adaptive behavior, social participation, health, and quality of life.

This study proposes to continue with the one-time, in-person interview and includes a contemporaneous comparison group of persons who, at age 10 years, were in regular education classes in the same schools as were the persons with developmental disabilities. The data generated from this study will continue to be used to estimate the burden of secondary health conditions, limited social participation, and economic disadvantage among young adults with long-standing, developmental impairments. The total cost to recipients is $0.00.

This request is for a one-year renewal of the currently-approved study.

Data Collection:

Respondents	No. of respondents	No. of responses/ respondent	Avg. burden of response (in hrs.)	Total burden (in hrs.)
Contacting	1,056	1	10/60	176
Interview	898	1	60/60	898
Call Backs	90	1	10/60	15
Total				1,089

Dated: September 10, 1999.

Nancy Cheal,

Acting Associate Director for Policy, Planning and Evaluation, Centers for Disease Control and Prevention (CDC).

[FR Doc. 99–24143 Filed 9–15–99; 8:45 am]

BILLING CODE 4163–18–P

DEPARTMENT OF HEALTH AND HUMAN SERVICES

Health Care Financing Administration

[HCFA–1039–CN2]

RIN 0938–AI87

Medicare Program; Hospice Wage Index; Correction

AGENCY: Health Care Financing Administration (HCFA), HHS.

ACTION: Notice; correction notice.

SUMMARY: This document corrects a typographical error that appeared in the notice published in the **Federal Register** on October 5, 1998, entitled ''Medicare Program; Hospice Wage Index.''

EFFECTIVE DATE: This correction is effective October 1, 1998.

FOR FURTHER INFORMATION CONTACT: Carol Blackford, (410) 786–5909.

SUPPLEMENTARY INFORMATION: On October 5, 1998, we published a notice in the **Federal Register** (63 FR 53446) announcing the annual update to the hospice wage index. The wage index is used to reflect local differences in wage levels. That update was effective October 1, 1998 and is the second year of a 3-year transition period. The provisions in this correction notice are effective as if they had been included in the document published in the **Federal Register** on October 5, 1998.

On November 1, 1998, we published a notice (63 FR 63326) correcting the October 5, 1998 notice. In that correction notice, we inadvertently failed to make one typographical correction. Therefore, in FR Doc. 98–26501 of October 5, 1998, we are now making the following correction:

- On page 53448, in Table A, under the MSA code number 1303 for Burlington, VT, the wage index ''1.1037'' is corrected to read ''1.0137''.

Authority: Section 1814(i) of the Social Security Act (42 U.S.C. 1395f(i)(1)).

(Catalog of Federal Domestic Assistance Program No. 93.773 Medicare—Hospital Insurance Program; and No. 93.774, Medicare—Supplementary Medical Insurance Program)

Dated: September 8, 1999.

Brian P. Burns,

Deputy Assistant, Secretary for Information, Resources Management.

[FR Doc. 99–24096 Filed 9–15–99; 8:45 am]

BILLING CODE 4120–01–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, DHHS.

ACTION: Notice.

SUMMARY: The inventions listed below are owned by agencies 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 contacting John Peter Kim, J.D., M.B.A., Technology Licensing Specialist, at the Office of Technology Transfer, National Institutes of Health, 6011 Executive Boulevard, Suite 325, Rockville, Maryland 20852–3804; telephone: 301/496–7056 ext. 264; fax: 301/402–0220; e-mail: jk141n@nih.gov. A signed Confidential Disclosure Agreement will be required to receive copies of the patent applications.

Oligonucleotides Which Specifically Bind Retroviral Nucleocapsid Proteins

Alan Rein, Jose Casas-Finet, Robert Fisher, Matthew Fivash, Louis E. Henderson (NCI)

Serial No. 09/180,903 filed 12 Jul 1999; PCT/US97/08936 filed 19 May 1997; Serial No. 60/017,128 filed 20 May 1996

The human immunodeficiency virus (HIV) is the causative agent of acquired immunodeficiency syndrome (AIDS). A retroviral protein species, the gag polyprotein, is involved in the assembly of retrovirus particles and capable of specific interactions with nucleic acids. After the virion is released from the cell, the polyprotein is cleaved by the virus-encoded protease. One of the cleaved products, the nucleocapsid (NC) protein, then binds to genomic RNA, forming the ribonucleoprotein core of the mature particle. The interaction between gag and genomic RNA is known to involve the NC domain of the polyprotein. In addition, the NC protein plays crucial roles in both the reverse transcription and integration steps in the viral life cycle.

