*Type of Review:* New Collection.

*Affected Public:* Individuals and Households, Businesses and Organizations, State, Local or Tribal Government.

*Average Expected Annual Number of activities:* 4.

*Respondents:* 740.

*Annual responses:* 740.

*Frequency of Response:* Once per request

*Average minutes per response:* 50.

*Burden hours:* 516.

An agency may not conduct or sponsor, and a person is not required to respond to, a collection of information unless it displays a currently valid Office of Management and Budget control number.

Dated: January 13, 2012.

**Glenda Conroy,**

*Executive Officer (OM Director), NIDA.*

[FR Doc. 2012–1267 Filed 1–20–12; 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.

**Enhancement of Cancer Imaging and Treatment With Somatostatin Analogs**

*Description of Technology:* Available for licensing is a novel method using short-term treatment with a glucocorticoid antagonist to increase the expression of somatostatin receptors in tumor cells and improve rates of tumor identification in patients with high cortisol levels.

Tumors express up to five different receptors for somatostatin analogs on their surface. This enables somatostatin and its analogs to bind to the tumor cells. When the compound has a radioactive or radiopharmaceutical ''tag'' it can allow the cell to be killed (via radiation) or imaged (via the radiopharmaceutical). Somatostatin analogs have variable affinity for the five somatostatin receptors (types 1–5). As a result, if tumors express less of the more avid receptors, imaging or treatment with the analogs is less likely to be successful. There is a large variability in functional type 2 receptor expression in these tumors. High cortisol levels (such as those seen in Cushing's syndrome) cause the type 2 receptor level to decrease, which (with type 5) is the primary binding site for $^{111}$1n-DTPA-D-Phe-pentetreotide, which is used to image tumors (in an octreotide nuclear medicine scan).

*Potential Commercial Applications:* Tumor imaging and radiopharmaceutical therapy using somatostatin analogs.

*Competitive Advantages:* Allows conversion of a negative to positive octreotide scan in patients with active hypercortisolism.

*Development Stage:* Pilot.

*Inventors:* Lynnette Nieman (NICHD), *et al.*

*Intellectual Property:* HHS Reference No. E–252–2011/0—U.S. Provisional Application No. 61/533,664 filed 12 Sep 2011.

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

**PARP Inhibitor/NO Donor Dual Prodrugs as Anticancer Agents**

*Description of Technology:* Scientists at NIH have developed a hybrid prodrug molecule with enhanced biological activity as anticancer agent. Novel cancer therapeutic strategies are in high demand. Diazeniumdiolate-based nitric oxide (NO)-releasing prodrugs are a growing class of promising anticancer agents. Poly (ADP-ribose) polymerase (PARP) inhibitors have also emerged as a promising class of therapeutic compounds for cancer. The two-component prodrug described in the instant invention is expected to deliver DNA damaging agent (NO release) along with an inhibitor of DNA repair (PARP inhibitor) simultaneously to a cancer cell. The prodrugs are activated by glutathione/glutathione S-transferase (GSH/GST) and release cytotoxic NO and a PARP inhibitor in the target cancer cell. The high levels of GSH/GST are often a feature of cancer cells. The compound is predicted to have strong synergy with other anticancer therapeutics.

*Potential Commercial Applications*

- Cancer therapeutics.
- Cancer therapeutics in combination with other anticancer therapies.

*Competitive Advantages:* Combination of DNA damaging agent and DNA repair inhibitor in one molecule has advantage over both individual drug treatments.

*Development Stage*

- Prototype.
- Early-stage.
- Pre-clinical.
- In vitro data available.

*Inventors:* Anna E. Maciag, Larry K. Keefer, and Joseph E. Saavedra (NCI).

*Publication:* PARP Inhibitor/NO Donor Dual Prodrugs as Anticancer Agents, manuscript in preparation.

*Intellectual Property:* HHS Reference No. E–220–2011/0—U.S. Patent Application No. 61/549,862 filed 21 Oct 2011.

