for companies and may also be available for licensing.

ADDRESSES: Licensing information and copies of the U.S. patent application 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 application.

High Efficiency Single Stranded Homologous Recombination in Host Cells Deficient for Mismatch Repair

Donald L. Court *et al.* (NCI); PCT Application No. PCT/US03/14657 filed 09 May 2003 (DHHS Reference No. E–038–2003/0-PCT–01); Licensing Contact: Norbert Pontzer; 301/435–5502; *pontzern@mail.nih.gov.*

Homologous recombination is the process of exchanging DNA between two molecules through regions of identical sequence. Homologous recombination provides an alternative to using restriction endonucleases and ligases for producing recombinant DNA. However, the background level of homologous recombination in *E. coli* is very low even with long homology arms. Previous improvements have provided methods of using bacterophage lambda Red recombination functions to greatly increase the recombination frequency of endogenous single- and double-stranded DNA with relatively short homology arms. This type of genetic engineering has been named ''recombineering,'' a convenient term to describe homology-dependent, recombination-mediated, genetic engineering. Recombination with endogenous linear single-stranded DNA (ssDNA) is likely to occur by annealing with transiently single-stranded regions of the chromosome such as the replication fork. We show that only the Beta component of the Red function is required for this activity. (Published PCT Application WO00/21449; Nat. Rev. Genet. 2001, 2:769–779.)

When the ssDNA used for recombineering introduces change(s) near the DNA replication fork, the change(s) may trigger mismatch repair (MMR), which in turn can reduce the level of recombination. In the present invention, altering MMR function achieves a 10-to 100-fold increase in Red recombination. This increase raises the number of recombinants to 25 to 30 percent of treated cells surviving electroporation of the oligo. Methods of transiently inhibiting MMR and bacterial strains deficient for the production of MMR genes are also provided. (Annu. Rev. Genet. 2002, 36:361–88.)

Dated: November 7, 2003.

Steven M. Ferguson,

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

[FR Doc. 03–28657 Filed 11–14–03; 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 invention listed below is owned by an agency of the U.S. Government and is 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.

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Automated Identification of Ileocecal Valve

Ronald Summers (NIHCC), Jianhua Yao (NIHCC), Daniel C. Johnson (Mayo Clinic); U.S. Provisional Application filed 10 Oct 2003 (DHHS Reference No. E–174–2003/0–US–01); Licensing Contact: Michael Shmilovich; 301–435–5019; *shmilovm@mail.nih.gov.*

Available for licensing is a system and software that analyzes digital representations of the colon and eliminates the occurrence of false positive colonic polyps. For example, in a scenario in which a list of polyp candidates is analyzed, the ileocecal valve can be removed from the list. Because the ileocecal valve is a normal structure and not a polyp (*i.e.*, a false positive), removing the ileocecal valve from the list of polyp candidates increases the usefulness and specificity of computer aided polyp detection techniques. Characteristics of a digital representation of at least a portion of a colon can be compared with paradigmatic characteristics of digital representations of ileocecal valves. Based on determining that the digital representation has the characteristics of an ileocecal valve, action can be taken. The digital representation can be removed from a list of polyp candidates or depicted distinctively in a visual depiction. Characteristics can include density, volume, intensity, attenuation, location within the colon, and the like.

Novel Non-Nucleoside Agents for the Inhibition of HIV Reverse Transcriptase for the Treatment of HIV–1

Christopher A. Michejda, Marshall Morningstar, Thomas Roth (NCI); U.S. Patent 6,369,235 issued 09 Apr 2002 (DHHS Reference No. E–076–1997/1–US–01); U.S. Patent Application No. 10/119,634 filed 09 Apr 2002 (DHHS Reference No. E–076–1997/1–US–02); Licensing Contact: Sally Hu; 301–435–5606; *hus@mail.nih.gov.*

Despite recent developments in drug and compound design to combat the human immunodeficiency virus (HIV), there remains a need for a potent, non-toxic compound that is effective against wild type reverse transcriptase (RT) as well as RTs that have undergone mutations and thereby become refractory to commonly used anti-HIV compounds. There are two major classes of RT inhibitors. The first comprises nucleoside analogues, which are not specific for HIV–RT and are incorporated into cellular DNA by host DNA polymerases. Nucleoside analogues can cause serious side effects and have resulted in the emergence of drug resistance viral strains that contain mutations in their RT. The second major class of RT inhibitors comprises non-nucleoside RT inhibitors (NNRTIs) that do not act as DNA chain terminators and are highly specific for HIV–RT. This technology is a novel class of NNRTIs (substituted benzimidazoles) effective in the inhibition of HIV–RT wild type as well as against variant HIV strains resistant to many non-nucleoside inhibitors. These NNRTIs are highly specific for HIV–1 RT and do not inhibit normal cellular polymerases, resulting in lower cytotoxicity and fewer side effects that the nucleoside analogues, such AZT. This novel class of compounds could significantly improve

the treatment of HIV by increasing compliance with therapy.

Dated: November 6, 2003.

Steven M. Ferguson,

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

[FR Doc. 03–28658 Filed 11–14–03; 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.

