galactosyltransferases of the invention can be used to synthesize a variety of products that, until now, have been very difficult and expensive to produce.

The invention also provides amino acid segments that promote the proper folding of a galactosyltransferase catalytic domain and mutations in the catalytic domain that enhance folding efficiency and make the enzyme stable at room temperature. The amino acid segments may be used to properly fold the galactosyltransferase catalytic domains of the invention and thereby increase their activity. The amino acid segments may also be used to increase the activity of galactosyltransferases that are produced recombinantly. Accordingly, use of the amino acid segments according to the invention allows for production of [beta](1,4)-galactosyltransferases having increased enzymatic activity relative to [beta](1,4)-galactosyltransferases produced in the absence of the amino acid segments.

Applications: Synthesis of polysaccharide antigens for conjugate vaccines, glycosylation of monoclonal antibodies, and as research tools.

Development Stage: The enzymes have been synthesized and preclinical studies have been performed.

Inventors: Pradman K. Qasba, Boopathy Ramakrishnan, Elizabeth Boeggeman (NCI).

Patent Status: U.S. and Foreign Rights Available (HHS Reference No. E–230–2002/2).

Licensing Status: Available for exclusive or non-exclusive licensing.

Licensing Contact: Peter A. Soukas, J.D.; 301/435–4646; *soukasp@mail.nih.gov.*

Collaborative Research Opportunity: The National Cancer Institute's Nanobiology Program is seeking statements of capability or interest from parties interested in collaborative research to further develop, evaluate, or commercialize the use of galactose and modified galactose to be linked to an N-acetylglucosamine that may itself be linked to a variety of other molecules. Please contact John D. Hewes, PhD. at 301–435–3121 or *hewesj@mail.nih.gov* for more information.

Rapid Motion Perception MRI Navigator Method

Description of Technology: Available for licensing and commercial development is a non-breathhold flow sensitive navigator technique for reducing respiratory motion artifacts in magnetic resonance (MR) images. The method, called Rapid Motion Perception (RaMP), tracks bulk translational motion of the heart in real-time. The position of the blood volume is a direct representation of the heart position. RaMP tracks fast-moving blood volume during systole as a marker for the heart position, while suppressing stationary or slow moving spins. This approach allows cardiac navigation in two orthogonal directions simultaneously, eliminates the need to obtain empirical correlations between the diaphragm and the heart, and increases tracking reliability among individual patients. The method uses a spoiled-Fast Low Angle Shot (FLASH) navigator and incorporates an alternating pair of bipolar velocity-encoding gradients. Data at 1.5T indicate that RaMP is capable of correcting bulk motion of the heart over multiple cardiac cycles to within +/−1.43 mm in the superior-inferior direction and +/−0.84 mm in the anterior-posterior direction.

Applications:

Reduction of MR image artifacts due to respiration motion.

Real-time tracking of cardiac motion.

Market: Magnetic Resonance Imaging.

Development Status: Late-stage technology.

Inventors: Vinay M. Pai and Han Wen (NHLBI).

Patent Status: U.S. Patent Application No. 10/244,903 filed 16 Sep 2002 (HHS Reference No. E–164–2002/0–US–01).

Licensing Status: Available for exclusive or non-exclusive licensing.

Licensing Contact: Chekesha S. Clingman, Ph.D.; 301/435–5018; *clingmac@mail.nih.gov.*

Collaborative Research Opportunity: The NHLBI is seeking statements of capability or interest from parties interested in collaborative research to further develop, evaluate, or commercialize this technology. Please contact Lili Portilla at 301–594–4273 or via e-mail at *Lilip@nih.gov* for more information.

Dated: September 7, 2007.

Steven M. Ferguson,

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

[FR Doc. E7–18189 Filed 9–14–07; 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.