The present invention relates to retroviral nucleocapsid proteins, such as NC and the gag precursor, and their ability of bind to specific nucleic acid sequences with high affinity. The high affinity of this interaction has potential applications in the design of new antiviral approaches and in sensitive detection of HIV particles. Accordingly, the invention provides for oligonucleotides which bind to

nucleocapsides proteins with high affinity, molecular decoys for retroviral nucelocapsid proteins which inhibit viral replication, targeted molecules comprising high affinity oligonucleotides, assays for selecting test compounds, and related kits.

Human Monoclonal Antibodies to HIV–1 Envelope Glycoprotein gp120

Brynmor A. Watkins and Marvin S. Reitz, Jr. (NCI) Serial No. 60/141,701 filed 30 Jun 1999

The human immunodeficiency virus (HIV) is the causative agent of acquired immunodeficiency syndrome (AIDS). Drug-resistance is a critical factor contributing to the gradual loss of clinical benefit to treatments for HIV infection. Accordingly, combination therapies have further evolved to address the mutating resistance of HIV. However, there has been great concern regarding the apparent growing resistance of HIV strains to current therapies.

The present invention relates to human monoclonal antibodies to type 1 human immunodeficiency virus (HIV–1) envelope glycoprotein gp120, to phage display libraries, and to diagnostic methods and pharmaceutical compositions which employ these antibodies therapeutically and prophylactically.

Antiviral Genetic Target Within HIV gag-pol Transframe Region

Shizuko Sei and Hiroaki Mitsuya (NCI) Serial No. 60/141,072 filed 25 Jun 1999

The human immunodeficiency virus type 1 (HIV–1) is a retrovirus that infects CD4+ T-lymphocytes, causing immunosuppression and the acquired immunodeficiency syndrome (AIDS). The subject invention provides the methods for the potent inhibition of HIV–1 replication, thus effective measure to treat HIV–1 infection, utilizing oligonucleotides and oligonucleotide analogues, including peptide nucleic acids, that can target either DNA or RNA sequences within the HIV gag-pol transframe region. Blocking the expression of the sequences mentioned in the subject invention leads to a decreased and discoordinated synthesis of viral protease, resulting in a significant reduction in the virion production from HIV–1-infected cells.

Identification and Use of High Efficacy Vaccine Antigens Which Modulate Antigen Presenting Cells

Polly Matzinger and John P. Ridge (NIAID)
Serial No. 09/313,487 filed 17 May 1999

Through modulation of the activation state of an antigen presenting cell (APC), the activation of a T cell is concordantly governed, *e.g.*, the activation of a killer T cell. The subject invention accordingly provides uses and applications in the field of immunology for novel pharmaceuticals, therapeutic and prophylactic agents, and vaccine components for the treatment and prevention of cancer, systemic infection, and autoimmune responses.

Thiazepine Inhibitors of HIV–1 Integrase

Yves Pommier, Nouri Neamati, Antonio Garafalo, Vito Nacci (NCI)
Serial No. 60/133,726 filed 12 May 1999

The human immunodeficiency virus (HIV) is the causative agent of acquired immunodeficiency syndrome (AIDS). Drug-resistance is a critical factor contributing to the gradual loss of clinical benefit to treatments for HIV infection. Accordingly, combination therapies have further evolved to address the mutating resistance of HIV. However, there has been great concern regarding the apparent growing resistance of HIV strains to current therapies.

It has been found that a certain class of compounds including thiazepines and analogs and derivatives thereof are effective and selective anti-integrase inhibitors. These compounds have been found to inhibit both viral replication and the activity of purified HIV–1 integrase. The subject invention provides for such compounds and for methods of inhibiting HIV integrase.

