DEPARTMENT OF HEALTH AND HUMAN SERVICES

National Institutes of Health

National Institutes of Health/National Institute of Environmental Health Sciences; Submission for OMB Review; Comment Request; Active Living by Design Program Evaluation

SUMMARY: Under the provisions of Section 3507(a)(1)(D) of the Paperwork Reduction Act of 1995, the National Institute of Environmental Health Sciences (NIEHS), the National Institute of Health (NIH) has submitted to the Office of Management and Budget (OMB) a request to review and approve the information collection listed below. This proposed information collection was previously published in the **Federal Register** on February 14, 2005 (Volume 70, Number 29, Pages 7508–7509, and allowed 60–days for public comment. No public comments were received. The purpose of this notice is to allow an additional 30 days for public comment. The National Institutes of Health may not conduct or sponsor, and the respondent is not required to respond to the collection of information unless it displays a currently valid OMB control number.

Proposed Collection: *Title:* Active Living by Design Program Evaluation. *Type of Information Collection Request:* NEW. *Need and Use of Information Collection:* The purpose of this study is to provide NIEHS with an overall evaluation of the Active Living by Design (ALbD) program to determine the extent to which program strategies to increase physical activity influence change, as measured by increased physical activity and reduction of Body Mass Index (BMI), in residents of participating communities. The objective of this study is to determine the degree to which the changes in the built environment, communication strategies and policy as a result of ALbD's program has impacted physical activity and BMI in residents within the twenty-five (25) participating communities relative to a set of ten (10) control communities.

Two types of data collection will occur throughout the study. A telephone survey, which relies on self-reports, and a clinical survey, which will collect physical activity data using measures of physical activity such as, accelerometers; measures of BMI and an interview on respondents' perceptions of their neighborhood. The findings of this study will provide valuable information concerning (1) The Impact ALbD strategies have on increasing physical activity and bringing about positive changes in health associated with exercise, such as weight loss; and (2) possible reduction of health risks and diseases related to physical inactivity through implementation of ALbd strategies. *Frequency of Response:* Three times over a period of five (5) years, during three rounds of data collection. *Affected Public:* Individuals or households. *Type of Respondents:* Respondents includes adults and children ages 13 through 17 years and their parents. The clinical procedures require respondents under 18 years of age to be accompanied by their parent/guardian, therefore the burden has been doubled for these respondents. The annual reporting burden is respected in the following table:

Type of respondents	Number of respondents	Frequency of response	Average time per response	Annual hour burden
Respondents to Telephone Survey	2,450	1	.334	818.3
Respondents to Clinical Study—Adults	1,855	1	.9185	1,703.8
Respondents to Clinical Study—Children/Parent	595	1	1.837	1,093.0
Total				3,615.1

There are no Capital Costs to report. There are no Operating or Maintenance Costs to report.

Request For Comments: Written comments and/or suggestions from the public and affected agencies should address one or more of the following points: (1) Evaluate 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) evaluate 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) enhance the quality, utility, and clarity of the information to be collected; and (4) minimize the burden of the collection of information on those who are to respond, including the use of appropriated 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: Shobha Srinivasan, Ph.D., Division of Extramural Research and Training, National Institute of Environmental Health Sciences, P.O. Box 12233, MD EC–21, 111 T.W. Alexander Drive, RTP, NC 27709. Phone: (919) 541–2506. Fax: (919) 316–4606. E-mail: *ss688k@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: September 15, 2005.

Richard A. Freed,

NIEHS, Associate Director for Management.

[FR Doc. 05–19175 Filed 9–26–05; 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.

Methods for Introducing Homologous Recombination in a Wide Variety of Bacteria Using Plasmids and Prophage

Donald L. Court (NCI).

U.S. Provisional Application No. 60/573,504 filed 21 May 2004 (HHS Reference No. E–207–2004/0–US–01; U.S. Provisional Application No. 60/653,259 filed 14 Feb 2005 (HHS Reference No. E–207–2004/1–US–01); U.S. Provisional Application No. 60/655,729 filed 22 Feb 2005 (HHS Reference No. E–207–2004/2–US–01); U.S. Patent Application filed 20 May 2005 (HHS Reference No. E–207–2004/3–US–01).

Licensing Contact: Norbert Pontzer; 301/435–5502; *pontzern@mail.nih.gov.*

Homologous recombination is the process of exchanging DNA between two DNA molecules through regions of identical sequence. Homologous recombination provides an alternative to using restriction endonucleases and ligases for producing recombinant DNA. Although the background level of homologous recombination in native *E. coli* is very low even with long homology arms, it is possible to modify or clone nucleic acids using homologous recombination in specific genetically modified strains of *E. coli.* Whereas, a defective prophage used in these recombineering strains is optimally suited for expression of the lambda RED functions for homologous recombination it is hard for experimenters not familiar with *E. coli* genetics to move the defective prophage from strain to strain. Thus, methods of introducing the defective prophage and its recombineering functions into other strains of *E. coli* and other bacteria, including other gram negative bacteria, are also needed.