New and Improved Chemotherapy Adjuvants: Folate Based Inactivators of *O*^{6}-alkylguanine-DNA alkyltransferase (alkyltransferase)

Description of Technology: O^6-Benzylguanine derivatives, some *O*^{6}-benzylpyrimidines, and related compounds are known to be inactivators of the human DNA repair protein *O*^{6}-alkylguanine-DNA alkyltransferase (alkyltransferase). This repair protein is the primary source of resistance many tumor cells develop when exposed to chemotherapeutic agents that modify the *O*^{6}-position of DNA guanine residues. Therefore, inactivation of this protein can bring about a significant improvement in the therapeutic effectiveness of these chemotherapy drugs. The prototype inactivator *O*^{6}-benzylguanine is currently in clinical trials in the United States as an adjuvant in combination with the chloroethylating agent 1, 3-bis (2-chloroethyl)-1-nitrosourea (BCNU) and the methylating agent temozolomide. A similar alkyltransferase inactivator, *O*^{6}-(4-bromothenyl) guanine is in clinical trials in the UK.

This technology is directed to the discovery of a new class of potent alkyltransferase inactivators, based on folate ester derivatives of *O*^{6}-benzyl-2′-deoxyguanosine and of *O*^{6}-[4-(hydroxymethyl)benzyl] guanine. All the folate ester derivatives of *O*^{6}-benzyl-2′-deoxyguanosine were able to sensitize human tumor cells to killing by 1, 3-bis (2-chloroethyl)-1-nitrosourea with *O*^{6}-benzyl-3′-O-[γ-folyl]-2′-deoxyguanosine being the most active. The 3′ ester was found to be more potent than the 5′ ester and was more than an order of magnitude more active than *O*^{6}-

benzylguanine, which is currently in clinical trials.

Applications

Promising candidates as chemotherapy adjuvants for the treatment of cancer.

Therapeutic application for drug resistant tumors where acquired resistance is caused by O^6-alkylguanine-DNA alkyltransferase.

Advantages

The folate ester derivatives are highly water soluble.

Conjugation of folic acid to an alkyltransferase inactivating compound should allow targeting of delivery to cells that express folate receptor as many tumor cells are known to do.

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

Inventors: Drs. Gary Pauly (NCI), Robert C. Moschel (NCI), Sahar Javanmard (NCI), *et al.*

Patent Status: This technology consists of U.S. Provisional Application No. 60/915,510 foreign equivalents, entitled ''Inactivators of O^6-Alkylguanine-DNA Alkyltransferase'' (HHS Reference No. E–200–2007/0).

Related Technology: HHS Reference No. E–274–2003/0, entitled ''2-Amino-O^4-Substituted Pteridines and Their Use as Inactivators of O^6-Alkylguanine-DNA Alkyltransferase''.

Licensing Status: Available for exclusive and non-exclusive licensing.

Licensing Contact: Adaku Nwachukwu, J.D.; 301/435–5560; *madua@mail.nih.gov.*

Papilloma Pseudovirus for Detection and Therapy of Tumors

Description of Technology: There is extensive literature on the use of viral vectors, particularly those based on the adenovirus and AAV, to increase the potency of anti-tumor gene therapy. However, these approaches have had limited success because of limited anti-tumor effects and unacceptable toxicity. This invention describes the use of papillomavirus pseudoviruses (PsV) as a gene transfer technology and a tumor diagnostic method. Preliminary studies showed that PsV bind to cells that were transplanted with human ovarian tumor (Shin-3) while normal tissues were not affected. PsV does not infect several other normal intact tissues but continues to selectively infect additional cell types that are damaged. Additionally, the inventors have constructed oligoT PsV vectors that can be engineered to express certain cytotoxic genes to induce tumor regression and simultaneous increase human papilloma virus' immunogenicity. This technology could be an effective anti-tumor therapy because it has shown increased infection of compromised cells with an inability to infect normal cells thereby reducing potential toxicity to patients. In addition to a potential anti-cancer therapeutic, this technology could also be used as a diagnostic tool in the detection of tumor masses. Detection can be achieved through the use of fluorescent dye coupled particles of PsV that have preferential binding to tumor tissues and not normal tissues.

