cost to respondents. There are no Capital Costs, Operating Costs and/or Maintenance Costs to report.

ANNUAL BURDEN HOURS FOR RESPONDENTS

Type of respondents	Estimated No. of respondents	Frequency of response	Activity	Average time per response	Estimated annual burden hours
NIH-Funded Behavioral Researchers.	50	1	Peruse Site	.168	8
	20	1	Complete Form	.5	10
High School Students	50	1	Peruse Site	.25	12
	5	1	Complete Form	.74	4
College Students	70	1	Peruse Site	.25	17
	15	1	Complete Form	.668	10
Graduate Students	100	1	Peruse Site	.25	25
	25	1	Complete Form	.5845	15
Post-doctoral Fellows	65	1	Peruse Site	.25	16
	20	1	Complete Form	.5	10
Junior Faculty	65	1	Peruse Site	.25	16
	10	1	Complete Form	.5	5
Total per year	400				148

Request for Comments: Written comments and/or suggestions from the public and affected agencies are invited on one or more of the following points: (1) Whether the proposed collection of information is necessary for the proper performance of the function of the agency, including whether the information will have practical utility; (2) The accuracy of the agency's estimate of the burden of the proposed collection of information, including the validity of the methodology and assumptions used; (3) Ways to enhance the quality, utility, and clarity of the information to be collected; and (4) Ways to minimize the burden of the collection of information on those who are to respond, including the use of appropriate automated, electronic, mechanical, or other technological collection techniques or other forms of information technology.

Direct Comments to OMB: Written comments and/or suggestions regarding the item(s) contained in this notice, especially regarding the estimated public burden and associated response time, should be directed to the: Office of Management and Budget, Office of Regulatory Affairs, New Executive Office Building, Room 10235, Washington, DC 20503, Attention: Desk Officer for NIH. To request more information on the proposed project or to obtain a copy of the data collection plans and instruments, contact: Ms. Dana Sampson, Program Analyst, OBSSR, OD, NIH Building 1, Room 256, 1 Center Drive, Bethesda, MD 20892, or call non-toll-free number (301) 402–1146 or E-mail your request, including your address to: *SampsonD@od.nih.gov*.

Comments Due Date: Comments regarding this information collection are best assured of having their full effect if received within 30 days of the date of this publication.

Dated: July 27, 2005.

LaVerne Stringfield,

Acting Executive Officer, Office of the Director, National Institutes of Health.

[FR Doc. 05–15239 Filed 8–2–05; 8:45 am]

BILLING CODE 4140–01–M

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.

A Method With Increased Yield for Production of Polysaccharide-Protein Conjugate Vaccines Using Hydrazide Chemistry

Che-Hung Robert Lee and Carl Frasch (FDA).

U.S. Provisional Application No. 60/493,389 filed 06 Aug 2003 (HHS Reference No. E–301–2003/0–US–01);

PCT Application No. PCT/US04/25477 filed 06 Aug 2004 (HHS Reference No. E–301–2003/0–PCT–02);

PCT Application No. PCT/US04/26431 filed 06 Aug 2004 (HHS Reference No. E–301–2003/1–PCT–01).

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

Current methods for synthesis and manufacturing of polysaccharide-protein conjugate vaccines employ conjugation reactions with low efficiency (about twenty percent). This means that up to eighty percent of the added activated polysaccharide (PS) is lost. In addition, inclusion of a chromatographic process for purification of the conjugates from unconjugated PS is required.

The present invention utilizes the characteristic chemical property of hydrazide groups on one reactant to react with aldehyde groups or cyanate esters on the other reactant with an improved conjugate yield of at least sixty percent. With this conjugation efficiency the leftover unconjugated protein and polysaccharide would not need to be removed and thus the purification process of the conjugate product can be limited to diafiltration to

remove the by-products of small molecules. The new conjugation reaction can be carried out within one or two days with reactant concentrations between 1 and 25 mg/mL at PS/protein ratios from 1:2 to 3:1, at temperatures between 4 and 40 degrees Centigrade, and in a pH range of 5.5 to 7.4, optimal conditions varying from PS to PS.

