For products regulated by the Center for Biologics Evaluation and Research: Elizabeth Callaghan, Center for Biologics Evaluation and Research (HFM–370), Food and Drug Administration, 1401 Rockville Pike, Rockville, MD 20852–1448, 301–827–3424.

SUPPLEMENTARY INFORMATION:

I. Background

FDA is announcing the availability of a draft guidance for industry entitled ''Bar Code Label Requirements—Questions and Answers.'' Under FDA regulations, certain human drug and biological product labels must have a bar code containing the drug's NDC number (69 FR 9120, February 26, 2004). Bar codes will help reduce the number of medication errors in hospitals and other health care settings by allowing health care professionals to use bar code scanning equipment to verify that the right drug (in the right dose and right route of administration) is being given to the right patient at the right time. This draft guidance is intended to explain certain bar code labeling requirements and their application to human drug and biological products.

This draft guidance is being issued consistent with FDA's good guidance practices regulation (21 CFR 10.115). The draft guidance, when finalized, will represent the agency's current thinking on certain questions and answers on bar code labeling requirements. It does not create or confer any rights for or on any person and does not operate to bind FDA or the public. An alternative approach may be used if such approach satisfies the requirements of the applicable statutes and regulations.

II. Comments

Interested persons may submit to the Division of Dockets Management (see **ADDRESSES**) written or electronic comments regarding this document. Submit a single copy of electronic comments or two paper copies of any mailed comments, except that individuals may submit one paper copy. Comments are to be identified with the docket number found in brackets in the heading of this document. Received comments may be seen in the Division of Dockets Management between 9 a.m. and 4 p.m., Monday through Friday.

III. Electronic Access

Persons with access to the Internet may obtain the document at either *http://www.fda.gov/cder/guidance/index.htm*, *http://www.fda.gov/cber/guidelines.htm*, or *http://www.fda.gov/ohrms/dockets/default.htm*.

Dated: May 27, 2005.

Jeffrey Shuren,

Assistant Commissioner for Policy.

[FR Doc. 05–11266 Filed 6–6–05; 8:45 am]

BILLING CODE 4160–01–S

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 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.

Farnesyltransferase Inhibitors for Treatment of Laminopathies, Cellular Aging and Atherosclerosis

Francis Collins (NHGRI) *et al.*

U.S. Provisional Application No. 60/648,307 filed 28 Jan 2005 (DHHS Reference No. E–055–2005/0–US–01).

Licensing Contact: Fatima Sayyid; 301/435–4521; *sayyidf@mail.nih.gov*.

Hutchinson-Gilford Progeria Syndrome (HGPS) is a very rare progressive childhood disorder characterized by premature aging (progeria). Recently, the gene responsible for HGPS was identified (Eriksson M, Brown WT, Gordon LB, Glynn MW, Singer J, Scott L, *et al.* Recurrent de novo point mutations in lamin A cause Hutchinson-Gilford progeria syndrome. Nature 2003; 423(6937): 293–8), and HGPS joined a group of syndromes—the laminopathies—all of which are caused by various mutations in the lamin A/C gene (LMNA). Lamin A is one of the family of proteins that is modified post-translationally by the addition of a farnesyl group. In progeria, the abnormal protein (progerin) can still be farnesylated, however, a subsequent cleavage is blocked.

The present invention describes a possible treatment of laminopathies, cellular aging and aging-related conditions such as HGPS through the use of farnesyltransferase inhibitors (FTIs) and other related compounds. This treatment should lead to a decrease in the accumulation of abnormal proteins such as progerin in case of HGPS patients and therefore reduce or eliminate many of the devastating clinical symptoms of the underlying biological defect of nuclear membrane instability (Goldman R, Shumaker DK, Erdos MR, Eriksson M, Goldman AE, Gordon LB, Gruenbaum Y, Khuon S, Mendez M, Varga R, Collins FS. Accumulation of mutant lamin A causes progressive changes in nuclear architecture in Hutchinson-Gilford progeria syndrome. Proc Natl Acad Sci U S A 2004; 8963–8968.).

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

Cell Culture System for Efficient Expression of Self-Replicating Norwalk Virus

Kyeong-Ok Chang, Stanislav Sosnovtsev, Gael M. Belliot, Kim Y. Green (NIAID).

U.S. Provisional Application filed 08 Apr 2005 (DHHS Reference No. E–043–2005/0–US–01).

Licensing Contact: Michael Shmilovich; 301/435–5019; *shmilovm@mail.nih.gov*.