Applications

Method to treat and selectively target cancer with limited toxicity.

Method to accurately diagnose cancer.

Anti-tumor therapeutic vaccines.

Anti-tumor cytoxic gene therapy constructs.

Market

An estimated 1,444,920 new cancer cases in 2007.

600,000 cancer deaths in the U.S. in 2006.

It is estimated that market for cancer drugs would double to $50 billion a year in 2010 from $25 billion in 2006.

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

Inventors: Jeffrey Roberts, John T. Schiller, Douglas R. Lowy (NCI).

Publications

1. CB Buck, *et al.* Generation of HPV pseudovirions using transfection and their use in neutralization assays. Methods Mol Med. 2005;119:445–462.

2. CB Buck, *et al.* Efficient intracellular assembly of papillomaviral vectors. J Virol. 2004 Jan;78(2):751–757.

Patent Status: U.S. Provisional Application No. 60/928,495 filed 08 May 2007 (HHS Reference No. E–186–2007/0–US–01).

Licensing Status: Available for exclusive or non-exclusive licensing.

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

New Synthetic Variants of 2-(4-isothiocyanatobenzyl)-6-methyldiethylenetriamine Pentaacetic Acid (1B4M–DTPA): Novel Macromolecular MRI Contrast Agents

Description of Technology: The present invention describes the synthesis and use of two protected variants of the 2-(4-isothiocyanatobenzyl)-6-methyldiethylenetriamine pentaacetic acid (1B4M–DTPA) (also known as the commercial bifunctional chelator, tiuxetan), bearing either an isothiocyanate or a succinimidyl ester moiety, respectively. These molecules were synthesized for the following uses: (1) Use in the introduction of the chelator to the N-terminus of peptides, aptamers, PNA, etc. wherein deprotection or cleavage from resin or solid phase support of the product is possible and (2) introduction of the chelator to macromolecular structures such as dendrimer wherein this is accomplished in organic solvents eliminating the gross inefficiency of the prior aqueous methods.

In both uses, the elimination or delay of any aqueous chemistry steps in the synthesis process obviates the possibilities of contamination by spurious metals. Metal contaminations could compromise latter radiolabeling or can also hinder the introduction of paramagnetic ions such as Gd(II1) for MRI applications. The chemistry used in this synthetic process is very flexible and provides the basis for an extensive list of conjugation functional groups to be introduced.

Comparative MR imaging with these dendrimer based molecules revealed equivalent enhancement of the vessels and organs such as the kidney and liver.

Applications

Useful in the conjugation of nearly all peptides for targeting antigens/peptides associated with cancers.

Useful for modification of macromolecules such as dendrimer, carbon tubes, etc., for labeling with radioactive metal ions suitable for imaging and/or therapy and paramagnetics for MRI.

Advantages

The chemistry is very flexible and provides the basis for an extensive list of conjugation functional groups to be introduced.

The elimination of aqueous chemistry steps obviates the possibilities of contamination by spurious metals that could compromise subsequent radiolabeling.

The elimination of aqueous steps aids in the introduction of paramagnetic ions such as Gd(III) for MRI applications.

The general synthesis process provides a procedure for preparing dendrimer-based MR agents with higher yields and efficiency while enhancing versatility.

Benefits: In spite of advances in cancer therapeutics and diagnostics, more than 600,000 cancer deaths are estimated to occur in 2007. Early and accurate detection is a key component of successful clinical management of cancer. This technology can contribute to the development of better MRI agents

for diagnosing cancer and thus improve overall survival and quality of life of patients suffering from cancer.

Inventors: Drs. Martin Brechbiel and Heng Xu (NCI).

Development Status: Synthesis process and data available.

Patent Status: U.S. Provisional Application No. 60/864,503 filed 06 Nov 2006 (HHS Reference No. E–226–2006/0–US–01).

