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. This proposed information collection was previously published in the **Federal Register** on March 26, 1998, page 14721 and allowed 60 days for public comment. No public comments were received. The purpose of this notice is to allow an additional 30 days for public comment. The National Institutes of Health 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 Collection: Title: American Stop Smoking Intervention Study for Cancer Prevention (ASSIST) Final Evaluation: ''Tobacco use Supplement to the 1998–1999 Current Population Survey''. Type of Information Request: OMB #0925–0368, Exp. 3/31/97, REINSTATEMENT, with change. Need and Use of Information Collection: The ''Tobacco use'' supplement to the Current Population Survey conducted by the Bureau of the Census will collect data from the civilian non-institutionalized population on tobacco use and smoking prevalence, smoking intervention dissemination of workplace smoking policies and cessation programs, and changes in smoking norms and attitudes. The data will be used by the National Cancer Institute to evaluate the effectiveness of the American Stop Smoking Intervention Study for Cancer Prevention (ASSIST), a large scale, 17-state demonstration project. This survey will also provide valuable information to Government agencies and to the general public necessary for tobacco control research. The survey will allow state specific estimates to be made. Data will be collected in September 1988, January 1999 and May 1999 from approximately 255,000 respondents. Frequency of Response: One-time study. Affected Public: Individuals or households. Type of Respondents: Persons 15 yrs of age or older. The annual reporting burden is as follows: Estimated Number of Respondents: 170,000; Estimated Number of Responses per Respondent: 1; Average Burden Hours per Response: .1169; and Estimated Total Annual Burden Hours Requested: 19,873. The annualized cost to respondents is estimated at: $198,727. There are no Capital Costs, Operating Costs, and/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) Evaluate 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) Enhance the quality, utility and clarity of the information to be collected; and (4) 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 on 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 Anne Hartman, Statistician, National Cancer Institute, Executive Plaza North, Room 313, Bethesda, Maryland 20892–7344, or call non-toll free number (301) 496–4970, or FAX your request to (301) 435–3710, or E-mail your request, including your address, to *ah42t@nih.gov* or Anne__Hartman@nih.gov.

Comments due date: Comments regarding this information collection are best assured of having their full effect if received on or before July 22, 1998.

Dated June 11, 1998.

Reesa L. Nichols,

NCI Project Clearance Liaison.

[FR Doc. 98–16428 Filed 6–19–98; 8:45 am]

BILLING CODE 4140–01–M

DEPARTMENT OF HEALTH AND HUMAN SERVICES

National Institutes of Health

Licensing Opportunity and/or Cooperative Research and Development Agreement (''CRADA'') Opportunity: Drug and Method To Prevent and Treat Graft-Versus-Host Disease and Allograft Rejection

AGENCY: National Institutes of Health, PHS, DHHS.

ACTION: Notice.

SUMMARY: The NIH is seeking Licensees to further develop, evaluate, and commercialize anti-Tac(Fv)–PE38, also known as LMB2. Anti-Tac(Fv)–PE38 is a recombinant toxin composed of the Fv portion of the anti-Tac antibody which binds to the a subunit of the IL2 receptor (also called P55, Tac, or CD25) fused to PE38 a mutant form of *Pseudomonas* Exotoxin A. Anti-Tac (Fv)–PE38 is very cytotoxic to normal or malignant cells expressing IL2 receptors and is being developed for several proposed applications including (1.) the prevention of Graft-versus Host Disease (''GVHD'') by purging bone marrow of potentially recipient-reactive donor T-cells, (2.) the treatment of Graft-versus Host Disease by i.v. administration, and (3.) the treatment or prevention of allograft rejection. The goal is to move this methodology into clinical trials. The inventions claimed in USPN 4,892,8927, Entitled: ''Recombinant *Pseudomonas* Exotoxins: Construction of an Active Immunotoxin with Low Side Effects''; USSN 07/865,722, Entitled: ''Recombinant Antibody-Toxin Fusion Protein''; USPN 5,696,237, Entitled: ''Recombinant Antibody-Toxin Fusion Protein''; and USSN 08/461,825, Entitled: ''Recombinant Antibody-Toxin Fusion Protein''; are available for either exclusive or nonexclusive licensing for these aforementioned applications (in accordance with 35 U.S.C. 207 and 37 CFR Part 404).

DATES: Respondees interested in licensing the invention(s) will be required to submit an ''Application for License to Public Health Service Inventions'' on or before September 21, 1998 for priority consideration.

