Inhibition of HIV Replication Using Soluble Tat Peptide Analogs

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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.

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Jack Spiegel,

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

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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
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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.

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Jack Spiegel,

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

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