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.

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.

Methods of Inhibiting Cancer Cells With ADNF III Antisense Oligonucleotides

I Gozes, R Zamostiano, E Gelber, A Pinhasov, M Bassan (all of Tel Aviv University), DE Brenneman (NICHD)
Serial No.: 09/364,609 filed 30 Jul 1999.
Licensing Contact: Susan S. Rucker; 301/496–7056 ext. 245; e-mail: sr156v@nih.gov.

This application describes methods of inhibiting the proliferation of cells using an antisense oligonucleotide derived from the polypeptide Activity Dependent Neurotrophic Factor III (ADNF III)/Activity Dependent Neuroprotective Protein (ADNP). Preferred antisense oligonucleotides are complementary to the 5′ region of ADNF III/ADNP. The ability of such antisense oligonucleotides to inhibit cell proliferation has been demonstrated in *in vitro* models such as the HT29 colon cancer cell line. Based on the location of ADNF III/ADNP on chromosome 20 at 20q13, a region which has been shown via CGH to be associated with breast, ovary, colon, head and neck, brain and pancreatic cancers, ADNF III/ADNP antisense molecules might also be expected to be useful in treating one or more of these cancers.

Orally Active Peptides That Prevent Cell Damage and Death

DE Brenneman, CY Spong (both of NICHD), I Gozes, A Pinhasov, E Giladi (all of Tel Aviv University)
Serial No.: 60/149,956 filed 18 Aug. 1999.
Licensing Contact: Susan S. Rucker; 301/496–7056 ext. 245; e-mail: sr156v@nih.gov.

This application describes two peptides which are orally active and which have been shown in *in vitro* assays to protect against neuronal cell death. In animal model systems for Alzheimer's disease and Fetal Alcohol Syndrome the peptide have also been demonstrated to be useful. The first peptide is D–SAL, a D-isomer of the peptide SAL (SALLRSIPA) derived from Activity Dependent Neurotrophic Factor I (ADNF I). The second peptide is D–NAP, a D-isomer of the peptide NAP (NAPVSIPQ) derived from a related protein Activity Dependent Neuroprotective Protein (ADNP)/Activity Dependent Neurotrophic Factor III (ADNF III). The peptides may be used alone or in combination. The peptides may be constructed solely of D-isomers of their amino acids or combinations of D and L amino acids. Other diseases involving neuronal cell death where D–SAL or D–NAP may be useful include Huntington's disease, epilepsy, Parkinson's disease and Tourette's syndrome.

Dated: November 9, 1999.

Jack Spiegel,

Director, Division of Technology Development and Transfer, Office of Technology Transfer.

[FR Doc. 99–30067 Filed 11–17–99; 8:45 am]

BILLING CODE 4140–01–P

DEPARTMENT OF HEALTH AND HUMAN SERVICES

National Institutes of Health

Notice of Meeting of Recombinant DNA Advisory Committee

Pursuant to section 10(a) of the Federal Advisory Committee Act, as amended (5 U.S.C. Appendix 2), notice is hereby given of a meeting of the Recombinant DNA Advisory Committee.

The meeting will be open to the public, with attendance limited to space available. Individuals who plan to attend and need special assistance, such as sign language interpretation or other reasonable accommodations, should notify the Contact Person listed below in advance of the meeting.

Name of Committee: Recombinant DNA Advisory Committee.

Date: December 8–10, 1999.

Time: December 8, 1999, 9 a.m. to 5 p.m.

Agenda: The Committee will discuss Proposed Actions under the NIH Guidelines for Research Involving Recombinant DNA Molecules (59 FR 34496) and other matters to be considered by the Committee. The Proposed Actions to be discussed will follow this notice of meeting.

Place: National Institutes of Health, 9000 Rockville Pike, Building 31, Conference Room 6, Bethesda, MD 20892.

Time: December 9, 1999, 8:30 a.m. to 5:00 p.m.

Agenda: See paragraph above for Agenda.

Place: National Institutes of Health, Building 31, Conference Room 10, Bethesda, MD 20892.

Time: December 10, 1999, 8:30 a.m. to 5:00 p.m.

Agenda: See paragraph above for Agenda.

Place: National Institutes of Health, Building 31, Conference Room 10, Bethesda, MD 20892.

Contact Person: Debra W. Knorr, Deputy Director, Office of Biotechnology Activities, NIH, MSC 7010, 6000 Executive Boulevard, Suite 302, Bethesda, MD 20892–7010.

OMB's ''Mandatory Information Requirements for Federal Assistance Program Announcements'' (45 FR 39592, June 11, 1980) requires a statement concerning the official government programs contained in the Catalog of Federal Domestic Assistance. Normally NIH lists in its announcements the number and title of affected individual programs for the guidance of the public. Because the guidance in this notice covers virtually every NIH and Federal research program in which DNA recombinant molecule techniques could be used, it has been determined not to be cost effective or in the public interest to attempt to list these programs. Such a list would likely require several additional pages. In addition, NIH could not be certain that every Federal program would be included as many Federal agencies, as well as private organizations, both national and international, have elected to follow the NIH Guidelines. In lieu of the individual program listing, NIH invites readers to direct questions to the information address above about whether individual programs listed in the Catalog of Federal Domestic Assistance are affected.

Dated: November 1, 1999.

Anna Snouffer,

Acting Director, Office of Federal Advisory Committee Policy.

[FR Doc. 99–30066 Filed 11–17–99; 8:45 am]

BILLING CODE 4140–01–M

DEPARTMENT OF HOUSING AND URBAN DEVELOPMENT

[Docket No. FR–4442–N–14]

Notice of Proposed Information Collection for Public Comment: Notice of Funding Availability and Application Kit for the Hispanic-Serving Institutions Work Study Program (HSI–WSP)

AGENCY: Office of Policy Development and Research, HUD.

ACTION: Notice.

SUMMARY: The proposed information collection requirement described below will be submitted to the Office of Management and Budget (OMB) for review, as required by the Paperwork Reduction Act. The soliciting public comments on the subject proposal.

DATES: *Comments Due Date:* January 18, 2000.

ADDRESSES: Interested persons are invited to submit comments regarding this proposal. Comments should refer to the proposal by name or OMB Control Number and be sent to: Reports Liaison Officer, Office of Policy Development and Research, U.S. Department of Housing and Urban Development, 451 7th Street, SW, Room 8226, Washington, DC 20410.