of employment before, during and after their program of study. The study will examine various measures associated with the career paths chosen by these graduates and by comparing these measures within and between the two groups of graduates. Comparisons of employment sites of graduates in schools receiving the preference with those of graduates in schools not receiving the preference will indicate the significance of funding preference in promoting program objectives of increasing access to care in underserved communities. Information on both the nursing-specialty of graduates and their current employment setting will be analyzed for each of the two groups.

The estimated burden is as follows:

Form	Number of respondents	Responses per respondent	Hours per response	Total burden hours
Nurses	4000	1	20 (in minutes)	1320
Ineligible nurses	37	1	5 (in minutes)	4
Total	4037			1324

Written comments and recommendations concerning the proposed information collection should be sent within 30 days of this notice to: Wendy A. Taylor, Human Resources and Housing Branch, Office of Management and Budget, New Executive Office Building, Room 10235, Washington, DC 20503.

Dated: November 10, 1999.

Jane Harrison,

Director, Division of Policy Review and Coordination.

[FR Doc. 99–30082 Filed 11–17–99; 8:45 am]

BILLING CODE 4160–15–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 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.

Peptide Inhibitor of Cyclin Dependent Kinase 4 (cdk4) Derived from MyoD

BM Paterson, J Zhang (NCI).
Serial No. 60/139,934 filed 18 Jun 1999.
Licensing Contact: Susan S. Ricker; 301/496–7056 ext. 245; e-mail: sr156v@nih.gov.

This invention pertains to cell cycle regulation and the activity of the GI cyclin-dependent kinase 4 (CDK4). The invention describes a 15 amino acid peptide and variants thereof derived from MyoD, which is an inhibitor of the CDK4. CDK4 is one of a number of cyclin-dependent kinases which control progression through the cell cycle through their ability to phosphorylate particular substrates at the correct phase of the cell cycle. CDK4 has been shown to be involved in cell cycle control through its ability to regulate the activity of the retinoblastoma protein, pRb, an activator of genes essential for cell division.

Inhibitors of the cyclin-dependent kinases (CKIs), such as the peptides described in this invention, prevent cell cycle progression and induce cells to exit the cell cycle into the Go state. The peptides described in this invention prevent the phosphorylation of pRb by cdk4, an obligate step for entry into the cell cycle. Osteosarcomas and habdosarcomas are two types of tumors known to over-express pRb. The inhibitor described in this invention may be useful in treating these cancers or other diseases which have been specifically linked to over-expression of active pRb.

Background material related to this invention has been published [Zhang. J. *et al.* EMBO J 18(4): 926–33 (Feb. 15, 1999)].

Chromatographic Separation of Proteins by Ammonium Sulfate Precipitation

Yoichiro Ito (NHLBI)
Serial No. 09/263,609 filed 05 Mar. 99
Licensing Contact: John Fahner-Vihtelic; 301/496–7735 ext. 270; e-mail: jf36z@nih.gov

Recently, a field flow fractionation apparatus and method for the chromatographic separation of proteins have been developed. Unique in design, the fractionation apparatus contains two spiral channels, a reagent channel and a sample channel carved into two mating disks separated by a semi-permeable membrane. The primary advantage to this design is that it allows proteins passing through the sample channel to be fractioned according to their ability to precipitate out in the presence of an exponential ammonium sulfate concentration gradient in the reagent channel. Protein elution is achieved by repetitive precipitation, and takes place along the sample channel without the tedious manual labor required by conventional fractionation procedures. This method can also utilize other precipitation reagents such as NaCl, ethanol and polyethylene glycols. Applications would include purification of monoclonal antibodies (IgM and IgG) from a culture medium and ascitic fluid and affinity separation of recombinant enzymes from E. coli lysate. A working prototype is undergoing additional refinement.

Calcium Channel Compositions and Methods of Use Thereof

Michael I. Lerman *et al.* (NCI)
Serial No. 60/114,359 filed 30 Dec 1998
Licensing Contact: Susan S. Rucker; 301/496–7056 ext. 245; e-mail sr156v@nih.gov

The invention described in this patent application relates to the identification, isolation and cloning of a three cDNAs identified during a search of the short arm of chromosome 3 for a tumor suppressor gene (TSG) associated with lung, breast and other cancers. The cDNAs are alternate isoforms which encode a protein which functions as a

L-type voltage-dependent calcium channel. Type L-voltage dependent calcium channels represent one of five families of calcium channels, L, R, P, N, Q, which have been identified. Type L voltage-dependent calcium channels are found in a wide variety of tissues including the brain, muscle and the endocrine system.

The gene has been mapped to the short arm of chromosome 3 at 3p21.3. The gene, which corresponds to this cDNAs is an alpha2delta ($\alpha 2\delta$) subunit, and has been shown to be deleted in lung and breast cancer. The scientists have demonstrated that the expression of this calcium channel has been shut off in lung cancer cells and hypothesize that this may lead to a malignant phenotype. Possible applications of this technology include its use in drug screening assays; its use as an early diagnostic marker and/or as a prognostic or treatment indicator; its use in gene therapy where defective cells would be reconstructed with the gene and as a therapeutic agent for clearing autoantibodies which develop toward the alpha2delta subunit in the disease Lambert-Eton myasthenia syndrome.