Publication: H Xu, CA Regino, M Bernardo, Y Koyama, H Kobayashi, PL Choyke, MW Brechbiel. Toward improved syntheses of dendrimer-based magnetic resonance imaging contrast agents: New bifunctional diethylenetriaminepentaacetic acid ligands and nonaqueous conjugation chemistry. J Med Chem. 2007 Jul 12;50(14):3185–3193. Epub 2007 Jun 7.

Licensing Contact: Mojdeh Bahar; 301/435–2950; *baharm@mail.nih.gov.*

Methods and Compositions for Treating FUS1 Related Disorders

Description of Technology: The FUS1 gene residing in the 3p21.3 chromosome region may function as a tumor suppressor gene. In animal models, disruption of FUS1 is associated with an increased frequency of spontaneous vascular tumors and signs of autoimmune disease. The investigators have in vivo data that demonstrate that FUS1 null mutants show a consistent defect in NK cell maturation that correlate with changes in the expression of IL–15. Injection of IL–15 into FUS1 knockout mice completely rescued the NK cell maturation defect suggesting that FUS1 plays an important role in the development and activation of the mammalian immune system.

Applications

Method to treat cancer, autoimmune diseases, and immune disorders such as HIV.

Method to boost immunity in conjunction with cancer and immune disorder therapies.

Method to diagnose FUS1 related disorders.

Animal model to study anti-tumor response and autoimmunity.

Market

An estimated 1,444,920 new cancer diagnoses in the U.S. in 2007.

600,000 deaths caused by cancer in the U.S. in 2006.

Cancer is the second leading cause of death in United States.

It is estimated that market for cancer drugs would double to $50 billion a year in 2010 from $25 billion in 2006.

An estimated 8.5 million Americans are afflicted with autoimmune diseases.

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

Inventors: Michael I. Lerman, *et al.* (NCI).

Publication: AV Ivanova, *et al.* Autoimmunity, spontaneous tumourigenesis, and IL–15 insufficiency in mice with a targeted disruption of the tumour suppressor gene Fus1. J Path. 2007 Apr;211(5):591–601.

Patent Status: PCT Patent Application No. PCT/US2006/026533 (HHS Reference No. E–137–2005/0–PCT–02).

Licensing Status: Available for exclusive or non-exclusive licensing.

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

Collaborative Research Opportunity: The National Cancer Institute Basic Research Laboratory is seeking statements of capability or interest from parties interested in collaborative research to further develop, evaluate, or commercialize cancer and immune disorder therapies Please contact John D. Hewes, Ph.D. at 301–435–3121 or *hewesj@mail.nih.gov* for more information.

Tumor Suppressor Genes

Description of Technology: Members of the inhibitor of growth (ING) family of tumor suppressor genes are involved in the regulation of diverse processes including cell cycle progression, apoptosis, and DNA repair as important cofactors of p53. ING members contain a highly evolutionary conserved sequence common in chromatin-regulating proteins, and there are overlapping functions between ING family members in negative regulation of cell growth as well as a dependent regulation between various ING members and p53.

Available for licensing are compositions for new tumor suppressor designated p28ING5, p33ING2, and p47ING3 (pING). Overexpression of these proteins has been shown to inhibit cell proliferation in human cancer cells lines, and these characteristics suggest that they may have important implications in cancer diagnosis and therapy. These compositions include nucleic acids, polypeptides, and antibodies that specifically bind to their respective ING members. Also claimed are cancer diagnostic and treatment methods.

Applications

Methods to treat and diagnose cancer with pING compositions.

Methods to identify pING modulating agents.

Research tool to study cell cycle regulation and p53 pathways. pING compositions.

Market

Cancer is the second leading cause of death in United States.

An estimated 600,000 deaths caused by cancer in 2006.

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

Inventors: Curtis C. Harris (NCI), *et al.*

Publications

1. T Okano, *et al.* Alterations in novel candidate tumor suppressor genes, ING1 and ING2 in human lung cancer. Oncol Rep. 2006 Mar;15(3):545–549.

