

## ANNUAL BURDEN ESTIMATES

Instrument	Number of respondents	Number of responses per respondent	Average burden hours per response	Total burden hours
Request for State data needed to determine the amount of a Tribal Family Assistance Grant .....	15	1	42	630

*Estimated Total Annual Burden Hours: 630.*

In compliance with the requirements of section 3506(c)(2)(A) of the Paperwork Reduction Act of 1995, the Administration for Children and Families is soliciting public comment on the specific aspects of the information collection described above. Copies of the proposed collection of information can be obtained and comments may be forwarded by writing to the Administration for Children and Families, Office of Administration, Office of Information Services, 370 L'Enfant Promenade, SW., Washington, DC 20447, Attn: ACF Reports Clearance Officer. E-mail address: [grjohnson@acf.hhs.gov](mailto:grjohnson@acf.hhs.gov). All requests should be identified by the title of the information collection.

The Department specifically requests comments on: (a) Whether the proposed collection of information is necessary for the proper performance of the functions of the agency, including whether the information shall have practical utility; (b) the accuracy of the agency's estimate of the burden of the proposed collection of information; (c) the quality, utility, and clarity of the information to be collected; and (d) ways to minimize the burden of the collection of information on respondents, including through the use of automated collection techniques or other forms of information technology. Consideration will be given to comments and suggestions submitted within 60 days of this publication

Dated: June 3, 2004.

**Robert Sargis,**

*Reports Clearance Officer.*

[FR Doc. 04-13078 Filed 6-9-04; 8:45 am]

**BILLING CODE 4184-01-M**

## DEPARTMENT OF HEALTH AND HUMAN SERVICES

### National Institutes of Health

#### Office of Loan Repayment and Scholarship; Proposed Collection; Comment Request; National Institutes of Health Undergraduate Scholarship Program for Individuals From Disadvantaged Backgrounds

**SUMMARY:** In compliance with the requirement of section 3506(c)(2)(A) of the Paperwork Reduction Act of 1995, the Office of Loan Repayment and Scholarship, the National Institutes of Health (NIH), has submitted to the Office of Management and Budget (OMB) a request to review and approve the information collection listed below.

*Proposed Collection: Title:* National Institutes of Health Undergraduate Scholarship Program for Individuals from Disadvantaged Backgrounds (UGSP). *Type of Information Collection Request:* Extension of a previously approved collection (OMB No. 0925-0438, expiration date July 31, 2004). *Form Numbers:* NIH 2762-1, NIH 2762-2, NIH 2762-3, NIH 2762-4, and NIH 2762-5. *Need and Use of Information Collection:* The NIH makes available scholarship awards to students from disadvantaged backgrounds who are

committed to careers in biomedical research. The scholarships pay for tuition and reasonable educational and living expenses up to \$20,000 per academic year at an accredited undergraduate institution. In return, for each year of scholarship support, the recipient is obligated to serve as a full-time paid employee in an NIH research laboratory for 10 consecutive weeks during the months of June through August and for 1 year after graduation. If the recipient is enrolled in an undergraduate program or pursues a postgraduate degree (doctoral, medical, dental, or veterinarian school), the post-graduation service obligation may be deferred with the approval of the Secretary of Health and Human Services. The Office of Loan Repayment and Scholarship will use information proposed for collection to determine an applicant's eligibility for participation in the UGSP and a participant's eligibility to defer his or her service obligation. The UGSP is authorized by section 487D of the Public Health Service (PHS) Act (42 U.S.C. 288-2), as amended by the NIH Revitalization Act of 1993 (Public Law 103-43). *Frequency of Response:* Initial application and annual renewal application. *Affected Public:* Applicants (high school or undergraduate students), recommenders, undergraduate institution financial aid staff, participants wishing to defer their service obligation, and graduate or undergraduate registrar staff. The annual reporting burden estimates are as follows:

Type of respondent	Estimated number of respondents	Estimated number of responses per respondent	Average burden hours per response	Estimated total annual burden hours requested
Applicant .....	300	1.0	3.167	950.10
Recommender .....	900	1.0	1.000	900.00
Financial Aid Staff .....	300	1.0	.500	150.00
UGSP Participant .....	40	1.0	.084	3.36
Registrar .....	40	1.0	.750	30.00
Totals .....	1,580	.....	.....	2,033.46

The annualized cost to respondents is estimated at \$40,249.70. There are no capital costs, operating costs, 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) 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) 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) ways to enhance the quality, utility, and clarity of the information to be collected; and (4) ways to minimize the burden of the collection of information on those who are to respond, including the use of appropriate automated, electronic, mechanical, or other technological collection techniques or other forms of information technology.

**FOR FURTHER INFORMATION CONTACT:** To request more information on the proposed project or to obtain a copy of the data collection plans and instruments, contact: Alfred C. Johnson, Ph.D., Deputy Director, Office of Loan Repayment and Scholarship, National Institutes of Health, 2 Center Drive, Room 2E28 (MSC 0230), Bethesda, Maryland 20892-0230. Dr. Johnson may be contacted via e-mail at [ACJohnson@nih.gov](mailto:ACJohnson@nih.gov) or by telephone at 301-402-6425.