Interested CRADA collaborators must submit a confidential proposal summary to the NCI on or before September 21, 1998 for consideration. Guidelines for preparing full CRADA proposals will be communicated shortly thereafter to all respondents with whom initial confidential discussions will have established sufficient mutual interest. CRADA proposals submitted thereafter may be considered if a suitable CRADA Collaborator has not been selected.

ADDRESSES: Questions about licensing opportunities may be addressed to J.R. Dixon, Ph.D., Technology Licensing Specialist, Office of Technology Transfer, National Institutes of Health, 6011 Executive Boulevard, Suite 325, Rockville, Maryland 20852–3804; Telephone: (301) 496–7056 ext. 206; Facsimile: (301) 402–0220; E-Mail: ''DixonJ@OD.NIH.GOV''. Information about Patent Applications and pertinent information not yet publicly described

can be obtained under the terms of a Confidential Disclosure Agreement.

Depending upon the mutual interests of the Licensee(s) and the NCI, a Cooperative Research and Development Agreement (CRADA) to collaborate to improve the properties of the Anti-Tac (Fv)-PE38 may also be negotiated. Proposals and questions about this CRADA opportunity may be addressed to Ms. Karen Maurey, Acting Deputy Director, Technology Development & Commercialization Branch, National Cancer Institute, 6120 Executive Boulevard, Room 450, Rockville, Maryland 20852; Telephone: (301) 496–0477; Facsimile: (301) 402–2117. Respondees interested in submitting a CRADA. Proposal should be aware that it may be necessary to secure a license to the above mentioned patent rights in order to commercialize products arising from a CRADA.

SUPPLEMENTARY INFORMATION: Bone marrow transplantation (''BMT'') is an useful therapy for the treatment of various malignant and nonmalignant genetic and acquired blood disorders which are otherwise incurable. However, a significant limitation of using allogeneic BMT is that only a minority (less than 30%) of patients have an HLA-identical sibling donor. The use of phenotypically matched unrelated donors can only partially overcome this problem, mainly because the time needed to search for an acceptable donor is often too long for patients with advanced disease. Another problem is that ethnic or racial minorities are under-represented in the volunteer bone marrow donor registries. As a result, the chances of finding an unrelated matched donor for such patients is limited.

Graft-versus-Host disease is one of the most frequent complications of allogenic BMT, and is particularly difficult to control in the mismatched setting. Not only does severe GVHD impact greatly on the quality of life of the transplant recipient, as well as contribute significantly to the cost of therapy, but it is the major cause of patient mortality either directly or indirectly (e.g. opportunistic infections due to long-term immunosuppressive therapy).

As has been well documented, GVHD is the result of alloreactive T-cells in the bone marrow graft that are capable of recognizing and attacking the tissues of the immunosuppressed recipient. As it also known, upon recognition and activation by foreign antigen, T-cells express the receptor for interleukine 2 (''LL–2)—which offers a possible method for the removal of alloreactive T-cells. If it were possible to eliminate the presence of contaminating recipient-alloreactive T-cells in the bone marrow graft, thus preventing or reducing the severity of GVHD, allogeneic transplantation might find greater applications and use in the treatment of a variety of other diseases (e.g., autoimmune diseases such as rheumatoid arthritis, etc.). In cases where haploidentical related donors may be readily available to serve as a donor, specific T-cell depletion would permit the haploidentical donor's immunity to be transferred with the graft while preventing severe GVHD, thus improving the overall patient outcome

While GVHD can be prevented by extensive non-selective T-cell depletion of the bone marrow graft, this procedure increases the risk of infection and graft rejections. In HLA genotypically identical sibling transplant, GVHD can be controlled somewhat through the use of immunosuppressive therapy (e.g., Cyclosporin, Methotrexate, etc.). However, such therapeutic modalities are much less effective in the mismatched setting and are associated with susceptibility to bacteria and viral infections, development of new malignancies, and end organ failure.

NIH/NCI scientists at the National Cancer Institute have developed and evaluated in animal models, a recombinant immunotoxin (e.g., Anti-Tac (Fv)/PE38) which kills activated T-cells at very low immunotoxin concentrations. The subject Immunotoxin is a single chain protein composed of the Fv portion of an antibody fused to the amino terminus of the PE. The toxin has three domains: IA is responsible for cell binding, II is required for translocation and has the proteolytic processing site, and III has the ADP-ribosylating activity. After call internalization, a truncated form of PE, generated by proteolytic cleavage translocates to the cytosol where ADP-ribosylation of elongation factor 2 terminates protein synthesis causing cell death.