Acetylated and Related Analogues of Chicoric Acid as HIV Integrase Inhibitors

Terrence R. Burke, Jr., Zhaiwei Lin, He Zhao, Nouri Neamati, Yves Pommier (NCI)
Serial No. 60/121,127 filed 22 Feb 1999

The human immunodeficiency virus (HIV) is the causative agent of acquired immunodeficiency syndrome (AIDS). Drug-resistance is a critical factor contributing to the gradual loss of clinical benefit to treatments for HIV infection. Accordingly, combination therapies have further evolved to address the mutating resistance of HIV. However, there has been great concern regarding the apparent growing resistance of HIV strains to current therapies.

Chicoric acid has been found to have potential in HIV therapies. The subject invention provides for new chicoric acid analogues and derivatives that inhibit HIV–1 integrase, as well as improved synthetic methods for enantiomers of chicoric acid itself as well as its analogues and derivatives. Also provided are methods for inhibiting the replication of HIV–1 either alone or in combination therapies.

Identification of Globotriaosylceramide as a Promoter of HIV–1 Entry Into Cells

Robert Blumenthal, Anu Puri, Peter Jug (NCI)
Serial No. 60/108,903 filed 17 Nov 1998

The human immunodeficiency virus (HIV) is the causative agent of acquired immunodeficiency syndrome (AIDS). It has been noted that human immunodeficiency virus type 1 (HIV–1) enters permissive cells by binding to the cellular receptor, CD4, and chemokine receptors specific for the envelope glycoprotein (gp120–g41) of a given HIV–1 isolate, followed by gp120–gp41 mediated fusion of the viral and target cell membranes.

The subject invention relates to the discovery of glycosphingolipid cofactors which are essential for entry of a broad range of HIV–1 isolates into cells expressing CD4 and appropriate chemokine receptors. The invention provides for diagnostics, prophylactics, therapeutics, and methods of use for the treatment and prevention of HIV–1 infection and/or AIDS.

Inhibition of Retroviral LTR Promoters by Calcium Response Modifiers

Elise C. Kohn, Kevin Gardner, Lance A. Liotta (NCI)
Serial No. 09/103,519 filed 23 Jun 1998; Serial No. 08/353,765 filed 12 Dec 1994

The human immunodeficiency virus (HIV) LTR is synergistically activated by the phorbol ester 12-myristic 13-acetate (PMA) and T cell specific mitogenic lectin phytohemagglutinin A (PHA). This reflects the activation of the HIV LTR by endogenous T cell mechanisms in vitro. A class of non-voltage-gated calcium influx inhibitor compounds is disclosed which is newly discovered to inhibit the activation of retroviral LTR promoters, including HIV–LTR, by PHA and PMA. This class of compounds can be used to delay or suppress the transition of HIV infection from a latent to a virulent condition, thereby preventing or ameliorating retroviral diseases such as Acquired Immune Deficiency Syndrome (AIDS). The compounds are also useful in cancer treatment, allowing for coordinated therapeutic approaches to retroviral diseases and related cancers such Kaposi's Sarcoma. The compounds can also be used to standardize in vitro assays of commercial importance for clinical and experimental application.

Inhibition of HIV Replication Using Soluble Tat Peptide Analogs

Fatah Kashanchi (NCI), M.R. Sadaie (FDA), John M. Brady (NCI)
Serial No. 09/269,991 filed 02 Oct 1997; PCT/US97/17704 filed 02 Oct 1997; Serial No. 60/027,658 filed 04 Oct 1996

The subject invention embodies the identification of a domain within the transactivator Tat protein of HIV–1, a protein which is necessary for replication of the virus. A number of peptide derivatives of this domain have been constructed. It has been demonstrated that some of these derivatives inhibit Tat transactivation of the human immunodeficiency virus (HIV) LTR (long terminal repeat) promoter. Most importantly, the peptide derivatives also inhibit virus replication and thus provide the basis for potential therapeutic antiviral agents for the treatment of HIV infections.

Dated: September 8, 1999.

Jack Spiegel,

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

[FR Doc. 99–24122 Filed 9–15–99; 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, DHHS.