Available for licensing and commercial development is a cell culture system for the efficient expression of self-replicating Norwalk virus (NV) RNA (NV replicons). This invention provides compositions and methods for preparing a cell-based system for molecular studies of NV replication and the development of antiviral drugs. A method related to effectively clearing NV replicons, by subjecting cells infected with NV replicon to IFN-alpha is included that demonstrates the applicability of this invention to drug development. A method of effectively clearing NV replicons, by subjecting cells expressing the NV replicon to nucleotide analogues is also provided. These methods provide molecular tools for the identification and development of treatments for NV and may also extend to other members

of the Calicivirus(es) (*e.g., Norovirus, Sapovirus, Lagovirus and Vesivirus*).

Therapeutic Delivery of Nitric Oxide From Novel Diazeniumdiolated Derivatives of Acrylonitrile-based Polymers

Joseph Hrabie, Michael Citro, Frank DeRosa, and Larry Keefer (NCI).

U.S. Provisional Application No. 60/613,257 filed 27 Sep 2004 (DHHS Reference No. E–188–2004/0–US–01).

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

Nucleophile/nitric oxide adduct ions (materials containing the $X–N_2O_2–$ functional group; known as diazeniumdiolates or NONOates) spontaneously dissociate at physiological pH to release nitric oxide (NO) with reproducible half-lives ranging from 2 seconds to 20 hours. The bulk of the known and patented NIH compositions and methods using diazeniumdiolates are derived from amine nucleophiles (*i.e.*, where X–is $R^1R^2N–$). These inventors more recently developed simple and efficient chemical methods to produce diazeniumdiolates by bonding the $N_2O_2–$functional group directly to carbon atoms. Using these methods, the NIH inventors have now produced and tested polymers in which the NO releasing group is attached directly to the carbon backbone of polyacrylonitrile containing polymers.

Available for licensing are compounds, compositions, medical devices, and methods of treatment using acrylonitrile-based polymers that release NO for a week or longer. Polyacrylonitrile itself, co-polymers, admixtures, and products such as cloth and hollow fiber hemofilters have been treated and shown to release NO over time. These polyacrylonitrile-based products could be useful in conjunction with medical devices where the many therapeutic actions of NO would be beneficial. Treatments using stents, extracorporeal blood tubing, shunts, wound dressings and many other devices could be greatly improved by NO actions including but not limited to prevention of clotting, promotion of tissue vascularization, and reduction of excessive tissue proliferation.

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

A New Antiviral Pathway that is Responsible for Viral Clearance: Modulation of ADAR1 Activities Enhance Antiviral Therapies and Virus Infection of Tissue Culture Systems

Deborah R. Taylor *et al.* (FDA).

U.S. Provisional Application No. 60/605,238 filed 27 Aug 2004 (DHHS Reference No. E–121–2004/0–US–01).

Licensing Contact: Robert M. Joynes; 301/594–6565; *joynesr@mail.nih.gov.*

This technology relates to the finding that the antiviral activity of interferon (IFN) is mediated by the activation of an enzyme RNA adenosine deaminase (ADAR1). This enzyme acts by deaminating adenosine residues in dsRNA molecules of the virus into inosine residues. This, in turn, may lead to mutations, genomic instability and ultimately to complete degradation and elimination of the virus. The subject patent application focuses on Hepatitis C virus (HCV), but may be broadly applied to the other viruses.

Based on the above-described finding, the technology offers two important utilities in the medical field:

1. *Antiviral therapeutics:* Because ADAR is so potent as an inhibitor of the growth of HCV, an agonist of this pathway or specifically of ADAR should enhance the clearance of the virus from the cells. Methods to identify such ADAR agonists are described in the subject patent applications.

2. *HCV cell line for drug and vaccine research:* The finding described in the subject patent application may lead to an efficient cell line for growing HCV. Currently, there is not a good system to grow this virus. The addition of ADAR inhibitors (such as RNAi or chemicals that target the catalytic domain of ADAR) to the system will result in a system that can efficiently grow the virus. Such a cell line is important for vaccine development against HCV as well as the development of anti-viral therapeutics.

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

Compositions Comprising T Cell Receptors and Methods of Use Thereof

Richard Morgan (NCI) and Steven Rosenberg (NCI).

PCT Application No. PCT/US2004/029608 filed 13 Sep 2004 (DHHS Reference No. E–106–2004/0–PCT–01).