This invention provides plasmids and methods of use that confer the recombineering function to a variety of cells, including strains like DH10B of *E. coli*, as well as other species like *Salmonella, Pseudomonas, Cyanobacteria, and Yeresinia*, among others. These plasmids can be isolated in vitro and can be used to transform bacterial cells, such as gram negative bacteria.

This research is described, in part, in: Thomason, L.C., Costantino, N., Sawitzke, JA., Datta, S., Bubunenko, M., Court, DL., Myers, R.S., Oppenheim, AB. 2005. Recombineering in Prokaryotes. In Phages: Their Role in Bacterial Pathogenesis and Biotechnology. pp. 383–399. (MK. Waldor, DI. Friedman, and SL. Adhya) ASM Press, Herndon, VA.

Also provided are Lambda phages and methods of use for their introduction as prophages to provide recombineering functions into *E. coli* cells (Virology 319: 185–189, 2004). These phages include appropriate amber mutations in genes to prevent cell death and allow high expression of lambda RED recombination functions. The phage also carry a selectable drug marker used to make lysogens. The phages can be used to infect an *E. coli* cell that includes a suppressor of the amber mutations which allows the phage to reproduce, lyse the infected cell, and produce high titers of the phage. However, the phage will not be able to destroy cells that do not carry the suppressor mutations and in these cells the phage can lysogenise and be used as a defective prophage to generate recombination activity in those cells. Such cells lacking the suppressor are DH10B cells in which genomic libraries of BACs are cloned. Such random libraries can be lysogenized in mass (or individually) with these phages by selecting for the drug marker they carry. These lysogens can then be manipulated for homologous recombination in the same way as BAC containing derivatives off DY380 described elsewhere.

In addition to licensing, the technology is available for further development through collaborative research opportunities with the inventors.

Regulation of INS (3456) P4 Signalling by a Reversible Kinase/Phosphatase and Methods and Compositions Related Thereto

Stephen Shears (NIEHS) *et al.*

U.S. Patent Application No. 10/508,363 filed 16 Sep 2004 (HHS Reference No. E–105–2002/0–US–03), claiming priority to 18 Mar 2002.

Licensing Contact: Marlene Shinn-Astor; 301/435–4426; *shinnm@mail.nih.gov.*

Receptor-dependent changes in Ins (3,4,5,6) P4 levels is a topic of general biological significance, since this regulates the activities of chloride channels that in turn regulate salt and fluid and mucus secretion from epithelial cells, cell volume homeostasis, and electrical excitability in neurons and smooth muscle.

The NIH announces new treatment methods for asthma, bronchitis and cystic fibrosis. The treatments consist of either increasing or decreasing the activity of inositol 1,3,4,5,6 pentakisphosphate 1-phosphatase in a patient, thereby controlling Ins (3,4,5,6) P4-signaling which in turn affects the chloride channels, ultimately regulating salt, fluid and mucus secretion. This modulation of inositol 1,3,4,5,6 pentakisphosphate 1-phosphatase is accomplished by either pharmacological or genetic intervention.

In addition to licensing, the technology is available for further development through collaborative research opportunities with the inventors.

Cancer Therapy Using Vasoactive Intestinal Peptide Antagonists

T. Moody (NCI), D. Brenneman (NICHD), *et al.*

U.S. Patent No. 5,217,953 issued 08 Jun 1993 (HHS Reference No. E–009–1991/0–US–01); U.S. Patent No. 5,565,424 issued 15 Oct 1996 (HHS Reference No. E–009–1991/1–US–01); U.S. Patent No. 6,630,124 issued 07 Oct 2003 (HHS Reference No. E–301–1998/2–US–06); Worldwide IP coverage.

Licensing Contact: Susan Carson; 301/435–5020; *carsonsu@mail.nih.gov*.

The second leading cause of death in the United States is cancer and more than one million Americans are diagnosed with cancer each year, with this number likely to increase as the population ages. There remains a need for effective therapeutics with improved safety profiles, and promising results can be obtained through targeting receptors which are highly expressed on specific cancers. Vasoactive Intestinal Peptide (VIP) is a 28 amino-acid peptide hormone and one of several small neuropeptides that can function as autocrine growth factors. VIP mediates a variety of physiological responses and has been shown to exert stimulating and trophic effects on neoplastic cells inducing its own receptors by feedback mechanisms. Studies have shown that VIP receptors are present in many epithelial cancers including breast, colon, non-small cell lung carcinoma, and pancreatic and prostate cancers. Work by NIH scientists and their collaborators has shown that VIP receptor antagonists such as the lipophilic VIP antagonist SNH inhibit the growth of cancer cell lines in vitro and in vivo and potentiate the cytotoxicity of chemotherapeutic drugs. For example, results have shown that

SNH and taxol are synergistic at inhibiting breast cell cancer growth and can potentiate the cytotoxicity of taxol in an in vivo human xenograft breast cancer mouse model.