*Related Technologies*

- HHS Reference No. E–093–1996/3—U.S. Patent No. 6,610,660 issued 26 Aug 2003.
- HHS Reference No. E–025–2010/0—PCT Application No. PCT/US2010/056446 filed 12 Nov 2010, which published as WO 2011/060215 on 19 May 2011

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

**Small Molecule Drugs for Treatment of Ataxia Telangiectasia or DNA Damage**

*Description of Technology:* Ataxia telangiectasia (A–T) is a rare neurodegenerative disease that is caused by mutations in the Ataxia Telangiectasia Mutated (ATM) gene, which is the chief activator of the cellular response to double stranded DNA breaks. Defects in this gene can lead to abnormal cell death, particularly in the brain and in the immune system, and the disease is also characterized by hypersensitivity to radiation and other DNA-damaging agents, as well as a predisposition to lymphoma. There is currently no effective treatment for this disease.

Investigators at the National Human Genome Research Institute (NHGRI) have shown that ATM-null cells treated with rottlerin, a small molecule protein kinase inhibitor, respond to double stranded DNA breaks by activating an

alternate DNA repair pathway. Similarly, ATM-null mice demonstrate increased protection from radiation when treated with this compound. Thus, rottlerin or related compounds may be an effective treatment for A–T or other diseases resulting from DNA damage.

*Potential Commercial Applications:* Therapy for ataxia telangiectasia or other diseases resulting from DNA damage.

*Competitive Advantages*

- There is currently no therapy for ataxia telangiectasia.
- Rottlerin is a readily-obtained, small molecule compound.

*Development Stage*

- Early-stage.
- In vitro data available.
- In vivo data available (animal).

*Inventors:* Wei Zheng *et al.* (NCTT).

*Intellectual Property:* HHS Reference No. E–038–2011/0—U.S. Provisional Application No. 61/524,177 filed 16 Aug 2011.

*Licensing Contact:* Tara L. Kirby, Ph.D.; (301) 435–4426; *tarak@mail.nih.gov*.

**Transgenic Human Interleukin-21 Mouse Model**

*Description of Technology:* Available for licensing is a mouse model that constitutively expresses human interleukin-21 (IL–21). Traditionally, human IL–21 transgenic mouse models are difficult to produce as those with high IL–21 levels exhibit growth retardation and die before sexual maturity. The investigators generated transgenic mice that express human IL–21, which can stimulate murine cells *in vitro* thereby providing an accurate model to elucidate IL–21's role in immunity, immune disorders, and cancer.

IL–21 is a type I cytokine whose receptor is expressed on T, B, and natural killer cells. IL–21 has pleiotropic actions ranging from augmenting the proliferation of T cells to driving the differentiation of B cells into memory cells and terminally differentiated plasma cells. Moreover, IL–21 has anti-tumor activity by augmenting natural killer cell activity. This mouse model allows studying human IL–21 in vivo and its role in a variety of diseases such as autoimmunity, immunodeficiency, allergy, and cancer.

*Potential Commercial Applications*

- Model to study human IL–21 in vivo.
- Research tool to elucidate IL–21's role in T, B, and natural killer cell function and regulating antibody production.
- Model to study IL–21's pathology in autoimmunity, immunodeficiency, allergy, and cancer.

*Competitive Advantages:* Mouse model that constitutively expresses human IL–21, without the negative side effects of growth retardation and high toxicity present in other human IL–21 transgenic mice.

*Development Stage*

- Pre-clinical.
- In vivo data available (animal).

*Inventors:* Warren Leonard and Katsutoshi Ozaki (NHLBI).

*Publication:* Ozaki K, *et al.* Regulation of B cell differentiation and plasma cell generation by IL–21, a novel inducer of Blimp-1 and Bcl-6. J Immunol. 2004 Nov 1;173(9):5361–5371. [PMID 15494482].

*Intellectual Property:* HHS Reference No. E–231–2010/0—Research Tool. Patent protection is not being pursued for this technology.

*Related Technologies*

- HHS Reference No. E–211–2002/1—U.S. Patent 7,332,645 issued 19 Feb 2008; U.S. Patent Application No. 11/958,540 filed 18 Dec 2007.
- HHS Reference No. E–120–2003/1—U.S. Patent 7,993,919 issued 09 Aug 2011.
- HHS Reference No. E–120–2003/2—U.S. Patent 7,378,276 issued 27 May 2008; U.S. Patent Application No. 12/126,166 filed 23 May 2008.
- HHS Reference No. E–137–2002/0—U.S. Patent Application No. 10/508,978 filed 19 Nov 2004; U.S. Patent Application No. 12/651,858 filed 04 Jan 2010.

