includes whether the acquisition of the nonbanking company complies with the standards in section 4 of the BHC Act (12 U.S.C. 1843). Unless otherwise noted, nonbanking activities will be conducted throughout the United States. Additional information on all bank holding companies may be obtained from the National Information Center website at *www.ffiec.gov/nic/*.

Unless otherwise noted, comments regarding each of these applications must be received at the Reserve Bank indicated or the offices of the Board of Governors not later than September 18, 2005.

A. Federal Reserve Bank of Cleveland (Cindy West, Manager) 1455 East Sixth Street, Cleveland, Ohio 44101-2566:

1. Rurban Financial Corp., Defiance, Ohio; to merge with Exchange Bancshares, Inc., and thereby acquire The Exchange Bank, both of Luckey, Ohio.

Board of Governors of the Federal Reserve System, August 9, 2005.

Robert deV. Frierson,

Deputy Secretary of the Board.

[FR Doc. 05–16089 Filed 8–12–05; 8:45 am]

BILLING CODE 6210–01–S

DEPARTMENT OF HEALTH AND HUMAN SERVICES

National Institutes of Health

Proposed Collection; Comment Request; Field Test of the Discovering the Science of Alcohol Curriculum

Summary: In compliance with the requirement of Section 3506(c)(2)(A) of the Paperwork Reduction Act of 1995, regarding the opportunity for public comment on proposed data collection projects, the National institute on Alcohol Abuse and Alcoholism (NIAAA), the National Institutes of Health (NIH) will publish periodic summaries of proposed projects submitted to the Office of Management and Budget (OMB) for review and approval.

Proposed Collection: Title: Field Test of the Discovering the Science of Alcohol Curriculum. *Type of Information Collection Request:* New. *Need and Use of Information Collection:* The Discovering the Science of Alcohol curriculum (DSA) was developed with a Phase II SBIR grant to bring accurate, research-based information to high school students in biology and science classrooms. The curriculum includes standards-based content objectives and assessment activities. Curriculum materials include a teacher's guide and website. The field test is necessary to estimate the DSA curriculum's effectiveness in conveying information to students and teachers. Specifically, the field study is designed to enable NIAAA to determine whether teachers and students who complete the DSA curriculum demonstrate significantly greater knowledge of the topics covered in the curriculum than teachers and students who do not use the DSA curriculum. In addition, the study is designed to enable NIAAA to determine whether the students who are exposed to the curriculum components self-report different beliefs, attitudes, and intentions regarding alcohol use than their counterparts who are not exposed to the curriculum at their schools.

Participating in this field test will be an experimental group of 30 high school biology classrooms with a total of approximately 400 to 500 students and a control group of 30 high school biology classrooms with approximately 400 to 500 students. Teachers and students from grades 9, 10, 11, and 12 will comprise both groups. The field test will include two surveys: (1) An online, computerized survey that measures teachers' knowledge of the DSA curriculum components and teacher satisfaction with the DSA curriculum components. (2) For students, an anonymous, online, computerized survey that measures three factors: (a) student knowledge of the DSA components, (b) student attitudes, beliefs, and intentions, and (c) student satisfaction with the DSA curriculum components. *Frequency of response:* Once per respondent. *Affected Public:* Individuals. *Type of Respondents:* Biology/Science teachers and high school students.

The reporting burden is as follows: *Estimated Number of Respondents:* It is estimated that we will be able to recruit approximately 60 teachers and approximately 1000 students. *Estimated Number of Responses per Respondent:* One response per respondent. *Average Burden Hours per Response:* 15 minutes per individual in the control group and 30 minutes per individual in the experimental group, for a total respondent burden of 662.5 hours. *Estimated Total Annual Burden Hours Requested:* 662.5 hours. *Estimated Costs to Respondents:* Assuming an hourly rate of $22 for teachers, we estimate the total costs to be $825. There are no Capital Costs to report. There are no Operating or Maintenance costs to report.

Request for Comments: Written comments and suggestions from the public and affected agencies are invited on the following points: (1) Whether the data collection 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; (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 Jason Lazarow, M.Ed., NIH/NIAAA/ORTC/HSEB, 5635 Fishers Lane, Room 3101, MSC 9304, Bethesda, MD 20892–9304, or e-mail your request to: *jlazarow@mail.nih.gov.* Mr. Lazarow can be contacted by telephone at 301–435–8043.

