National Institutes of Health

Submission for OMB Review; Comment Request; NIH Undergraduate Scholarship Program for Individuals From Disadvantaged Backgrounds (UGSP)

AGENCY: Under the provisions of Section 3506(c)(2)(A) of the Paperwork Reduction Act of 1995, the Office of Loan Repayment and Scholarship Programs/OIR, the National Institutes of Health 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 March 22, 1996, page 11851, and allowed 60 days for public comment. No public comments were received. The purpose of this notice is the allow an additional 30 days for public comment. The NIH may not conduct or sponsor, and the respondent is not required to respond to, an information collection that has been extended, revised, or implemented on or after October 1, 1995, unless it displays a currently valid OMB control number.

PROPOSED DATA COLLECTION: *Title:* NIH Undergraduate Scholarship Program for Individual from Disadvantaged Backgrounds (UGSP). *Type of Information Request:* New. *Need and Use of Information Collection:* The information collected is needed to assess eligibility and potential of applicants for the UGSP, a program intended to recruit undergraduate students into research field required by the NIH. The program is for students from disadvantaged backgrounds and provides full-time undergraduate students with scholarship of up to $20,000 per year for up to 4 years. As part of the UGSP scholars must enter research service agreements with the NIH for 10 consecutive weeks during the summer and, after graduation, 1 year for each scholarship year. *Frequency of Response:* Annual. *Affected Public:* Individuals; small business entities. *Type of Respondents:* Students applying for the scholarships, educational institutions, and persons recommending the applicants. The annual reporting burden is presented below:

Type of respondent	Estimate Number of respondents	Number of responses per respondent	Average hourly burden	Estimated total annual burden hours requested
Student Applicant	500	1	3.0	1,500
Educational Institution	500	1	0.5	500
Recommenders	1,500	1	0.5	750
Totals	2,500	.....	.....	2,750

The annualized cost to respondents is estimated at: $34,798. 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 are invited on one or more of the following points: (1) Whether the purposed 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 assumption 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.

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 Mr. Marc S. Horowitz, Director, Office of Loan Repayment and Scholarship Programs/OIR/NIH, Rm. 604, 7550 Wisconsin Ave., Bethesda, MD 20892–9121, or call non-toll-free number (301) 402–5666 or e-mail your request, including your address, to <mh8k@nih.gov> or access the Scholarship Office on the Internet at <HTTP://HELIX.NIH.GOV:8001/OSE/CATALOG/LOANREPAY.HTML.>

COMMENTS DUE DATES: Comments regarding this information collection are best assured of having their full effect if received on or before August 26, 1996.

Dated: July 18, 1996.

Ruth L. Kirschstein,

Deputy Director, NIH.

[FR Doc. 96–18967 Filed 7–25–96; 8:45 am]

BILLING CODE 4140–01–M

Government-Owned Inventions; Availability for Licensing

AGENCY: National Institutes of Health, HHS.

ACTION: Notice.

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 U.S. companies and may also be available for licensing.

ADDRESS: Licensing information and copies of the U.S. patent applications and issued patents listed below may be obtained by contacting Cindy K. Fuchs, J.D., at the Office of Technology Transfer, National Institutes of Health, 6011 Executive Boulevard, Suite 325, Rockville, Maryland 20852–3804; telephone: 301/496–7735 ext 232; fax: 301/402–0220. A signed Confidential Disclosure Agreement will be required to receive copies of the patent applications.

Inhibition of Retroviral LTR Promoters by Calcium Response Modifiers

EC Kohn, LA Liotta, KL Gardner (NCI)
Filed 12 Dec 94
Serial No. 08/353,765

The pathogenesis of HIV infection can be divided into two phases based upon the activity of the HIV virus. The latent phase is characterized by low transciptional activity and/or low

replication frequency of the virus whereas the lytic phase is characterized by high transcriptional activity and/or high replication frequency. Although the mechanism(s) involved in the switch from the latent to lytic phase is not completely understood, inhibition of the viral LTR promoter is an important strategy in AIDS treatment. The invention concerns the use of the compound carboxyamidotriazole (CAI), a calcium response modifier, and structurally related compounds that are capable of preventing the activation of the LTR promoter in the treatment of HIV infection and AIDS. In addition, CAI has antimetastatic properties and currently is being tested in clinical trials for the treatment of cancer. A further advantage is that CAI has shown no severe side effects during these trials. Therefore treatment of AIDS patients with CAI would also allow for the treatment of related cancers such as Kaposi's sarcoma. (portfolio: Infectious Diseases—Therapeutics, anti-virals, AIDS)

Diastereoselective Process Leading to a Key Intermediate for the Preparation of Fluorinated Reverse Transcriptase Inhibitors

VE Marquez, JS Driscoll, MA Siddiqui (NCI)
Serial No. 08/189,095 filed 31 Jan 94
U.S. Patent No. 5,498,719 issued 12 Mar 96