ACTION: Notice.

SUMMARY: The inventions listed below are owned by agencies 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.

Novel HIV Related Peptides

Giuseppe Scala, Xueni Chen, Oren J. Cohen, Anthony S. Fauci (NIAID)
Serial No. 60/132,760 filed 6 May 1999 (with priority to 11 Jan. 1999)
Licensing Contact: Robert Benson; 301/496–7056 ext. 267; e-mail: rb20manih.gov

This invention concerns novel peptides that selectively react with sera from people who are HIV infected. The peptides were selected by screening random peptide libraries displayed phages with sera from long-term non-progressor (LTNP) subjects followed by counterscreening with non-infected sera. The peptides are potentially useful as vaccines against HIV, and to raise antisera for passive immunization against HIV. In fact, the peptides behaved as antigenic mimics of linear or conformational HIV–1 epitopes generated in vivo in subjects infected with different HIV–1 strains and quasispecies. Moreover, the selected epitopes fulfilled the requirements for an effective immunogen; in fact, the inventors have shown that antisera from immunized mice decrease HIV replication in an in vitro assay. Claimed are the methodology, which allows the identification of pools of HIV-specific peptides by taking advantage of the HIV-specific antibody repertoire induced by the natural infection; peptides, alone or as part of larger vaccine constructs; and antibodies raised against the peptides.

Method of Detecting and Treating Inflammatory Disease

Esther M. Sternberg, Ruth M. Barrientos, Samuel Listwak, Mehrnaz J. Tehrani (NIMH)
Serial No. 60/132,921 filed 6 Apr 1999
Licensing Contact: Kai Chen; 301/496–7735 ext. 247; e-mail: kc169a@nih.gov

A new diagnostic tool for screening for resistance, or susceptibility to certain forms of inflammatory disease (including Alzheimer's, Systemic Lupus Erythematosis, Sarcoidosis, Scleroderma, and Arthritis) was identified using a mutation of the Angiotensin Converting Enzyme (ACE) gene. The mutation in the ACE cDNA was associated with a high level of ACE activity and resistance to exudative inflammation. Related mutations could confer or predict susceptibility to these diseases. Drugs designed to interact with the enzyme, or at the active site near the mutation could be used to treat such illnesses. This could have important implications in the study of human populations with related inflammatory diseases and may be linked to a variety of autoimmune and inflammatory diseases. It is available for immediate licensing, and research collaborations via Cooperative Research and Development Agreements (CRADAs) will be considered.

Nucleic Acid and Amino Acid Sequences of Hemoglobin-Response Genes in Candida albicans and the Use of Reagents Derived From these Sequences in the Diagnosis of Disseminated Candida albicans Infections

David D. Roberts, Sizhuang Yan (NCI)

Serial No. 09/258,634 filed 26 Feb 1999

Licensing Contact: George Keller; 301/496–7735 ext. 246; e-mail: gk40j@nih.gov

Candida albicans is a commensal yeast flora commonly found in the gastrointestinal tract in about 60% of healthy individuals. However, it is also the most common pathogen causing fungal infections in immunocompromised individuals, including AIDS and cancer patients, and organ transplant recipients. Infections caused by Candida albicans range from superficial to deep-seated, and systemic candidiasis is a common complication in immunosuppressed hosts. Invasive infections leading to candidemia in this patient population have high morbidity and mortality. The Centers for Disease Control and Prevention found that candidemia increased tenfold within the past ten years and constitutes the third most common cause of positive blood cultures. Currently, there is no quick diagnostic method to identify candidemia, except the traditional fungal culture. It has been demonstrated that, in the presence of hemoglobin, several new genes are expressed, and hemoglobin induces and facilitates the invasion and colonization of the opportunistic pathogen to hose tissues. The DNA sequences of these new genes could be useful targets to develop molecular diagnostic kits for rapid diagnosis of disseminated candidiasis. Such kits can also be widely used as research tools to define the molecular mechanism of candidemia.

Dated: September 8, 1999.

Jack Spiegel,

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

[FR Doc. 99–24123 Filed 9–15–99; 8:45 am]

BILLING CODE 4140–01–M