2. H Kataoka, *et al.* ING1 represses transcription by direct DNA binding and through effects on p53. Cancer Res. 2003 Sep 15;63(18):5785–5792.

3. M Nagashima, *et al.* A novel PHD-finger motif protein, p47ING3, modulates p53-mediated transcription, cell cycle control, and apoptosis. Oncogene. 2003 Jan 23;22(3):343–350.

4. M Nagashima, *et al.* DNA damage-inducible gene p33ING2 negatively regulates cell proliferation through acetylation of p53. Proc Natl Acad Sci USA. 2001 Aug 14;98(17):9671–9676.

Patent Status

U.S. Patent No. 6,790,948 issued 14 Sep 2004 (HHS Reference No. E–272–1998/0–US–02)

U.S. Patent Application No. 10/868,270 filed 14 Jun 2004 (HHS Reference No. E–272–1998/0–US–03)

PCT Patent Application No. PCT/US2001/04425 filed 09 Feb 2001 (HHS Reference No. E–254–1999/0–PCT–02)

U.S. Patent Application No. 10/203,532 filed 02 Aug 2002 (HHS Reference No. E–254–1999/0–US–03)

PCT Patent Application No. PCT/US2003/02174 filed 23 Jul 2003 (HHS Reference No. E–300–2001/0–PCT–02)

U.S. Patent Application No. 10/502,431 filed 22 Jul 2004 (HHS Reference No. E–300–2001/0–US–03)

Licensing Status: Available for exclusive or non-exclusive licensing.

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

Peptide Inhibitor of Cyclin Dependent Kinase 4 (CDK4) Derived From MyoD

Description of Technology: This invention pertains to cell cycle regulation and the activity of the G1 cyclin-dependent kinase 4 (CDK4). The invention describes a 15 amino acid peptide and variants thereof derived from muscle determination factor,

MyoD, which is an inhibitor of the CDK4. CDK4 is one of a number of cyclin-dependent kinases which control progression through the cell cycle through their ability to phosphorylate particular substrates at the correct phase of the cell cycle. CDK4 has been shown to be involved in cell cycle control through its ability to regulate the activity of the retinoblastoma protein, pRb, an activator of genes essential for cell division.

Inhibitors of the cyclin-dependent kinases, such as the peptides described in this invention, prevent cell cycle progression and induce cells to exit the cell cycle into the Go state. The peptides described in this invention prevent the phosphorylation of pRb by CDK4, an obligate step for entry into the cell cycle. Osteosarcomas and habdosarcomas are two types of tumors known to over-express pRb. The inhibitor described in this invention may be useful in treating these cancers or other diseases which have been specifically linked to over-expression of active pRb.

Applications

Method to treat proliferative disorders, including cancer.

Anti-proliferative therapeutics.

Research tool to study the cell cycle.

Advantages: Expression of this peptide either as a fusion protein with GST or GFP results in the cessation of cell growth.

Market

An estimated 1,444,920 new cancer diagnoses in the U.S. in 2007.

600,000 deaths caused by cancer in the U.S. in 2006.

Cancer is the second leading cause of death in the United States.

It is estimated that market for cancer drugs would double to $50 billion a year in 2010 from $25 billion in 2006.

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

Inventors: Bruce M. Paterson and Jian-min Zhang (NCI).

Publication: JM Zhang, *et al.* Coupling of the cell cycle and myogenesis through the cyclin D1-dependent interaction of MyoD with cdk4. EMBO J. 1999 Feb 15;18(4):926–933.

Patent Status: U.S. Patent Application No. 10/018,964 filed 11 Apr 2002, claiming priority to 18 Jun 1999 (HHS Reference No. E–153–1998/0–US–03).

Licensing Status: Available for exclusive or non-exclusive licensing.