NIH/NCI scientists have shown that Anti-Tac(Fv)–PE38 may prevent and reduce the severity of GVHD by specific elimination or reduction of recipient-alloreactive donor T-cells without adversely affecting other T-cell population or compromising stem cell engraftment and recipient hematopoietic rescue and survival. These experiments have demonstrated that it is possible to inexpensively and selectively eliminate or reduce the numbers of alloreactive T-cells present in a bone marrow graft resulting in prevention of or a reduction in the severity of GVHD after bone marrow transplantation procedures, but does not compromise stem cell engraftment and recipient hematopoietic rescue and survival. The methodology is simple and does not involve significant lengths of time or specialized equipment. Thus it should be possible to transition these findings to the clinical situation without significant problems. If clinical results approximate the observed animal finding it might then be possible to utilize BMT in many other disease conditions.

In addition NIH/NCI scientists have shown in a Phase I Trial that Anti-Tac(Fv)–PE38 can be safely administered intravenously to patients with cancer; good blood levels of the immunotoxin are also achieved. Thus Anti-Tac(Fv)–PE38 may also be used to treat patients with GVHD or the treat patients undergoing allograft rejection.

A Cooperative Research and Development Agreement or CRADA means the anticipated joint agreement to be entered into by NCI pursuant to the Federal Technology Transfer Act of 1986 and Executive Order 12591 of April 10, 1987 as amended by the National Technology Transfer Advancement Act of 1995 to collaborate to improve the properties of Anti-Tac(Fv)–PE38. The expected duration of the CRADA would be from one (1) to five (5) years.

The role of the NCI in the CRADA may include, but not be limited to:

1. Providing intellectual, scientific, and technical expertise and experience to the research project.

2. Providing the Collaborator with samples of the subject compounds to create, optimize, test and develop targeted drugs for clinical studies.

3. Planning research studies and interpreting research results.

4. Carrying out research to improve the properties of Anti-Tac(Fv)–PE38 which include, but are not restricted to, increased production yield, decreased side effects, increased cyotoxic activity and better tissue penetration.

5. Publishing research results.

The role of the CRADA Collaborator may include, but not be limited to:

1. Providing significant intellectual, scientific, and technical expertise or experience to the research project.

2. Planning research studies and interpreting research results.

3. Providing samples of the subject compounds to create, optimize, test and develop targeted drugs for clinical studies.

4. Providing technical and/or financial support to facilitate scientific goals and for further design of

applications of the technology outlined in the agreement.

5. Providing immunotoxin for laboratory and animal studies.

6. Publishing research results.

Selection criteria for choosing the CRADA Collaborator may include, but not be limited to:

1. The ability to collaborate with NCI on further research and development of this technology. This ability can be demonstrated through experience and expertise in this or related areas of technology indicating the ability to contribute intellectually to ongoing research and development.

2. The demonstration of adequate resources to perform the research and development of this technology (e.g., facilities, personnel and expertise) and accomplish objectives according to an appropriate timetable to be outlined in the CRADA Collaborator's proposal.

3. The willingness to commit best effort and demonstrated resources to the research and development of this technology, as outlined in the CRADA Collaborator's proposal.

4. The demonstration of expertise in the commercial development and production of products related to this area of technology.

5. The level of financial support the CRADA Collaborator will provide for CRADA-related Government activities.

6. The demonstration of expertise pertinent to the development of models to evaluate and improve the efficacy of immunotoxin in the prevention or treatment of graft-versus-host disease and/or allograft rejection.

7. The willingness to cooperate with the National Cancer Institute in the timely publication of research results.

8. The agreement to be bound by the appropriate DHHS regulations relating to human subjects, and all PHS policies relating to the use and care of laboratory animals.

9. The willingness to accept the legal provisions and language of the CRADA with only minor modifications, if any. These provisions govern the distribution of patent rights to CRADA inventions. Generally, the right of ownership are retained by the organization that is the employer of the inventor, with (1) the grant of a license for research and other Government purposes to the Government when the CRADA Collaborator's employee is the sole inventor, or (2) the grant for an option to elect an exclusive or nonexclusive license to the CRADA Collaborator when the Government employee is the sole inventor.

Dated: June 11, 1998.

