

award to the school in the form of a Federal Capital Contribution (FCC). The award is used to establish a distinct account for the NFLP loan fund at the school or is deposited into an existing NFLP fund. The school of nursing makes loans from the NFLP fund to eligible students enrolled full-time in a master's or doctoral nursing education program that will prepare them to become qualified nursing faculty. Following graduation from the NFLP lending school, loan recipients may receive up to 85 percent NFLP loan cancellation over a consecutive four-year period in exchange for service as full-time faculty at a school of nursing. The NFLP lending school collects any

portion of the loan that is not cancelled. The lending school deposits monies from loan collection and repayment into the NFLP loan fund to make additional NFLP loans. The school of nursing must keep records of all NFLP loan fund transactions.

The NFLP Annual Operating Report is used to collect information relating to the NFLP loan fund operations and financial activities for a specified reporting period (July 1 through June 30 of the academic year). Participating schools will complete and submit an electronic copy of the AOR annually to provide the Federal Government with current and cumulative information on: (1) The number and amount of loans

made, (2) the number of NFLP recipients and graduates, (3) the number and amount of loans collected, (4) the number and amount of loans in repayment, (5) the number of NFLP graduates employed as nurse faculty, and (6) NFLP loan fund receipts, disbursements and other related costs. The NFLP loan fund balance is used with other criteria to determine the annual award to the school.

Once the AOR is completed by the participating school, the AOR will be submitted electronically through the HRSA Electronic Handbook.

The estimate of burden for this form is as follows:

Form	Number of respondents	Responses per respondent	Total responses	Hours per responses	Total burden hours
Nurse Faculty Loan Program Annual Operating Report (AOR)	150	1	150	8	1200
Total Burden	150	1	150	8	1200

Written comments and recommendations concerning the proposed information collection should be sent within 30 days of this notice to the desk officer for HRSA, either by e-mail to OIRA_submission@omb.eop.gov or by fax to 202-395-6974. Please direct all correspondence to the "attention of the desk officer for HRSA."

Dated: September 10, 2007.

Alexandra Huttinger,

Acting Director, Division of Policy Review and Coordination.

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BILLING CODE 4165-15-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.

Suppression of Allergic Asthma by *Ascaris* Antigens

Description of Technology: Available for licensing and commercial development are compositions and methods for suppressing allergic reactions, as well as Th-1 and Th-2 associated immunological diseases, by administering any of the two identified *Ascaris* polypeptide antigens, or active fragments or variants thereof, to the affected subject.

Allergic asthma is characterized by antigen-specific IgE production, reversible airway hyper-reactivity and eosinophilic infiltration of the airways. There is a dramatic increase in the prevalence of allergic disorders in emerging and industrialized countries and studies suggest that the hygienic environment in those countries may not provide allergy-protective mechanisms associated with some forms of infection. Recent studies have found that helminth infection may suppress the development of allergic disease. Helminth infections currently affect over 2 billion people

worldwide, causing significant morbidity. The most successful geohelminths are members of the *Ascaris* species, including *A. lumbricoides* and *A. suum*, which are known to infect 1.5 billion people. The inventors studied the modulation of allergic disease mediated by a chronic *A. suum* infection in their murine model of ragweed-induced allergic conjunctivitis and allergic asthma, and demonstrated that the infection prevents allergic inflammation in sites distal from larval migration. This protection was due, in part, to the induction of immunoregulatory cytokines such as IL-10. In further studies, they demonstrated that a cocktail of antigens from the pseudocoelomic fluid (PCF) of *A. suum*, administered during ragweed sensitization, significantly reduced the eosinophil migration into the conjunctiva, pulmonary eosinophilic inflammation, and total lung pathology induced by the ragweed. PCF exposure also reduced the secretion of the pro-allergic cytokines IL-5 and IL-13 in the broncho-alveolar lavage fluid after ragweed exposure. All findings suggest PCF is capable of suppressing the allergic response to a traditional allergen and at multiple tissue sites.

In further studies, the inventors determined that the protection conferred by PCF to allergic inflammation was through a specific first antigenic protein isolated from PCF, results that were confirmed by using the recombinant form of the first antigen.

Furthermore, it is known that Toll-like receptors (TLRs) on dendritic cells (DCs) and other antigen presenting cells recognize specific molecular patterns on invading pathogens, leading to the development of host immunity. A number of pathogens, including helminths, have used pattern recognition by TLRs to modulate host immunity and inflammation to establish a chronic infection. In further studies, the inventors identified a second specific antigenic protein, also isolated from PCF, which can modulate activation of bone marrow derived DCs in response to stimuli with bacterial lipopolysaccharide (LPS); and to stimulate DCs to produce significant increases in IL-10 but not IL-12 upon co-stimulation with LPS. Studies in various genetically deficient mice suggested that this second antigen augments the IL-10 production dependent on one of the TLRs, TLR4. In further studies with the cloned and expressed form of the second antigen, as well as its two domains, the inventors showed that the activity is dependent on domain 2 but not domain 1. The purified second antigen exhibits different properties than unfractionated PCF. PCF administration prevents an initial response from occurring, as it inhibits the initiation of the inflammatory cascade. By contrast, the second antigen can activate DCs and alter cells such that they ultimately suppress responses through the production of IL-10 and can therefore act on the effector phase of the inflammatory response (i.e., modulate a response that is already occurring).

Applications: Suppression of allergic responses to traditional allergens by administering the identified *Ascaris* polypeptide antigens, or active fragments or variants thereof, to the affected subjects. The inventions provide different ways to treat allergic diseases or prevent allergic reactions, rather than merely ameliorating the symptoms. The inventions are also applicable to other Th-1 and Th-2 associated immunological diseases.