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

Collaborative Research Opportunity: The National Cancer Institute's Laboratory of Biochemistry and Molecular Biology is seeking statements of capability or interest from parties interested in collaborative research to further develop, evaluate, or commercialize the described cdk4 inhibitory peptides or equivalent peptide mimetics. Please contact John D. Hewes, PhD at 301–435–3121 or *hewesj@mail.nih.gov* for more information.

Dated: September 6, 2007.

Steven M. Ferguson,

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

[FR Doc. E7–18192 Filed 9–14–07; 8:45 am]

BILLING CODE 4140–01–P

DEPARTMENT OF HEALTH AND HUMAN SERVICES

National Institutes of Health

National Institute of Neurological Disorders And Stroke; Notice of Closed Meeting

Pursuant to section 10(d) of the Federal Advisory Committee Act, as amended (5 U.S.C. Appendix 2), notice is hereby given of a meeting of the Board of Scientific Counselors, National Institute of Neurological Disorders and Stroke.

The meeting will be closed to the public as indicated below in accordance with the provisions set forth in section 552b(c)(6), Title 5 U.S.C., as amended for the review, discussion, and evaluation of individual intramural programs and projects conducted by the National Institute of Neurological Disorders And Stroke, including consideration of personnel qualifications and performance, and the competence of individual investigators, the disclosure of which would constitute a clearly unwarranted invasion of personal privacy.

Name of Committee: Board of Scientific Counselors, National Institute of Neurological Disorders and Stroke.

Date: September 30–October 2, 2007.

Time: September 30, 2007, 7 p.m. to 10 p.m.

Agenda: To review and evaluate personal qualifications and performance, and competence of individual investigators.

Place: Hyatt Regency Bethesda, One Bethesda Metro Center, 7400 Wisconsin Avenue, Susquehanna/Severn Room, Bethesda, MD 20814.

Time: October 1, 2007, 8:30 a.m. to 6:30 p.m.

Agenda: To review and evaluate personal qualifications and performance, and competence of individual investigators.

Place: National Institutes of Health, Neuroscience Center, 6001 Executive Boulevard, Conference Room A, Rockville, MD 20852.

Time: October 2, 2007, 8:30 a.m. to 12 p.m.

Agenda: To review and evaluate personal qualifications and performance, and competence of individual investigators.

Place: Hyatt Regency Bethesda, One Bethesda Metro Center, 7400 Wisconsin Avenue, Susquehanna/Severn Room, Bethesda, MD 20814.

Contact Person: Alan P. Koretsky, PhD, Scientific Director, Division of Intramural Research. National Institute Of Neurological Disorders & Stroke, NIH, 35 Convent Drive, Room 6A 908, Bethesda, MD 20892, 301–435–2232, *koretskya@ninds.nih.gov.*

(Catalogue of Federal Domestic Assistance Program Nos. 93.853, Clinical Research Related to Neurological Disorders; 93.854, Biological Basis Research in the Neurosciences, National Institutes of Health, HHS).

Dated: September 6, 2007.

Jennifer Spaeth,

Director, Office of Federal Advisory Committee Policy.

[FR Doc. 07–4566 Filed 9–14–07; 8:45 am]

BILLING CODE 4140–01–M

DEPARTMENT OF HEALTH AND HUMAN SERVICES

National Institutes of Health

National Institutes of Nursing Research; Notice of Meeting

Pursuant to section 10(d) of the Federal Advisory Committee Act, as amended (5 U.S.C. Appendix 2), notice is hereby given of a meeting of the National Advisory Council for Nursing Research.

The meeting will be open to the public as indicated below, with attendance limited to space available. Individuals who play to attend and need special assistance, such as sign language interpretation or other reasonable accommodations, should notify the Contact Person listed below in advance of the 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, and personal information concerning individuals associated with the grant applications, the disclosure of which would constitute a clearly unwarranted invasion of personal privacy.

Name of Committee: National Advisory Council for Nursing Research.

Date: September 25–26, 2007.

Open: September 25, 2007, 1 p.m. to 5 p.m.