Therefore, this invention can reduce the cost of conjugate vaccine manufacture.

Modulators of Nuclear Hormone Receptor Activity: Novel Compounds, Diverse Applications for Infectious Diseases, Including Anthrax (B. anthracis)

E.M. Sternberg (NIMH), J.I. Webster (NIMH), L. H. Tonelli (NIMH), S. H. Leppla (NIAID), and M. Maoyeri (NIAID).

U.S. Provisional Application No. 60/416,222 filed 04 Oct 2002 (HHS Reference No. E–247–2002/0–US–01);

U.S. Provisional Application No. 60/419,454 filed 18 Oct 2002 (HHS Reference No. E–348–2003/0–US–01);

PCT Application No. PCT/US03/31406 filed 03 Oct 2003 (HHS Reference No. E–247–2002/1–PCT–01);

U.S. Patent Application No. 10/530,254 filed 04 Apr 2005 (HHS Reference No. E–247–2002/1–US–02).

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

Technology summary and benefits: Nuclear hormones such as glucocorticoids dampen inflammatory responses, and thus provide protection to mammals against inflammatory disease and septic shock. The Anthrax lethal factor represses nuclear hormone receptor activity, and thus may contribute to the infectious agent causing even more damage to the host. This observation can be exploited to find new means of studying and interfering with the normal function of nuclear hormone receptors. Scientists at NIH have shown that under the appropriate conditions, these molecules can be used to modulate the activity of various nuclear hormone receptors. Identifying useful agents that modify these important receptors can provide relief in several human disorders such as inflammation, autoimmune disorders, arthritis, malignancies, shock and hypertension.

Long-term potential applications: This invention provides novel agents that can interfere with the action of nuclear hormone receptors. It is well known that malfunction or overdrive of these receptors can lead to a number of diseases such as enhanced inflammation; worse sequelae of infection including shock; diabetes; hypertension and steroid resistance. Hence a means of controlling or fine-tuning the activity of these receptors can be of great benefit. Current means of affecting steroid receptor activity are accompanied by undesirable side-effects. Since the conditions for which these treatments are sought tend to be chronic, there is a critical need for safer drugs that will have manageable side-effects.

Uniqueness or innovativeness of technology: The observation that the lethal factor from Anthrax has a striking effect on the activity of nuclear hormone receptors opens up new routes to controlling their activity. The means of action of this repressor is sufficiently different from known modulators of hormone receptors (*i.e.* the classical antagonists). For instance, the repression of receptor activity is non-competitive, and does not affect hormone binding or DNA binding. Also, the efficacy of nuclear hormone receptor repression by Anthrax lethal factor is sufficiently high that the pharmacological effect of this molecule is seen at vanishingly small concentrations. Taken together, these attributes may satisfy some of the golden rules of drug development such as the uniqueness or novelty of the agent's structure, a low threshold for activity, high level of sophistication and knowledge in the field of enquiry, and the leeway to further refine the molecule by rational means.

Stage of Development: In vitro studies have been completed, and a limited number of animal studies have been carried out.

Methods and Compositions for Production and Purification of Recombinant Staphylococcal Enterotoxin B (rSEB)

Daniel Coffman, Steven Giardina, Jianwei Zhu (NCI).

U.S. Provisional Application No. 60/328,017 filed 09 Oct 2001 (HHS Reference No. E–075–2001/0–US–01);

PCT Application No. PCT/US02/31114 filed 27 Sep 2002 (HHS Reference No. E–075–2001/0–PCT–01);

U.S. Patent Application No. 10/492,105 filed 08 Apr 2004 (HHS Reference No. E–075–2001/0–US–02).