Hepatitus C Virus (HCV) Envelope Protein Modified for Expression on the Host Cell Surface and Use of DNA Constructs Encoding the Modified Protein as a Vaccine and of Host Cells Expressing the Protein in Diagnostic and Screening Assays

Xavier Forns, Suzanne U. Emerson, Jens Bukh, Robert H. Purcell (NIAID)
Serial No. 60/089,779 filed 18 Jun 1998
Licensing Contact: J. Peter Kim; 301/496–7056 ext. 264;
e-mail: jk41n@nih.gov

Hepatitis C virus (HCV) is a single stranded RNA virus responsible for the majority of non-A non-B hepatitis. Hepatitis C virus (HCV) has a worldwide distribution and is a major cause of liver cirrhosis and hepatocellular carcinoma in the U.S., Europe, and Japan. For this reason, development of a vaccine against hepatitis C is of great importance.

The present invention provides for hepatitis C virus (HCV) vaccines and diagnostic assays. The invention provides chimeric genes, expression vectors which comprise these chimeric genes, and DNA based vaccines which employ the expression vectors as immunogens to produce protective antibodies to HCV in a mammal. The invention further provides for diagnostic assays to screen sera for the presence of antibodies to HCV envelope proteins, as antigens in the screening of phage display combinatorial libraries, and as reagents to develop tissue culture systems suitable for testing anti-HCV envelope antibodies for neutralizing activity.

Human FRP and Fragments Thereof Including Methods for Using Them

US Rubin (NCI), PW Finch, SA Aaronson, and X He
Serial No. 09/087, 031 field 29 May 1998
Licensing Contact: Susan S. Rucker; 301/496–7056 ext 245; e-mail: sr156v@nih.gov

This application relates to signal transduction pathways and mechanisms. More particularly, the application describes the isolation, cloning of the cDNA encoding, and characterization of a human protein denoted ''Frizzled Related Protein'' or FRP. FRP, also known as sFRP–1, is a secreted protein which contains an N-terminal cysteine-rich domain (CRD) which is a similar to the CRDs of the frizzled family of membrane anchored Wnt receptors. sFRP–1 lacks any transmembrane region or cytoplasmic domain characteristic of molecules capable of transducing a signal within a cell but is preferentially distributed to the cell surface or matrix.

Wnt signaling has been implicated in the development of cancers and various organs. sFRP–1 has been demonstrated to antagonize Wnt signaling and therefore may function as an inhibitor of Wnt activity or otherwise modulate Wnt signaling. In addition, others have suggested that sFRP–1 plays a role in regulating apoptosis by sensitizing cells to apoptotic agents and modulating levels of β-catenin. The gene encoding sFRP–1 is found on the short arm of chromosome 8 at 8p11.1–12. RNA transcripts have been identified in multiple adult tissues such as the heart, kidney, ovary, prostate, testis, small intestine and colon but have not been detected in a number of other tissues.

In view of this sFRP–1 derived products may be useful in further study of sFRP–1—Wnt interactions, drug screening assays, the development of diagnostics for cancer or other conditions which are related to Wnt signaling, or may be developed as therapeutic agents themselves. Recombinant FRP, expression vectors containing FRP cDNA and cDNA containing the full length FRP coding sequence are available. Limited quantities of rabbit polyclonal antisera which specifically binds FRP is also available.

This work has appeared, in part, in Finch, PW, *et al.* PNAS 94(13): 6770–75 (June 24, 1997) and has been published as WO 98/54325 (Dec. 3, 1998).

Use of Lipoxygenase Inhibitors as Anti-Cancer Therapeutic and Intervention Agents

James L. Mulshine, Marti Jett (NCI)
Serial No. 08/704,569 filed 03 Dec 96
Licensing Contact: Girish Barua; 301/496–7056 ext. 263; email gb18t@nih.gov

We have reported that S-Lipoxygenase inhibitors can treat or prevent certain epithelial cancers such as lung cancer, breast cancer, and head and neck cancer. This is believed to occur from the interruption of the 5-lipoxygenase pathway which results in increased tumor cell apoptosis. We have demonstrated this effect for the growth factor-induced stimulation in several model systems so we propose this as a robust anti-promotional chemoprevention approach as well.

Suitable 5-lipoxygenase inhibitors useful for the methods of the present invention include 2-(12-Hydroxydodeca-5, 10-dinyl) 3,5,6-trimethyl-1,4benzoquinone and derivatives thereof; Nordihydroguiaretic acid and derivatives and 3-[1-(4-chlorobenzy)-3-t-butylthio-t-isopropyl-indol-2-yl] -2, 2-dimethylpropionic acid and derivatives thereof. Also intended to be encompassed by this are hydroxyurea derivatives as inhibitors of 5-lipoxygenase for use in the prevention and treatment of the cancers mentioned above.

Dated: November 9, 1999.

Jack Spiegel,

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

[FR Doc. 99–30065 Filed 11–17–99; 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, HHS.

ACTION: Notice.

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