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

**Method for Producing Significant Amounts of B19 Virus for Development of Killed or Attenuated Vaccines**

*Description of Technology:* Human parvovirus B19 (B19) is a common infection of children and adults and is the cause of fifth disease. B19 selectively infects erythroid progenitor cells of bone marrow, fetal liver and a small number of specialized cell lines. These specific cell lines demonstrate limited infectibility and commonly produce little or no virus following initial inoculation with B19. Current methods for producing infectious B19 require phlebotomy of infrequently available infected donors. The available technology describes a method of producing pure populations of human erythroid progenitor cells that are fully permissive to B19 infection. The ability to efficiently generate significant amounts of infectious B19V in cells is useful for the development of killed or attenuated vaccines, therapeutics and efficient diagnostic tools for prevention and treatment of B19V.

*Potential Commercial Applications*

- Human parvovirus B19 diagnostic.
- Vaccine manufacture.
- Research and development of anti-parvovirus agents.

*Competitive Advantages:* Method produces pure populations of human erythroid progenitor cells that are fully permissive of B19 infection.

*Development Stage*

- Pre-clinical.
- In vitro data available.

*Inventors:* Susan Wong and Neal S. Young (NHLBI).

*Publications*

1. Giarratana MC, *et al.* Ex vivo generation of fully mature human red blood cells from hematopoietic stem cells. Nat Biotechnol. 2005 Jan; 23(1):69–74. [PMID 15619619].
2. Freyssinier JM, *et al.* Purification, amplification and characterization of a population of human erythroid progenitors. Br J Haematol. 1999 Sep; 106(4):912–922. [PMID 10519992].

*Intellectual Property:* HHS Reference No. E–188–2006/0—U.S. Patent Application No. 12/301,960 filed 21 Nov 2008.

*Licensing Contact:* Kevin W. Chang, Ph.D.; (301) 435–5018; *changke@mail.nih.gov*.

*Collaborative Research Opportunity:* The NHLBI Hematology Branch is seeking statements of capability or interest from parties interested in collaborative research to further develop, evaluate or commercialize novel methods to produce parvovirus B19 and use as diagnostic or vaccine. For collaboration opportunities, please contact Dr. Neal Young at (301) 496–5093 or *youngns@mail.nih.gov*.

**HIV Therapeutics Utilizing Peptide Secreting Commensal Bacteria**

*Description of Technology:* Available for licensing and commercial development is a patent estate covering genetically engineered commensal bacteria compositions and their methods of use that secrete HIV infectivity interfering peptides with the aid of co-expressed translocation mediators such as *HylB, HylD* or t*olC* gene products. The bacteria can be, for example, *Escherichia coli,* and are preferably those that colonize the gastrointestinal or genitourinary tracts. The secreted anti-HIV peptide can be a

functional inhibitory fragment from the C-terminus of HIV, SHIV or SIV, or an inhibitory peptide derived from the N-terminus receptor-binding domain of SIV gp41, HIV–1 gp41, or HIV–2 gp41. The secreted anti-HIV peptide can also be a peptide from the allosteric domain of gp120, an extracellular loop of CCR5, an anti-CD4 immunoglobulin, a mimetic of CD4, an alpha-defensin or theta-defensin, a CD38 fragment homologous to the V3 loop of gp120, polphemusin II (a CXCR4 antagonist), a RANTES peptide that binds to CCR5 or an HIV surface binding peptide such as cyanovirin.

*Potential Commercial Applications:* HIV therapeutics.

*Competitive Advantages:* Utilizes naturally occurring commensal bacteria.

*Development Stage*

- Pre-clinical.
- In vivo data available (animal).

*Inventor:* Dean H. Hamer (NCI).

*Publications*

1. Lagenaur LA, *et al.* Prevention of vaginal SHIV transmission in macaques by a live recombinant Lactobacillus. Mucosal Immunol. 2011 Nov;4(6):648–657. [PMID 21734653].

2. Rao S, *et al.* Toward a live microbial microbicide for HIV: commensal bacteria secreting an HIV fusion inhibitor peptide. Proc Natl Acad Sci U S A. 2005 Aug 23;102(34):11993–11998. [PMID 16040799].

*Intellectual Property*

HHS Reference No. E–233–2004/0—

- U.S. Patent Application No. 11/710,512 filed 26 Feb 2007.
- Various international issued patents.