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

This invention claims processes and compositions for fermentation, recovery, and purification of recombinant bacterial superantigens (rSAgs), exemplified by a recombinant staphylococcal enterotoxin B SEB (rSEB) protein mutated for use in administration to a mammalian recipient. This process generates an economically viable quantity of rSEB vaccine protein meeting FDA parenteral drug specifications. The purification methods generally involve multiple steps including hydrophobic interaction chromatography (HIC), buffer exchange (desalting), and cation exchange. The final product of the purification is a highly purified rSAg composition satisfying clinical safety criteria and is immunogenic and protective against lethal aerosol challenge in a murine model. The methods and compositions claimed in the patent application provide possible therapeutics and prophylactics for diseases caused by bacterial SAgs, such as food poisoning, bacterial arthritis and other autoimmune disorders, toxic shock syndrome, and the potential use of SAg biowarfare agents.

Method for Determining Sensitivity to a Bacteriophage

Carl R. Merril (NIMH), Sankar Adhya (NCI), Dean M. Scholl (NIMH).

U.S. Provisional Application No. 60/351,458 filed 23 Jan 2002 (HHS Reference No. E–318–2000/0–US–01);

PCT Application No. PCT/US03/02179 filed 23 Jan 2003 (HHS Reference No. E–318–2000/0–PCT–02);

U.S. Patent Application No. 10/498,428 filed 10 Jun 2004 (HHS Reference No. E–318–2000/0–US–03).

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

Traditionally, chemical antibiotics have been used to treat a variety of bacterial infections. However, bacterial resistance to current antibiotics is an increasingly serious problem in human and veterinary health as well as agriculture. Many experts believe that strains of disease-causing bacteria resistant to all common antibiotics will arise in the next ten to twenty years. Bacteriophages offer a promising therapeutic alternative to antibiotics for these antibiotic resistant bacteria. There are also situations in which bacteriophage may be more suitable than antibiotics to treat infections caused by against antibiotic-sensitive bacteria. Bacteriophages are highly host-specific, thus determining whether a phage would be therapeutically useful against a particular bacterium or strain of bacteria is very important but can be a time-consuming and labor-intensive process.

The current invention claims a method for selecting a therapeutic bacteriophage that would be effective against a particular disease-causing bacteria, comprising a number of bacteriophages containing reporter nucleic acids capable of being expressed

when the bacteriophage infects a bacterial cell. These bacteriophages are separately contacted with a sample contaminated by a bacterium. Expression of the reporter is then detected, indicating which bacteriophage has infected a bacterial cell and is thus a potential therapeutic phage against the particular bacteria. Also claimed in the application are kits allowing for the rapid identification of potentially therapeutic bacteriophages.

Bacteriophage Having Multiple Host Range

Carl Merril (NIMH), Sankar Adhya (NCI), Dean Scholl (NIMH).

U.S. Provisional Application No. 60/220,987 filed 25 Jul 2000 (HHS Reference No. E–257–2000/0–US–01);

PCT Application No. PCT/US01/22390 filed 25 Jul 2001 (HHS Reference No. E–257–2000/0–PCT–02);

U.S. Patent Application No. 10/350,256 filed 21 Jan 2003 (HHS Reference No. E–257–2000/0–US–03).

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

Recently, there has been a renewed interest in the use of phages to treat bacterial infections. The inventors have discovered FK1–5, a highly lytic, non-lysogenic, stable bacteriophage with the ability to kill bacteria rapidly, making it a good candidate for phage therapy. The designation FK1–5 denotes the phage's ability to infect E. coli strains that contain the K1 polysaccharide in their outer capsule as well as E. coli strains that contain the K5 polysaccharide in their outer capsule. Sequence analysis of the tail proteins of phage FK1–5 by the inventors has shown that they are arranged in a cassette structure, suggesting that the host range of phages can be broadened to other K antigens, and even possibly other species of bacteria by recombinant techniques. FK1–5 has a particular advantage because it recognizes and attaches to the structures that confer virulence to bacteria. The inventors' demonstration that a phage can contain multiple tail proteins that expand its host range is useful for generating phage with broad-spectrum antibacterial properties for the treatment of infectious diseases. The inventors have completed in vitro studies on this phage. Furthermore, because of the possibility of engineering the expression of recombinant tail proteins, gene transfer to organisms that are not normally infected by phages is also contemplated by the invention.