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: August 8, 2005.

Stephen Long,

Executive Officer, NIAAA.

[FR Doc. 05–16139 Filed 8–12–05; 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 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.

Transgenic Mice in which the Gene for MCP–1 is Deleted

Teizo Yoshimura (NCI).
HHS Reference No. E–241–2005/0—Research Tool.
Licensing Contact: Susan S. Rucker; 301/435–4478; *ruckersu@mail.nih.gov*.

Dr. Yoshimura has developed a transgenic mouse which does not express the chemokine MCP–1 due to a deletion of the gene for MCP–1. MCP–1 is a CC chemokine which is responsible for recruiting monocytes into sites of inflammation and cancer. Using a thioglycollate challenge as a measure of the impact of the deletion of MCP–1, MCP–1 deficient mice exhibit a 60% reduction in the number of monocytes/macrophages at 96 hours compared to wild type mice. Although the gene for MCP–1 has been deleted the expression of the neighboring gene for MCP–3 is unaffected. This mouse may be useful as an in vivo model for evaluating the role of MCP–1 in cancer or other diseases associated with inflammation due to the accumulation of monocytes.

This work has not yet been published. These mice are not the subject of any patent or patent application filed by the NIH and are available under a biological materials license.

In addition to licensing, the technology is available for further development through collaborative research opportunities with the inventors.

Monoclonal Antibody to the Protein NCOA6 (Also Called ASC–2, AIB–3)

Paul S. Meltzer (NHGRI).
HHS Reference No. E–168–2005/0—Research Tool.
Licensing Contact: Mojdeh Bahar; 301/435–2950; *baharm@mail.nih.gov*.

The invention relates to monoclonal antibodies that bind to the transcription factor NCOA6 (ASC–2, AIB–3, TRB, TRAP250, NRC). The antibodies have proven successful reagents for Western blotting and for purifying complexes containing NCOA6. The Western blot experiments revealed that NCOA6 is over-expressed in several breast cancer cell lines, and the purification experiments identified a protein complex containing NCOA6 (the ASCOM complex). The monoclonal antibodies may be useful reagents for studying the role of NCOA6 in transcription and for studying the ASCOM complex. Additional information on the antibodies can be found in Goo *et al.* (2003) Mol Cell Biol 23:140–9 and Lee *et al.* (1999) J Biol Chem 274:34283–93.

In addition to licensing, the technology is available for further development through collaborative research opportunities with the inventors.

An Enzymatic Reagent for Removing C-Terminal Polyhistidine Tags From Recombinant Proteins

David S. Waugh (NCI).
HHS Reference No. E–162–2005/0—Research Tool.
Licensing Contact: Mojdeh Bahar; 301/435–2950; *baharm@mail.nih.gov*.

The technology is a new method for removing affinity tags from fusion proteins. Affinity tags are commonly used to purify recombinant proteins, but the tag's influence on the protein is usually unknown. Accordingly, removal of the affinity tag is often desired prior to functional or structural studies.

In contrast to tags added to the amino-terminus (N-terminal tag), removal of tags added to the carboxy-terminus (C-terminal tag) of proteins is problematic. A new carboxypeptidase capable of removing C-terminal tags has been discovered. This enzyme, MeCPA, can remove histidines and other amino acids from the C-terminus of proteins and could be used to remove affinity tags. Because MeCPA will only digest disordered/unstructured residues, it could also be used to remove native amino acids from the C-terminus of proteins to facilitate crystallization.

The inventors have cloned the gene that encodes MeCPA and over-produced the enzyme. A tagged version of MeCPA has been produced to facilitate removal of MeCPA from the products of the cleavage reaction. Background information for this invention is described in Joshi and Leger (1999) JBC 274: 9803–9811.

Induction of C/EBPalpha and Uses Thereof

Robert H. Shoemaker (NCI) *et al.*
U.S. Provisional Application filed 15 Jul 2005 (HHS Reference No. E–140–2005/0–US–01).
Licensing Contact: Michelle A. Booden; 301/451–7337; *boodenm@mail.nih.gov*.