**Comments Due Date:** Comments regarding this information collection are best assured of having their full effect if received within 60 days of the date of this publication.

Dated: June 3, 2004.

**Raynard S. Kington,**

*Deputy Director, National Institutes of Health.*  
[FR Doc. 04-13153 Filed 6-9-04; 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, 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.

#### A Peptide That Elicits Neutralizing Antibodies Targeting the HIV Co-Receptor CCR5

Drs. Hana Golding and Surender Khurana (FDA)

U.S. Provisional Application filed 09 Apr 2004 (DHHS Reference No. E-150-2004/0-US-01)

**Licensing Contact:** Sally Hu; 301/435-5606; [hus@mail.nih.gov](mailto:hus@mail.nih.gov).

This invention identifies a peptide sequence that closely mimics the conformational epitope in CCR5, recognized by the HIV neutralizing monoclonal antibody targeting the co-receptor, by using a random peptide phage display library. This peptide upon immunization of rabbits generated antibodies that bind to the HIV-1 co-receptor CCR5 resulting in blocking HIV transmission to target cells, including peripheral blood lymphocytes from human and monkeys. Thus, such antibodies could be directly used for preventing mother to child HIV transmission, for therapy of HIV-1 infected individuals, and may also have particular value when used in combination treatments with other antiviral therapies directed at viral targets, such as protease and reverse transcriptase. The peptide sequence can be used for potential vaccine development. This peptide can also be used for screening of human antibody phage display libraries to isolate human monoclonal with HIV entry-blocking potential. In addition, the peptide and antibodies recognizing it can be used as research tools for increasing the understanding of the mechanisms by which HIV, CCR5 and the HIV receptor, CD4, interact, and in general to understand mechanisms of HIV infectivity.

#### Inhibition of HIV Replication in Resting CD4+ Lymphocytes by Murr1

Gary J. Nabel et al. (NIAID)

U.S. Provisional Application No. 60/523,683 filed 21 Nov 2003 (DHHS Reference No. E-042-2004/0-US-01)

**Licensing Contact:** Susan Ano; 301/435-5515; [anos@mail.nih.gov](mailto:anos@mail.nih.gov).

This technology describes the inhibition of HIV-1 growth in resting CD4+ T cells by Murr1, a highly conserved protein. This finding therefore could be used to prolong the asymptomatic phase of HIV infection.

HIV-1 infects both proliferative and quiescent CD4+ T cells, although the virus replicates poorly in the latter. It has been demonstrated that Murr1 restricts HIV-1 replication by inhibiting basal and cytokine nuclear factor (NF)- $\kappa$ B activity. Short interfering RNAs (siRNAs) experiments that used specific Murr1 siRNAs resulted in lower levels of I $\kappa$ B-A and higher NF- $\kappa$ B activity and HIV-1 replication. These results allude to the potential for a more effective HIV therapeutic that uses Murr1 to regulate viral replication. A Murr1 anti-viral drug that can block viral replication in quiescent lymphocytes and latent cells with provirus might increase the number of patients that remain in the HIV-1 asymptomatic phase and thus lower the number that progress to the AIDS state.

This technology is further described in Ganesh *et al.*, *Nature* (18/25 December 2003), 426(6968): 853-857.

#### Mechanisms for Improving the Breadth of the Immune Response to Diverse Strains and Clades of HIV

Gary J. Nabel et al. (NIAID)

U.S. Provisional Application No. 60/503,509 filed 15 Sep 2003 (DHHS Reference No. E-335-2003/0-US-01)

**Licensing Contact:** Susan Ano; 301/435-5515; [anos@mail.nih.gov](mailto:anos@mail.nih.gov).

This technology describes a multiclade Env vaccine candidate that elicited neutralizing antibodies to a diverse group of primary HIV-1 isolates as compared to antibodies generated from immunization with single clade vaccines. The immunogens of the vaccine included V3 loops from clades A, B, and C and had the cleavage site, fusion peptide, and interhelical regions deleted. Competition studies suggested that the neutralization activity is directed toward shared, conserved epitopes other than the V3 loop. Also described in this technology are immunogens involving deletion of the V3 loop that generated more potent neutralizing antibodies, suggesting that the highly conserved subregions within V3 may be relevant targets to elicit neutralizing antibody responses and increase the immunogenicity of HIV/AIDS vaccines. Such selective deletions in the V3 loop are effective in combination with deletions of other V loops. Immunogens with deletions of the V regions in general (V1-V4), including combinations of deletion immunogens, were also shown to elicit potent neutralizing antibodies. Previous studies of the cell-mediated immune response in mice using the multiclade vaccines of this current technology have shown that they induce Env-specific CD4 and CD8 immune response to