease state/outcome.

• Administration of recombinant protein accelerates the formation of blood clots.

*Development Status:* Pre-clinical.

*Inventors:* Daniel McVicar *et al.* (NCI).

Relevant Publications:

1. Washington AV *et al.* TREM-like transcript-1 protects against inflammation-associated hemorrhage by facilitating platelet aggregation in mice and humans. J Clin Invest. 2009 Jun;119(6):1489–1501. [PubMed: 19436112].

2. Ford JW, McVicar DW. TREM and TREM-like receptors in inflammation and disease. Curr Opin Immunol. 2009 Feb;21(1):38–46. [PubMed: 19230638].

*Patent Status:*

• U.S. Provisional Application No. 61/177,242 filed 11 May 2009 (HHS Reference No. E–197–2009/0–US–01).

• PCT Application No. PCT/US10/34263 filed 10 May 2010 (HHS Reference No. E–197–2009/0–PCT–02).

*Licensing Status:* Available for licensing.

*Licensing Contact:* Patrick P. McCue, PhD; 301–435–5560; mccuepat@mail.nih.gov.

**Treatment and Prevention of Inflammatory Bowel Disease (IBD) Using Mutant and Chimeric IL–13 Molecules**

*Description of Invention:* Ulcerative colitis (UC) is a chronic inflammatory disease of the colorectum and affects approximately 400,000 people in the United States. The cause of UC is not known, although an abnormal immunological response to bacterial antigens in the gut microflora is thought to be involved. Present treatments for UC include anti-inflammatory therapy using aminosalicylates or corticosteroids, as well as immunomodulators and diet. However, 25–40% of ulcerative colitis patients must eventually have their colons removed due to massive bleeding, severe illness, rupture of the colon, risk of cancer or due to side effects of corticosteroids and novel treatments are still actively being sought. NIH scientists and their collaborators have used a mouse model of experimental colitis (oxazolone colitis, OC) to show that IL–13, a Th2 cytokine, is a significant pathologic factor in OC and that neutralizing IL–13 in these animals effectively prevents colitis.

OC is a colitis induced by intrarectal administration of a relatively low dose of the haptenating agent oxazolone subsequent to skin sensitization with oxazolone. A highly reproducible and chronic colonic inflammation is obtained that is histologically similar to human ulcerative colitis. Studies show that Natural Killer T (NKT) cells, rather than conventional CD4+T cells, mediate oxazolone colitis and are the source of IL–13 as well as being activated by CD1-expressing intestinal epithelial cells. Tissue removed from ulcerative colitis patients were also shown to contain increased numbers of nonclassical NKT cells that produce markedly increased amounts of IL–13 and that in keeping with epithelial damage being a key factor in UC, these NKT cells are cytotoxic for epithelial cells. Building on their previous work, scientists at NIAID and FDA have shown that an Il-13 chimeric fusion protein linked to an effector molecule was able to prevent colitis in a mouse model of ulcerative colitis.

Available for licensing are methods for treating or preventing the inflammatory response of IBD by inhibiting the binding of IL–13 to IL–13 receptors on NKT cells. Additionally, these mutant and chimeric Il-13 molecules are able to block the chronic

inflammatory response that results in fibrosis as seen in Crohn's disease. Preventing the inflammatory response of colitis by either modulating or blocking IL–13 and NKT cell activity continues to be an effective therapeutic approach in animal models of colitis with implications for the treatment of human ulcerative colitis and for the treatment of fibrosis associated with Crohn's disease.

*Inventors:* Warren Strober (NIAID), Ivan J. Fuss (NIAID), Peter Mannon (NIAID), Jan Preiss (NIAID), Raj Puri (FDA), Koji Kawakami (FDA), Stefan Fichtner-Feigl (NIAID), Atsushi Kitani (NIAID).

Related Publications:

1. F Heller, IJ Fuss, EW Nieuwenhuis, RS Blumberg, W Strober. Oxazolone colitis, a Th2 colitis model resembling ulcerative colitis, is mediated by IL–13-producing NK–T cells. Immunity 2002 Nov;17(5):629–628. [PubMed: 12433369].

2. IJ Fuss, F Heller, M Boirivant, F Leon, M Yoshida, S Fichtner-Feigl, Z Yang, M Exley, A Kitani, RS Blumberg, P Mannon, W Strober. Nonclassical CD1d-restricted NK T cells that produce IL–13 characterize an atypical Th2 response in ulcerative colitis. J Clin Invest. 2004 May 15;113(10):1490–1497. [PubMed: 15146247].

*Patent Status:* U.S. Patent Application No. 11/918,711 filed 14 Apr 2006 (HHS Reference No. E–003–2005/0–US–03) and related international filings.