*Licensing Contact:* Michael Shmilovich, Esq.; (301) 435–5019; *shmilovm@mail.nih.gov*.

Dated: January 17, 2012.

**Richard U. Rodriguez,**

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

[FR Doc. 2012–1264 Filed 1–20–12; 8:45 am]

**BILLING CODE 4140–01–P**

## DEPARTMENT OF HEALTH AND HUMAN SERVICES

### National Institutes of Health

### Government-Owned Inventions; Availability for Licensing

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**Thioxothiazolidinone Derivatives—A Novel Class of Anti Cancer Agents**

*Description of Technology:* The invention provides for a novel class of heterocyclic compounds (*i.e.* thioxothiazolidinone derivatives) that exhibit anticancer activity in a unique mechanism. More specifically, the compounds of the invention act as inhibitors of the enzyme human tyrosyl DNA phosphodiesterase1 (Tdp1), a DNA repair enzyme involved in topoisomerase1 (Top1) mediated DNA damage, such as damage induced by the Top1 inhibitors and chemotherapeutic agents, camptothecins. As such, these compounds can serve as potentiators of camptothecins. The experimental data indeed point at a synergistic effect achieved in a combination therapy of the thioxothiazolidinone derivatives of the invention and the established anticancer agents camptothecins. Moreover, due to this synergistic effect, a lower therapeutic dose of the latter may be needed, resulting in reduced side effects. In addition, it is possible that the Tdp1 inhibitors of the invention may be effective as anti tumor agents on their own. This is based on the fact that Tdp1 is involved also in repairing DNA damage resulting from oxygen radicals, and the observation that tumors contain excess free radicals.

*Potential Commercial Applications*

- Effective cancer therapy in combination with camptothecins.
- Cancer therapy as standalone anti cancer agents.

*Competitive Advantages:* The compounds of the invention act in unique mechanism that can enhance the therapeutic efficacy of the anticancer drugs camptothecins, and at the same time can serve as standalone anticancer agents.

*Development Stage:* In vitro data available.

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

*Publications*

1. Marchand C, *et al.* Identification of phosphotyrosine mimetic inhibitors of human tyrosyl-DNA phosphodiesterase I by a novel AlphaScreen high-throughput assay. Mol Cancer Ther. 2009 Jan;8(1):240–248. [PMID 19139134].

2. Dexheimer TS, *et al.* Tyrosyl-DNA phosphodiesterase as a target for anticancer therapy. Anticancer Agents Med Chem. 2008 May;8(4):381–389. [PMID 18473723].

3. Dexheimer TS, *et al.* 4–Pregnen-21-ol-3,20-dione-21-(4-bromobenzenesufonate) (NSC 88915) and related novel steroid derivatives as tyrosyl-DNA phosphodiesterase (Tdp1) inhibitors. J Med Chem. 2009 Nov 26;52(22): 7122–7131. [PMID 19883083].

*Intellectual Property:* HHS Reference No. E–239–2011/0—U.S. Provisional Patent Application No. 61/545,308 filed 10 Oct 2011.

*Licensing Contact:* Uri Reichman, Ph.D., MBA; (301) 435–4616; *reichmau@mail.nih.gov.*

**Monospecific and Bispecific Human Monoclonal Antibodies Targeting IGF–II**

*Description of Technology:* The type 1 insulin-like growth factor (IGF) receptor (IGF1R) is over-expressed by many tumors and mediates proliferation, motility, and protection from apoptosis. Agents that inhibit IGF1R expression or function can potentially block tumor growth and metastasis. Its major ligands, IGF–I, and IGF–II are over-expressed by multiple tumor types. Previous studies indicate that inhibition of IGF–I, and/or IGF–II binding to its cognizant receptor negatively modulates signal transduction through the IGF pathway and concomitant cell proliferation and growth. Therefore, use of humanized or fully human antibodies against IGFs represents a valid approach to inhibit tumor growth. The present invention discloses two monoclonal antibodies, designated m610.27 and m630, and a bispecific monoclonal antibody, m660, generated by linking domains from m610.27 and m630. All three antibodies display high affinities for IGF–I and IGF–II in the pM to nM range. The antibodies inhibited signal transduction mediated by the IGF–1R interaction with IGF–I and IGF–II and blocked phosphorylation of IGF–IR and the