SUMMARY: The invention listed below is owned by an agency of the U.S. Government and is 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. patents and patent applications listed below may be obtained by contacting Michael Ambrose, Ph.D., at the Office of Technology Transfer, National Institutes of Health, 6011 Executive Boulevard, Suite 325, Rockville, Maryland 20852–3804; telephone: 301/594–6565; fax: 301/402–0220; e-mail: *ambrosem@mail.nih.gov.* A signed Confidential Disclosure Agreement will be required to receive copies of any patent applications.

Mouse Lacking the Chemokine Receptor CX3CR1

Philip Murphy, Christopher Combadiere, Ji-liang Gao (NIAID).

DHHS Reference No. E–216–2003/0—Research Tool.

This mouse has been generated by targeted gene disruption. The mouse provides a model to investigate the function of the chemokine receptor CX3CR1, which is a proinflammatory receptor for the leukocyte chemoattractant CX3CL1 (aka fractalkine). As an example, the mouse is in use in the study of atherosclerosis. Further, the mouse may serve as a model study the role of the immune system during infection with pathogens as well as other immunologically mediated diseases and responses to tumors.

This mouse has been described in the publication ''Decreased atheroscelerotic lesion formation in CX3R1/ApoE double knockout mice''. Combadiere C., Potteaux S., Gao J–L., Esposito B., Casanova S., Lee EJ., Debre P., Tedgui A., Murphy PM., Mallat Z. Circulation. 2003; 1009–1016.

Factors That Bind Intestinal Toxins

Joel Moss (NHLBI), Masatoshi Noda (EM).

U.S. Provisional Application No. 60/409,742 filed 10 Sep 2002 (DHHS Reference No. E–223–2002/0–US–01); PCT Application No. PCT/US03/28282 filed 09 Sep 2003 (DHHS Reference No. E–223–2002/0–PCT–02).

This invention discloses and covers polyphenolic compounds that will bind bacterial toxins, methods for the treatment of such infections, specifically Stx-1 toxins from STEC strains of *E. coli.*

Bacterial infections not only cause disease by their presence but also upon the release of toxins. The common enteric bacteria, *E. coli* O157:H7 releases such toxins (Stx-1) upon treatment with antibiotics. These toxins, when released into the lumen of the intestinal tract, will cause cellular damage thus increasing the severity of the infection. Thus not only does the patient become sick by the infection, but treatment can exacerbate the condition and clinical picture. Further, the indiscriminate use of antibiotics has lead to an increase in the number of resistant strains thus limiting the effectiveness of therapy as well.

The disclosed invention uses an extract from the bracts of *Humulus lupulus* that binds the toxins thus eliminating them as a source of cellular damage. The enclosed methods and devices to isolate such polyphenolic components, the methods to use such components in the detection of such bacteria in biological samples and potential therapies based on the isolated components.

Molecular Diagnosis of Disseminated *Candida albicans* Infection Using Hemoglobin-Response Gene

David D. Roberts, Sizhuang Yan (NCI).

U.S. Patent Application No. 09/258,634 filed 26 Feb 1999 (DHHS Reference No. E–086–1999/0–US–01).

Three hemoglobin-response genes from *Candida albicans* have been isolated. These genes are induced when the organism initiates systemic infections, coming into contact with hemoglobin. Further, the methods and composition of the included nucleic acid sequences and encoded proteins can be used in the development of reagents and kits used to discriminate between commensal colonization and the more life threatening disseminated infection.

Mucosal Cytotoxic T Lymphocyte Responses

Jay A. Berzofsky (NCI), Igor M. Belyakov (NCI), Michael A. Derby (NCI), Brian L. Kelsall (NIAID), Warren Strober (NIAID).

U.S. Patent Application No. 09/508,552 filed 12 Jun 2000 (DHHS Reference No. E–268–1997/2–US–02).

This invention claims methods and compositions for inducing a protective mucosal cytotoxic T lymphocyte (CTL) response in a mammal involving administering a soluble antigen or a soluble antigen with one or more active agents such as a cytokine or co-stimulatory molecule to a mucosal surface or tissue. As a preferred embodiment, the invention contemplates intrarectal administration of the peptide vaccine because the inventors have shown that there is a greater CTL response through intrarectal administration rather than intranasal administration. The synthetic peptide vaccines utilized in the invention to elicit protective immune responses after mucosal infection comprise a multideterminant helper peptide containing a cluster of overlapping helper epitopes (a PCLUS or cluster peptide) colinearly synthesized with a peptide epitope target for neutralizing antibodies and CTL. The inventors have generated data showing that an intrarectally administered synthetic multiepitope HIV/SIV peptide vaccine administered to macaques in conjunction with mutant *E. coli* heat labile enterotoxin as an adjuvant induces mucosal CTL responses that provide better protection against intrarectal SHIV infection when compared to a subcutaneously administered vaccine comprising the same peptides inducing as high or higher systemic CTL responses. The invention is further described in Belyakov *et al., Proc. Natl. Acad. Sci. USA* 1998 Feb 17;95(4):1709–14 and Belyakov *et al., J. Clin. Invest.* 102: 2072–2081, 1998.

Conformationally Locked Nucleoside Analogues

Victor E. Marquez, Juan B. Rodriguez, Marc C. Nicklaus, Joseph J. Barchi, Jr., Maqbool A. Siddiqui (NCI).

U.S. Patent 5,629,454 issued 13 May 1997 (DHHS Reference No. E–231–1993/1–US–01); U.S. Patent 5,869,666 issued 09 Feb 1999 (DHHS Reference No. E–231–1993/1–US–02); and