Related Technologies:

- IL–13 modulators and inhibitors—U.S. Patent No. 7,666,411 issued 23 Feb 2010 (HHS Reference No. 131–2002/0–US–02), U.S. Patent Application No. 12/709,029 filed 19 Feb 2010 (HHS Reference No. E–131–2002/0–US–10), and related international filings.
- NF-kappa B decoy oligonucleotides—U.S. Patent Application No. 11/920,214 filed 09 Nov 2007 (HHS Reference No. E–108–2005/0–US–03).

*Licensing Status:* Available for licensing.

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

Dated: October 21, 2010.

**Richard U. Rodriguez,**

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

[FR Doc. 2010–27177 Filed 10–26–10; 8:45 am]

**BILLING CODE 4140–01–P**

## DEPARTMENT OF HEALTH AND HUMAN SERVICES

### National Institutes of Health

### Guidelines for Use of Stored Specimens and Access to Ancillary Data and Proposed Cost Schedule: Stored Biologic Specimens and Ancillary Data From the Collaborative Perinatal Project (CPP)

**ACTION:** Notice and request for comments.

**SUMMARY:** The Division of Epidemiology, Statistics and Prevention Research (hereafter, Division) of the *Eunice Kennedy Shriver* National Institute of Child Health and Human Development (NICHD) maintains an extensive repository of datasets from completed studies, biospecimens, and ancillary data. The Division intends to make datasets and biospecimens more widely available to the research community for use by qualified researchers and to establish procedures for access consistent with the National Institutes of Health (NIH) Data Sharing Policy. The Division has established an internal committee, the Biospecimen Repository Access and Data Sharing Committee (BRADSC), to oversee the repository access and data sharing program. *The purpose of this notice is to request comments on this program and present the initial proposed cost schedule.* After full consideration of comments submitted in response to this notice, the BRADSC will finalize proposal guidelines and procedures, publish the cost schedule to the Division Web site, and begin to accept proposals for use of the stored biologic samples and for access to ancillary data that may not be available electronically. The first specimens and ancillary data that will be made available under this program are those from the national Collaborative Perinatal Project (CPP).

The CPP is a large, prospective cohort study, conducted by the National Institute of Neurological Diseases and Stroke (NINDS) of the National Institutes of Health (NIH), which recruited and enrolled 48,197 women who contributed 54,390 pregnancies that were prospectively followed from 1959–1966 at twelve academic medical centers across the United States. Custody for disposition of the CPP serum specimens was transferred to the Division from the NINDS in 1993 and for the microfiche archives in 1999. However, under the Federal Privacy Act of 1974 the samples and archive still belong to NINDS. Since 1992, the specimens have had limited public availability through Division investigators. Going forward, the Biospecimen Repository Access and Data Sharing Committee (BRADSC) will oversee the repository access and data sharing program. Access to other Division resources will be announced on the Division Web site. The BRADSC reserves the right to amend the procedures and costs schedules as necessary to maintain the integrity of the program and to suit the conditions under which other specimens were collected. Announcements and current proposal guidelines will be available under the Research link at *http://despr.nichd.nih.gov,* and interested researchers should consult the Division Web site for resources available, the most recent guidelines for proposal submission and evaluation, and cost schedules. Procedures may vary depending on the age and nature of the samples and original institutional review board (IRB) approval, although the general outline of the procedures should remain the same. Cost schedules may vary depending on the nature and complexity of the request.

No funding is provided as part of this notice nor will any be available as part of the program either to support laboratory analyses or data management. Samples will only be provided to approved projects upon receipt of evidence of necessary IRB approval(s), funding and payment of repository costs and shipping. Approved projects that do not obtain funding will be canceled within one year of their approval date. A more complete description of this program follows. Comments or requests for clarification on all aspects of the program are welcome.

**DATES:**

- *Comment Receipt Date:* December 15, 2010.
- *Invitation to Submit Proposal:* Proposals can be submitted on an ongoing basis.
- *Scientific Review Dates:* Technical Panels for reviews will be assembled beginning on January 1, May 1, or September 1 of the calendar year so that proposals can be evaluated well in advance of Federal funding deadlines.
- *Anticipated Distribution of Samples:* Within one month of demonstrable proof of applicant IRB approval and receipt of payment to cover repository costs and shipping.

**ADDRESSES:** To send comments and to request information, *contact:* Dr. Mary L. Hediger, Division of Epidemiology, Statistics and Prevention Research, *Eunice Kennedy Shriver* National Institute of Child Health and Human Development, 6100 Executive Blvd,