

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 bacteriophage 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.

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.

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 than the nucleoside analogues, such as AZT. This novel class of compounds could significantly improve