Combination therapy using these agents may therefore greatly enhance the response rate of different cancers to these drugs and may significantly reduce side effects by permitting a lower therapeutic dose to be administered. Available for licensing are compositions of matter and methods of use of VIP receptor antagonists.

Dated: September 15, 2005.

Steven M. Ferguson,

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

[FR Doc. 05–19172 Filed 9–26–05; 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, HHS.

ACTION: Notice.

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HIV-Encoded siRNA, microRNA and Suppressor of RNA Silencing

Yamina Bennasser *et al.* (NIAID)
U.S. Provisional Application No. 60/677,839 filed 05 May 2005 (HHS Reference No. E–203–2005/0–US–01).
Licensing Contact: Susan Ano; 301/435–5515; *anos@mail.nih.gov.*

The present invention relates to virus-encoded siRNA and miRNA species and the use of such RNAs in the diagnosis, prevention and/or treatment of retroviral infection, especially HIV or SIV infection. This invention conveys the first evidence that HIV–1 encodes viral siRNA precursors in its genome and that natural HIV–1 infection provokes nucleic acid-based immunity in human cells. To overcome this cellular defense, the HIV–1 Tat protein has evolved to include a suppressor of RNA silencing (SRS) function. Additionally, this invention identifies five microRNA (miRNA) precursor candidates that regulate cellular gene expression at a post-transcriptional level. The five miRNA precursors (21–25 nucleotides in length) are encoded in highly conserved regions of HIV such as TAR sequence, gag, pol and nef genes. These findings indicate that viruses utilize RNA interference as a mechanism to regulate cellular gene expression.

This technology is further described in: Bennasser *et al.*, ''HIV–1 encoded candidate micro-RNAs and their cellular targets,'' Retrovirology 2004 Dec 15, 1(1):43, doi:10.1186/1742–4690–1–43; and Bennasser *et al.*, ''Evidence that HIV–1 encodes an siRNA and a suppressor of RNA silencing,'' Immunity 2005 May, 22(5):607–619, doi:10.1016/j.immuni.2005.03.010.

In addition to licensing, the technology is available for further development through collaborative research opportunities with the inventors.

Miniature Laser-Induced Fluorescence Detector

Paul Smith, Nicole Morgan, Edward Wellner, Terry Phillips (ORS)
U.S. Provisional Application No. 60/682,847 filed 20 May 2005 (HHS Reference No. E–129–2005/0–US–01).
Licensing Contact: Michael Shmilovich; 301/435–5019; *shmilovm@mail.nih.gov.*

Available for licensing and commercial development is a miniature laser-induced fluorescence detector having an in-line microfluidic detection cell. The detection cell finds application in High Performance Liquid Chromatography (HPLC), Capillary Electrophoresis (CE) and Mass Spectroscopy (MS) applications, among others. The cell for fluorescence measurements can have a measurement volume of 1 nL or less and a sample can be excited using two excitation wavelengths. The detection cell can include a 5 mm to 5 cm long capillary tube and an excitation fiber proximate to the capillary tube. A detection fiber is also proximate to the capillary tube, and the detection fiber has a diameter the same size or larger than the external diameter of the capillary tube. A plurality of both excitation and detection fibers can be used.

In addition to licensing, the technology may be available for further development through collaborative research opportunities with the inventors.

Cellular Receptor for Varicella-Zoster Virus and Cell-to-Cell Spread of Virus

Jeffery Cohen *et al.* (NIAID)
U.S. Provisional Application No. 60/684,526 filed 26 May 2005 (HHS Reference No. E–289–2004/0–US–01).
Licensing Contact: Chekesha S. Clingman; 301/435–5018; *clingmac@mail.nih.gov.*

This technology relates to identification of insulin degrading enzyme (IDE) as a cellular receptor for Varicella-Zoster-Virus (VZV), the etiologic agent of varicella (chickenpox) and zoster (shingles). Acute infection of VZV is followed by cell-associated viremia and the development of varicella rash. The virus establishes life-long latency in the nervous system and can reactivate to cause zoster. The mechanism of VZV entry into target cells and spread from cell-to-cell is not well understood. The inventors have shown that antibodies to IDE and recombinant IDE partially inhibit infection with the virus in cell culture. Reducing the level of IDE in the cell (with siRNA), or blocking the ability of IDE to bind with a VZV glycoprotein, markedly diminishes cell-to-cell spread of the virus in cell culture and partially inhibits infection of cells with cell-free virus. This invention further describes molecules that may have a role in the treatment or prevention of VZV infections, including antibodies to IDE, peptides that block IDE–VZV interactions, and other molecules that block binding activity of IDE.

In addition to licensing, the technology is available for further development through collaborative research opportunities with the inventors.

A Novel Amplification Method Permits Pathogens To Be Detected With Microarrays

Michael J. Brownstein, Charles Xiang, and Zhi-Qing Qi (NIMH)
U.S. Provisional Application No. 60/635,239 filed 09 Dec 2004 (DHHS Reference No. E–184–2004/0–US–01).
Licensing Contact: Cristina Thalhammer-Reyero; 301/435–4507; *thalhamc@mail.nih.gov.*