A novel process has been developed for synthesizing a key intermediate in the preparation of fluorinated reverse transcriptase inhibitors. Recently, several fluorinated dideoxynucleotides have been found to be effective inhibitors of reverse transcriptase and, thus, offer promise for replacing or augmenting current drugs for the treatment of HIV–1 infection; however, chemically synthesizing these fluorinated dideoxynucleotides is quite expensive, making it economically difficult to produce large-scale amounts for testing. This new process allows the synthesis of a key intermediate in the production of fluorinated dideoxynucleotides at much lower costs because the reaction is diastereoselective, meaning that there are fewer side reactions and more primary product is produced. (portfolio: Infectious Diseases—Therapeutics, anti-virals, AIDS)

C–C Chemokines That Inhibit Retrovirus Infection

P Lusso, R Gallo, F Cocchi, A De Vico, A Garzino-Demo (NCI)
Filed 30 Nov 95
DHHS Reference No. E–008–96/0

This invention concerns three members of the human C-C chemokine family, RANTES, macrophage inflammatory protein 1α (MIP–1α) and macrophage inflammatory protein 1β (MIP–1β), which are produced and secreted by several cell types, including CD8-positive T lymphocytes, and which act *in vitro* as HIV suppressive factors. These factors and their respective genes may be used in the diagnosis, prognosis, treatment and prevention of AIDS and other retrovirus-induced diseases. The invention provides a therapeutic preparation, methods for therapeutic and prophylactic treatment of retroviral infection, and a method of prognosis for retroviral infection. The technology was reported in *BioWorld Today* (6(234):1 (December 7, 1995) and *Science* 270(8):1560–1561 (December 8, 1995). (portfolio: Infectious Diseases—Therapeutics, anti-virals, AIDS; Infectious Diseases—Diagnostics, viral, AIDS)

Dated: July 16, 1996.

Barbara M. McGarey,

Deputy Director, Office of Technology Transfer.

[FR Doc. 96–18969 Filed 7–25–96; 8:45 am]

BILLING CODE 4140–01–M

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AGENCY: National Institutes of Health, HHS.

ACTION: Notice.

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ADDRESSES: Licensing information and copies of the U.S. patent applications listed below may be obtained by contacting Jaconda Wagner, J.D., at the Office of Technology Transfer, National Institutes of Health, 6011 Executive Boulevard, Suite 325, Rockville, Maryland 20852–3804; telephone: 301/496–7735 ext 284; fax: 301/402–0220). A signed Confidential Disclosure Agreement will be required to receive copies of the patent applications.

Methods of Treating Established Colitis Using Antibodies Against IL–12

W Strober, M Neurath, I Fuss (NIAID)
Filed 25 Oct 95
Serial No. 08/547,979

Interleukin-12 (IL–12) is a recently characterized cytokine with unique structure and pleiotropic effects. IL–12 is produced mainly by macrophages/monocytes and can be efficiently induced by intracellular parasites, bacteria and bacterial products. A method for treating the established colitis of an inflammatory bowel disease, including Cohn's disease and ulcerative colitis, by inhibiting the colitis-inducing effects of the cytokine IL–12 has been invented. Additionally, a method for treating their effectiveness in reducing the inflammatory response is also presented. (portfolio: Internal Medicine—Diagnostics, anti-inflammatory; Internal Medicine—Therapeutics, anti-inflammatory; Internal Medicine—Miscellaneous)

Truncated Hepatocyte Growth Factor Variants

AML Chan, JS Rubin, DP Bottaro, SA Aaronson, SJ Stahl,
PT Wingfield, V Cioce (NCI)
Filed 07 Jun 95
Serial No. 08/484,841 (CIP of 08/130,134, which is CIP of 07/655,502)

[HGF/NK2], a truncated form of a hepatocyte growth factor (HGF), may offer an improved method of diagnosing and treating proliferative disorders such as cancers. Elevated levels of HGF are associated with both cancerous and noncancerous conditions. This truncated form of HGF is an antagonist of HGF and can be used to effectively counteract its effects on cells. Its cDNA can also be used as a probe to detect increased levels of HGF mRNA in cells.

HGF/NK1, another truncated form of HGF, has partial agonist/antagonist properties. Thus, it may be useful either as an antagonist of an HGF or as an agonist to reinforce the action of endogenous growth factor, depending on the circumstances. A technique has been developed to produce large quantities of biologically active HGF/NK1 and HGF/NK2 using a prokaryotic expression system. (portfolio: Cancer—Therapeutics, biological response modifiers, growth factors; Cancer—Diagnostics)

IL–13 Receptor Specific Chimeric Proteins and Uses Thereof

R Puri (FDA), W Debinski (Penn State), I Pastan (NCI), N Obiri (FDA)
Filed 15 Mar 95
Serial No. 08/404,685

A chimeric molecule that binds specifically to IL–13 receptors has been identified. The molecule, IL13–PE38QQR, targets tumor cells with less binding to healthy cells in comparison