Kathleeen Sybert,

Acting Director, Technology Development and Commercialization Branch, National Cancer Institute, National Institutes of Health.

Dated: April 30, 1998.

Jack Spiegel,

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

[FR Doc. 98–16427 Filed 6–19–98; 8:45 am]

BILLING CODE 4140–01–M

DEPARTMENT OF HEALTH AND HUMAN SERVICES

National Institutes of Health

Licensing Opportunity and/or Cooperative Research and Development Agreement (''CRADA'') Opportunity: Drug and Method for the Therapeutic Treatment of Ovarian Cancer and Mesotheliomas

AGENCY: National Institutes of Health, PHS, DHHS.

ACTION: Notice.

SUMMARY: The NIH is a seeking Licensee(s) and/or Cooperative Research and Development Agreement (''CRADA'') Collaborators to further develop, evaluate, and commercialize a recombinant immunotoxin, termed SS(dsFv)–PE38. SS(dsFv)–PE38 is a disulfide-linked recombinant immunotoxin fused to PE38, a mutant form of *Pseudomonas* Exotoxin, that binds to mesothelin. Mesothelin is a differentiation antigen present on the surface of most ovarian cancers, mesoltheliomas, and several other types of human cancers including cervical cancer. In normal tissue, mesothelin is limited in its expression to mesothelial cells and basal cells of the trachea (low expression). Therefore, it represents an excellent target for antibody-mediated delivery of cytotoxic agents. The antigen is a 40 kD glycoprotein that is attached to the cell surface by phosphatidylinositol. SS (dsFv)–PE38 immunotoxin is very cytotoxic to cancer cells expressing mesothelin and binds with an affinity of approximately 11 nanomolar. The SS (dsFv)–PE38 immunotoxin also produces complete regressions of mesothelin containing solid tumors growing in nude mice. The goal is to move this drug and methodology into clinical trials. The invention is claimed in USPA SN 08/776,271 and PCT patent application PCT/US97/00224, entitled: ''Mesothelin, A Differentiation Antigen Present on Mesothelium, Mesotheliomas and Ovarian Cancers and Methods and Kits for targeting the Antigen'' and is available for either exclusive or non-exclusive licensing (in accordance with 35 U.S.C. 207 and 37 CFR Part 404).

DATES: Respondees interested in licensing the invention(s) will be required to submit an ''Application for License to Public Health Service Inventions'' on or before September 21, 1998 for priority consideration.

Interested CRADA Collaborators must submit a confidential proposal summary to the NCI on or before September 21, 1998 for consideration. Guidelines for preparing full CRADA proposals will be communicated shortly thereafter to all respondents with whom initial confidential discussions will have established sufficient mutual interest. CRADA proposals submitted thereafter may be considered if a suitable CRADA Collaborator has not been selected.

ADDRESSES: Questions about licensing opportunities may be addressed to J.R. Dixon, Ph.D., Technology Licensing Specialist, Office of Technology Transfer, National Institutes of Health, 6011 Executive Boulevard, Suite 325, Rockville, Maryland 20852–3804; Telephone: (301) 496–7056 ext. 206; Facsimile: (301) 402–0220; E-Mail: ''DixonJOD.NIH.GOV''. Information about Patent Applications and pertinent information not yet publicly described can be obtained under the terms of a Confidential Disclosure Agreement. Respondees interested in licensing the invention(s) will be required to submit an ''Application for License to Public Health Service Inventions''.

Depending upon the mutual interests of the Licensee(s) and the National Cancer Institute (''NCI''), a Cooperative Research and Development Agreement (CRADA) to collaborate to improve the properties of the SS(dsFv)–PE38 immunotoxin may also be negotiated. Proposals and questions about this CRADA opportunity may be addressed to Ms. Karen Maurey, Acting Deputy Director, National Cancer Institute, Technology Development & Commercialization Branch, 6120 Executive Plaza South-Room 450, Rockville, Maryland 20852; Telephone: (301) 496–0477; Facsimile: (301) 402–2117. Respondees interested in submitting a CRADA proposal should be aware that it may be necessary to secure a license to the above mentioned patent rights in order to commercialize products arising from a CRADA.

SUPPLEMENTARY INFORMATION: NIH/NCI scientists have done toxicity studies with the SS(dsFv)-PE38 immunotoxin in mice and with an earlier single chain variant (SSFv-PE38) in Cynomolgus monkeys. Treatment of mice with 5μg