Development Status: The technologies are currently in the pre-clinical stage of development.

Inventors: Andrea Keane-Myers *et al.* (NIAID).

Relevant Publications: Manuscripts describing the above technologies will be available as soon as they are accepted for publication.

Patent Status:

U.S. Provisional Application No. 60/902,506 filed 22 February 2007 (HHS Reference No. E-126-2007/0-US-01).

U.S. Provisional Application No. 60/924,537 filed 18 May 2007 (HHS Reference No. E-174-2007/0-US-01).

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

Licensing Contact: Cristina Thalhammer-Reyero, Ph.D, MBA; 301/435-4507; thalhamc@mail.nih.gov.

***Citrobacter freundii* WR7011 as a Vaccine Strain or Source of Vi Capsular Antigen for Protection Against Typhoid Fever**

Description of Invention: According to the WHO, typhoid fever remains a serious public health problem throughout the world, with an estimated 16-33 million cases and 500,000 to 600,000 deaths annually. The Vi capsule of *S. typhi*, the causative agent of typhoid fever, is a surface-bound carbohydrate polymer to which antibodies have been shown to protect against typhoid fever. Purification of this polymer from virulent *S. typhi* strains poses a danger to those handling the live organisms. However, an unusual strain of *Citrobacter freundii*, WR7004 was mutated by the inventors to create a strain (WR7011) that makes Vi polysaccharide on its surface. Specifically, the strain was mutated using nitrosoguanidine. *C. freundii* WR7011 makes several times as much Vi polysaccharide as strains of *S. typhi*, is nonpathogenic, and is much safer to work with for Vi production or use as a vaccine strain. The inventors anticipate that this strain of *C. freundii* will reduce costs of purifying the Vi polysaccharide and also provide an increased level of safety during manufacture of the polysaccharide.

Applications and Modality: Synthesis of *S. typhi* Vi polysaccharide.

Market: Research tool useful for vaccine studies and/or vaccine production.

Development Status: The technology is a research tool.

Inventors: Dennis Kopecko and DeQi Xu (CBER/FDA).

Pertinent References:

1. NJ Snellings *et al.* Genetic regulation of variable Vi antigen expression in a strain of *Citrobacter freundii*. *J Bacteriol.* 1981 Feb;145(2):1010-1017.
2. H-S Houg *et al.* Expression of Vi antigen in *Escherichia coli* K-12: characterization of ViaB from *Citrobacter freundii* and identity of ViaA with RcsB. *J Bacteriol.* 1992 Sep;174(18):5910-5915.
3. JT Ou *et al.* Specific insertion and deletion of insertion sequence 1-like DNA element causes the reversible expression of the virulent capsular antigen Vi of *Citrobacter freundii* in

Escherichia coli. *Proc Natl Sci USA.* 1988 June;85(12):4402-4405.

4. SC Szu *et al.* Vi capsular polysaccharide-protein conjugates for prevention of typhoid fever. *J Exp Med.* 1987 Nov 1;166(5):1510-24.

Patent Status: HHS Reference No. E-004-2007/0—Research Tool.

Licensing Status: This technology is not patented. The mouse model will be transferred through a Biological Materials License.

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

Collaborative Research Opportunity: The FDA-CBER Laboratory of Enteric and Sexually Transmitted Diseases is seeking statements of capability or interest from parties interested in collaborative research to further develop, evaluate, or commercialize Vi polysaccharide from *Citrobacter freundii*. Please contact Dr. Dennis J. Kopecko at 301-496-1893 or (dennis.kopecko@fda.hhs.gov) for more information.

Catalytic Domains of [beta](1,4)-galactosyltransferase I Having Altered Donor and Acceptor Specificities, Domains That Promote In Vitro Protein Folding, and Methods for Their Use

Description of Technology: [beta](1,4)-galactosyltransferase I catalyzes the transfer of galactose from the donor, UDP-galactose, to an acceptor, N-acetylglucosamine, to form a galactose-[beta](1,4)-N-acetylglucosamine bond. This reaction allows galactose to be linked to an N-acetylglucosamine that may itself be linked to a variety of other molecules. The reaction can be used to make many types of molecules having great biological significance. For example, galactose-[beta](1,4)-N-acetylglucosamine linkages are very important for cellular recognition and binding events as well as cellular interactions with pathogens, such as viruses. Therefore, methods to synthesize these types of bonds have many applications in research and medicine to develop pharmaceutical agents and improved vaccines that can be used to treat disease.

The present invention is based on the surprising discovery that the enzymatic activity of [beta](1,4)-galactosyltransferase can be altered such that the enzyme can make chemical bonds that are very difficult to make by other methods. These alterations involve mutating the enzyme such that the mutated enzyme can transfer many different types of sugars from sugar nucleotide donors to many different types of acceptors. Therefore, the mutated [beta](1,4)-

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: Cheksha 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.

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New and Improved Chemotherapy Adjuvants: Folate Based Inactivators of O⁶-alkylguanine-DNA alkyltransferase (alkyltransferase)

Description of Technology: O⁶-Benzylguanine derivatives, some O⁶-benzylpyrimidines, and related compounds are known to be inactivators of the human DNA repair protein O⁶-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 O⁶-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 O⁶-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, O⁶-(4-bromophenyl) 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 O⁶-benzyl-2'-deoxyguanosine and of O⁶-[4-(hydroxymethyl)benzyl] guanine. All the folate ester derivatives of O⁶-benzyl-2'-deoxyguanosine were able to sensitize human tumor cells to killing by 1, 3-bis (2-chloroethyl)-1-nitrosourea with O⁶-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 O⁶-