Licensing Contact: Michelle A. Booden; 301/451–7337; *boodenm@mail.nih.gov.*

Historically, adoptive immunotherapy has shown promise in treating cancer. Traditionally, these adoptive techniques developed to date have relied on isolating and expanding T-cells reactive to a specific tumor associated antigen. However, the approach has been limited by number of isolatable T cells specific to a tumor-associated antigen in a cancer patient's immune system and a very time consuming procedure to isolate and expand the appropriate T-cells.

This invention describes the composition and use of nucleic acid sequences that encode polypeptides capable of forming a T cell receptor (TCR) in a genetically engineered cell. Specifically, these nucleic acid sequences will encode TCR's specific to tumor associated antigens (TAA), gp100, NY–ESO–1, and MART–1. T Cells engineered with these tumor associated antigen specific TCRs show specific immune responses against TAA expressing cancer cells. This observation has a profound effect on the potential efficiency of new adoptive therapies targeted towards cancer.

An adoptive therapy method has been developed using the TAA specific TCR nucleic acids to engineer isolated, non-specific T-cells. This method could eliminate the need to isolate and expand T-cells that may or may not be present in a cancer patient. Clinical trials are currently underway to prove the efficacy of this new adoptive therapy in malignant melanoma.

Details of this invention are published in:

1. Morgan RA, Dudley ME, Yu YY, Zheng Z, Robbins PF, Theoret MR, Wunderlich JR, Hughes MS, Restifo NP, Rosenberg SA. High efficiency TCR gene transfer into primary human lymphocytes affords avid recognition of melanoma tumor antigen glycoprotein 100 and does not alter the recognition of autologous melanoma antigens. J Immunol. 2003 Sep 15;171(6):3287–95.

2. Zhao Y, Zheng Z, Robbins PF, Khong HT, Rosenberg SA, Morgan RA. Primary human lymphocytes transduced with NY–ESO–1 antigen-specific TCR genes recognize and kill diverse human tumor cell lines. J Immunol. 2005 Apr 1;174(7):4415–23.

3. Hughes MS, Yu YY, Dudley ME, Zheng Z, Robbins PF, Li Y, Wunderlich J, Hawley RG, Moayeri M, Rosenberg SA, Morgan RA. Transfer of a TCR Gene Derived from a Patient with a Marked Antitumor Response Conveys Highly Active T-Cell Effector Functions. Hum Gene Ther. 2005 Apr;16(4):457–72.

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

Retrovirus-Like Particles and Retroviral Vaccines

David E. Ott (NCI).

PCT Application filed 27 Oct 2003 (DHHS Reference No. E–236–2003/0–PCT–01).

Licensing Contact: Susan Ano; 301/435–5515; *anos@mail.nih.gov.*

This technology describes retrovirus-like particles and their production from retroviral constructs in which the gene encoding all but seven amino acids of the nucleocapsid (NC) protein was deleted. This deletion functionally eliminates packaging of the genomic RNA, thus resulting in non-infectious retrovirus-like particles. These particles can be used in vaccines or immunogenic compositions. Specific examples using HIV–1 constructs are given. Furthermore, efficient formation of these particles requires inhibition of the protease enzymatic activity, either by mutation to the protease gene in the construct or by protease inhibitor thereby ensuring the production of non-infectious retrovirus-like particles. This technology is further described in Ott *et al.*, Journal of Virology, 2003, 77(5), 5547.

Dated: May 26, 2005.

Steven M. Ferguson,

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

[FR Doc. 05–11221 Filed 6–6–05; 8:45 am]

BILLING CODE 4140–01–P

DEPARTMENT OF HEALTH AND HUMAN SERVICES

National Institutes of Health

National Heart, Lung, and Blood Institute; Notice of Meeting

Pursuant to section 10(d) of the Federal Advisory Committee Act, as amended (5 U.S.C. Appendix 2), notice is hereby given of a meeting of the National Heart, Lung, and Blood Advisory Council.

The meeting will be open to the public as indicated below, with attendance limited to space available. Individuals who plan to attend and need special assistance, such as sign language interpretation or other reasonable accommodations, should notify the Contact Person listed below in advance of the meeting.

The meeting will be closed to the public in accordance with the provisions set forth in secions 552b(c)(4) and 552b(c)(6), title 5 U.S.C., as amended. The grant applications and the discussions could disclose confidential trade secrets or commercial property such as patentable material, and personal information concerning individuals associated with the grant applications, the disclosure of which would constitute a clearly unwarranted invasion of personal privacy.

Name of Committee: National Heart, Lung, and Blood Advisory Council.

Date: June 16, 2005.

Open: 8:30 a.m. to 1 p.m.