CCAAT/enhancer binding protein alpha (C/EBPalpha) is a leucine-zipper structure transcription factor that plays a key role in regulating the differentiation and proliferation of a variety of cell types. For example, conditional expression of C/EBPalpha is sufficient to trigger neutrophilic differentiation. In addition, administration of antisense molecules against C/EBPalpha has been shown to interfere with proliferation of the late myeloblast and promyelocytic leukemic cell lines HL60 and NB4.

Dominant negative mutations of the *CEBPA* gene have been identified in a large percentage of subjects with t(8;21) acute myeloid leukemia (AML), subtypes M1 and M2. AML is a cancer of the blood and bone marrow characterized by rapid and uncontrolled growth of myelocytes and a lack of myeloid cell differentiation. Approximately 8% of all AML cases are of the t(8;21) variety. Therefore, pharmacologic modulators of C/EBPalpha may be useful as a means to induce cell differentiation, and thus limit proliferation of AML cells.

The present invention describes methods for treating various leukemic disorders by administrating compounds. Additional embodiments describe the mechanism of action of these sterol mesylate compounds through their ability to modulate C/EBPalpha. This disclosure also provides methods for screening for C/EBPalpha inducing compounds. Sterol mesylate compounds and derivatives thereof have the potential to result in more effective therapeutics for the treatment of leukemia and lymphoma.

In addition to licensing, the technology is available for further development through collaborative research opportunities with the inventors.

Use of Discoidin Domain Receptor 1 (DDR1) and Agents That Affect the DDR1/Collagen Pathway

Teizo Yoshimura (NCI).
U.S. Patent Application No. 10/507,385 filed 09 Sep 2004 (HHS Reference No. E–083–2002/2–US–02).
Licensing Contact: Jesse Kindra; 301/435–5559; *kindraj@mail.nih.gov.*

Dendritic cells (DCs) are pivotal antigen-presenting cells for initiation of an immune response. Indeed, dendritic cells provide the basis for the production of an effective immune response to a vaccine, particularly for antigens wherein conventional vaccination is inadequate. DCs are also important in the production on an immune response to tumor antigens.

The present invention discloses methods of using the receptor tyrosine kinase discoidin domain receptor 1 (DDR1) to facilitate the maturation/differentiation of DCs or macrophages. Activating agents of DDR1 may be

useful in the induction of highly potent, mature DCs or highly differentiated macrophages from DC precursors, such as monocytes. Use of this method may enhance the antigen presenting capabilities of the immune system, leading to a more effective overall immune response.

This research is further described in H. Kamohara *et al.*, FASEB J. (October 15, 2001) 10.1096/fj.01–0359fje; and W. Matsuyama *et al.*, FASEB J. (May 8, 2003) 10.1096/fj.02–0320fje.

Methods for Reducing Tumor Growth and Metastasis by Inhibiting MCP–1 Activity

William J. Murphy *et al.* (NCI).

PCT Patent Application No. PCT/US01/16058, filed May 18, 2001 [HHS Ref. No. E–131–2000/0–PCT–02]; Australian Patent Application No. 2001261743, filed May 18, 2001 [HHS Ref. No. E–131–2000/0–AU–03]; Canadian Patent Application No. 2409298, filed May 18, 2001 [HHS Ref. No. E–131–2000/0–CA–04]; European Patent Application No. 01935670.8–24, filed May 18, 2001 [HHS Ref. No. E–131–2000/0–EP–05]; and U.S. Patent Application No. 10/276,644, filed March 10, 2003 [HHS Ref. No. E–131–2000/0–US–06].

Licensing Contact: Jesse S. Kindra; 301/435–5559; *kindraj@mail.nih.gov*.

Monocyte Chemotactic Protein 1 (MCP–1) is a chemokine that is abundantly produced in a variety of inflammatory diseases. Consistent with its role in inflammation, MCP–1 is known to be chemotactic for monocytes, T lymphocytes, basophiles and NK cells.

Based on its chemotactic effect on monocytes, MCP–1 has been observed to have an anti-tumor effect in certain mouse/tumor experimental designs. In those mouse systems, MCP–1 production by tumor cells was positively correlated with the number of intratumoral macrophages and inversely correlated with tumor growth. These studies have led to the hypothesis that MCP–1 possesses anti-tumorigenic activity.