CC Chemokine Receptor 5 DNA, New Animal Models and Therapeutic Agents for HIV Infection

C. Combadiere, Y. Feng, E.A. Berger, G. Alkahatib, P.M. Murphy, C.C. Broder, P.E. Kennedy (NIAID).

U.S. Provisional Application No. 60/018,508 filed 28 May 1996 (HHS Reference No. E–090–1996/0–US–01);

U.S. Patent Application No. 08/864,458 filed 28 May 1997 (HHS Reference No. E–090–1996/0–US–04);

U.S. Patent Application No. 10/439,845 filed 15 May 2003 (HHS Reference No. E–090–1996/0–US–05);

U.S. Patent Application No. 10/700,313 filed 31 Oct 2003 (HHS Reference No. E–090–1996/0–US–06);

U.S. Patent Application No. 10/846,185 filed 14 May 2004 (HHS Reference No. E–090–1996/0–US–07);

PCT Application No. PCT/US97/09586 filed 28 May 1997 (HHS Reference No. E–090–1996/0–PCT–02);

European Patent Application No. 97929777.7 filed 28 May 1997 (HHS Reference No. E–090–1996/0–EP–03).

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

Chemokine receptors are expressed by many cells, including lymphoid cells, and function to mediate cell trafficking and localization. CC chemokine receptor 5 (CCR5) is a seven-transmembrane, G protein-coupled receptor (GPCR) which regulates trafficking and effector functions of memory/effector T-lymphocytes, macrophages, and immature dendritic cells. Chemokine binding to CCR5 leads to cellular activation through pertussis toxin-sensitive heterotrimeric G proteins as well as G protein-independent signalling pathways. Like many other GPCR, CCR5 is regulated by agonist-dependent processes which involve G protein coupled receptor kinase (GRK)-dependent phosphorylation, beta-arrestin-mediated desensitization and internalization.

Human CCR5 also functions as the main coreceptor for the fusion and entry of many strains of human immunodeficiency virus (HIV–1, HIV–2). HIV–1 transmission almost invariably involves such CCR5-specific variants (designated R5); individuals lacking functional CCR5 (by virtue of homozygosity for a defective CCR5 allele) are almost completely resistant to HIV–1 infection. Specific blocking of CCR5 (*e.g.* with chemokine ligands, anti-CCR5 antibodies, CCR5-blocking low MW inhibitors, *etc.*) inhibits entry/infection of target cells by R5 HIV strains. Cells expressing CCR5 and CD4 are useful for screening for agents that inhibit HIV by binding to CCR5. Such agents represent potential new approaches to block HIV transmission and to treat infected people. A small animal expressing both human CCR5 along with human CD4 supports entry of HIV into target cells, a necessary hurdle that must be overcome for development of a small animal model (*e.g.* transgenic mouse, rat, rabbit, mink) to study HIV infection and its inhibition.

The invention embodies the CCR5 genetic sequence, cell lines and transgenic mice, the cells of which coexpress human CD4 and CCR5, and which may represent valuable tools for the study of HIV infection and for screening anti-HIV agents. The invention also embodies anti-CCR5 agents that block HIV env-mediated membrane fusion associated with HIV entry into human CD4-positive target cells or between HIV-infected cells and uninfected human CD4-positive target cells.

This technology was reported in Alkhatib *et al.*, ''CC CKR5: a RANTES, MIP–1alpha, MIP–1beta receptor as a fusion cofactor for macrophage-tropic HIV–1,'' Science 272:1955–1958 (1996). The technology is available for exclusive or nonexclusive licensing.

Dated: July 19, 2005.

Steven M. Ferguson,

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

[FR Doc. 05–15347 Filed 8–2–05; 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, DHHS.

ACTION: Notice.

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