Agenda: Discussion of program policies and issues.

Place: National Institutes of Health, Building 31, 31 Center Drive, Bethesda, MD 20892.

Closed: 1 p.m. to 4 p.m.

Agenda: To review and evaluate grant applications.

Place: National Institutes of Health, Building 31, 31 Center Drive, Bethesda, MD 20892.

Contact Person: Deborah P. Beebe, PhD, Director, Division of Extramural Affairs, National Heart, Lung, and Blood Institute, National Institutes of Health, Two Rockledge Center, Room 7100, 6701 Rockledge Drive, Bethesda, MD 20892. 301/435–0260.

This notice is being published less than 15 days prior to the meeting due to the timing limitations imposed by the review and funding cycle.

Any interested person may file written comments with the committee by forwarding the statement to the Contact Person listed on this notice. The statement should include the name, address, telephone number and when applicable, the business or professional affiliation of the interested person.

In the interest of security, NIH has instituted stringent procedures for entrance into the building by non-government employees. Persons without a government I.D. will need to show a photo I.D. and sign-in at the security desk upon entering the building.

Information is also available on the Institute's/Center's home page *http://www.nhlbi.nih.gov/meetings/index.htm*, where an agenda and any additional information for the meeting will be posted when available.

(Catalogue of Federal Domestic Assistance Program Nos. 93.233, National Center for Sleep Disorders Research; 93.837, Heart and Vascular Diseases Research; 93.838, Lung Diseases Research; 93.839, Blood Diseases and Resources Research, National Institutes of Health, HHS.)

Dated: May 31, 2005.

LaVerne Y. Stringfield,

Director, Office of Federal Advisory Committee Policy.

[FR Doc. 05–11218 Filed 6–6–05; 8:45 am]

BILLING CODE 4410–01–M

DEPARTMENT OF HEALTH AND HUMAN SERVICES

National Institutes of Health

National Institute of Neurological Disorders and Stroke; Amended Notice of Meeting

Notice is hereby given of a change in the meeting of the Neurological Sciences and Disorders C, June 14, 2005, 8 a.m. to June 15, 2005, 5 p.m. Wyndham Washington, DC 1400 M Street, NW., Washington, DC 20005 which was published in the **Federal Register** on April 27, 2005, 70 FR Doc: 05–8413.

The meeting will be held for one day on June 14, 2005 from 8 a.m. to 5 p.m. The meeting is closed to the public.

Dated: May 27, 2005.

LaVerne Y. Stringfield,

Director, Office of Federal Advisory Committee Policy.

[FR Doc. 05–11222 Filed 6–6–05; 8:45 am]

BILLING CODE 4140–01–M

DEPARTMENT OF HEALTH AND HUMAN SERVICES

National Institutes of Health

National Institute of Dental & Craniofacial Research; Notice of Closed Meetings

Pursuant to section 10(d) of the Federal Advisory Committee Act, as amended (5 U.S.C. Appendix 2), notice is hereby given of the following meetings.

The meetings will be closed to the public in accordance with the provisions set forth in sections 552b(c)(4) and 552b(c)(6), Title 5 U.S.C., as amended. The grant applications and the discussions could disclose confidential trade secrets or commercial property such as patentable material, and personal information concerning individuals associated with the grant applications, the disclosure of which would constitute a clearly unwarranted invasion of persoanl privacy.

Name of Committee: National Institute of Dental and Craniofacial Research Special Emphasis Panel, 05–84, Review K22.

Date: June 30, 2005.

Time: 2 p.m. to 3 p.m.

Agenda: To review and evaluate grant applications.

Place: National Institutes of Health, Natcher Building, 45 Center Drive, Bethesda, MD 20892, (Telephone Conference Call).

Contact Person: Soheyla Saadi, PhD, Scientific Review Administrator, Intern, Scientific Review Branch, 45 Center Dr. Rm 4AN32A, National Inst of Dental & Craniofacial Research, National Institutes of Health, Bethesda, MD 20892, (301) 594–4805, *saadisoh@nidcr.nih.gov*.

Name of Committee: National Institute of Dental and Craniofacial Research Special Emphasis Panel, 05–79, Review R13s.

Date: July 6, 2005.

Time: 2 p.m. to 4 p.m.

Agenda: To review and evaluate grant applications.

Place: National Institutes of Health, Natcher Building, 45 Center Drive, Bethesda, MD 20892, (Telephone Conference Call).

Contact Person: Mary Kelly, Scientific Review Specialist, National Institute of