The present invention is based on the surprising discovery that inhibition of MCP–1 activity inhibits tumor metastasis and prolongs survival. Accordingly, this invention generally relates to methods of inhibiting tumor growth and/or metastasis in a subject, and methods of treating cancer and/or increasing survival of a subject with a tumor, by inhibiting MCP–1 activity in the subject.

In addition to licensing, the technology is available for further development through collaborative research opportunities with the inventors.

Novel DNA Liposome Complexes for Increased Systemic Delivery and Gene Expression

Nancy Smyth-Templeton and George N. Pavlakis (NCI).

U.S. Patent No. 6,413,544 issued 02 Jul 2002 (HHS Reference No. E–143–1996/0–US–03); U.S. Patent No. 6,770,291 issued 03 Aug 2004 (HHS Reference No. E–143–1996/0–US–04); U.S. Patent Application No. 10/825,803 filed 15 Apr 2004 (HHS Reference No. E–143–1996/0–US–16).

Licensing Contact: John Stansberry; 301/435–5236; *stansbej@mail.nih.gov*.

Improved liposomes have been created that could increase the efficacy of treatments for cancer, cardiovascular diseases, and HIV–1 related diseases in small and large animal models. These liposomes efficiently condense nucleic acids, proteins, viruses, drugs, and mixtures of these agents on the interior of bilamellar invaginated structures produced by a novel extrusion procedure. This technology is an improved delivery system for all biologically active reagents. By using extruded DOTAP:Cholesterol liposomes to form complexes with DNA encoding specific proteins, expression has been improved dramatically. These nucleic acid:liposome complexes have extended half-life in the circulation, are stable in serum, have broad biodistribution, efficiently encapsulate various sizes of nucleic acids and other molecules including viruses and drugs, are targetable to specific organs and cell types, penetrate through tight barriers in several organs, are fusogenic with cell membranes and avoid endosomes, are optimized for nucleic acid:lipid ratio and colloidal suspension in vivo, can be size fractionated to produce a totally homogenous population of complexes prior to injection; are non-toxic, non-immunogenic and can be repeatedly administered, and liquid suspensions and freeze-dried formulations are stable. These complexes have been injected into mice, rats, rabbits, pigs, nonhuman primates, and humans. Currently, these complexes are injected intravenously into patients in clinical trials to treat lung cancer and will be used in upcoming trials to treat breast, pancreatic, head and neck cancers; and Hepatitis B and C.

In addition to licensing, the technology is available for further development through collaborative research opportunities with the inventors.

Methods of Delivering Agents to Target Cells

Andrew J. George *et al.* (NCI).

U.S. Patent No. 5,861,156 issued 19 Jan 1999 (HHS Reference No. E–130–1993/0–US–01).

Licensing Contact: George G. Pipia; 301/435–5560; *pipiag@mail.nih.gov*.

The present invention relates to methods of delivering agents to target cells. The target cells are modified by one or more monospecific binding proteins reactive with one or more consistent naturally occurring target cell surface markers. The monospecific binding protein reactive with the cell surface marker is tagged, fused to, or labeled with a chemical moiety which is recognized by, and binds to a site on a multivalent antibody, which also binds an agent to be delivered. The agent is bound to the multivalent antibody, which in turn, is also bound to a tagged monospecific binding protein which is bound to a cell surface marker on a target cell. Thus, the agent is delivered, or directed, to the target cells.

Chemical moiety, as used herein, includes a genetically fused or otherwise coupled peptide, one or more peptides within the sequence of a mono- or bispecific binding protein, a posttranslationally or chemically modified peptide, a chemical substituent such as biotin, incorporated into the protein, or any non-natural amino acid incorporated into the binding protein. Chemical moiety also includes any protein or parts thereof, or peptide comprising an amino acid sequence that is reactive with a recognition site, including a linker connecting variable regions of a single-chain Fv (sFv) or sFv fusion protein, or an epitope of the monospecific binding protein.

The present invention further relates to a method of immunotherapy in a host whereby target cells are destroyed with enhanced selectivity using target cell-directed cytotoxic agents. This method of immunotherapy involves two concepts: the specific modification of the target cell with chemical moiety-labeled monospecific binding proteins and the targeting of cytotoxic agents to the modified target cells.

Dated: August 5, 2005.

Steven M. Ferguson,

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

[FR Doc. 05–16136 Filed 8–12–05; 8:45 am]

BILLING CODE 4140–01–P