system (TRL–5) entirely based on cis-cleaving (trimming) hairpin ribozymes (triplex system) that release R434 from long transcripts. Because of the modular structure of the hairpin ribozyme, the catalytic domain B can independently recognize cis or trans targets allowing the use of the same ribozymes for both trimming and therapeutic duties. Thus, this improved system was designed as a three-ribozymes unit in a canonical triplex using an inverted cleavage from one trimming ribozyme.

The Rz434bis system was designed to use a single R434 ribozyme to catalyze both trimming and trans-acting activities. This procedure resulted in a reduced-size triplex system that uses R434 catalytic domain to self-excise itself. RNA from Rz434bis and TRL–5 templates released R434 by a self-processing mechanism thus allowing for the individual activity of multiple trans-acting ribozymes. Both Rz434bis and TRL–5 systems produced an increased cleavage efficiency of HPV–16 target site nt 410 to 445 when expressed from linear or circular templates. Furthermore, duplex Rz434bis and TRL–5 were more efficient in cleaving E6 than duplex single R434. The use of triplex configurations with multi-target ribozymes will ultimately result in better in vivo HPV–16 E6/E7 mRNA degradation. Therefore, implementation of the triplex systems that significantly enhance R434 in vitro activity is offered as an alternative to the antisense oligodeoxynucleotide treatment of cervical cancer.

Genomic Nucleic Acid Sequence for Cyanovirin-N and Signal Peptide Thereof

Dr. Angela Gronenborn (NIDDK),
U.S. Provisional Application No. 60/695,599 filed 05 Jul 2005 (HHS Reference No. E–133–2005/0–US–01),
Licensing Contact: Sally Hu; 301/435–5606; *hus@mail.nih.gov.*

The invention provides composition claims for an isolated or purified genomic nucleic acid sequence encoding a CV–N signal peptide, as well as an isolated or purified nucleic acid comprising a genomic sequence encoding a Cyanovirin-N (CV–N) polypeptide native to the cyanobacterium species *Nostoc ellipsosporum.* The signal peptide can be used for directing the secretion of CV–N polypeptide. Further development of the invention may yield novel therapies and methods in the prevention of HIV and other retroviruses, such as HTLV–1 and 2, FLV, and treatment of chronic infection in patients with resistance to current HIV therapies. The invention also includes vectors and cells comprising this sequence, methods for producing a polypeptide, and a method for inhibiting viral infection in a mammal by administering a viral-infection inhibiting amount of the nucleic acid, vector and/or cell of the invention. It also provides a method of inhibiting virus in biological samples or inanimate objects, and can also be used ex vivo for virucidal sterilization.

GP41 Inhibitor

G. Marius Clore *et al.* (NIDDK),
U.S. Provisional Application No. 60/339,751 filed 17 Dec 2001 (HHS Reference No. E–252–2001/0–US–01); PCT Application No. PCT/US02/40684 filed 17 Dec 2002 (HHS Reference No. E–252–2001/0–PCT–02); U.S. Patent Application No. 10/499,094 filed 14 Jun 2004 (HHS Reference No. E–252–2001/0–US–03),
Licensing Contact: Susan Ano; 301/435–5515; *anos@mail.nih.gov.*

The technology relates to a chimeric molecule, NCCG-gp41, in which the internal trimeric helical coiled-coil of the ectodomain of gp41 is fully exposed and stabilized by both fusion to a minimal ectodomain core of gp41 and by engineered intersubunit disulfide bonds. NCCG-gp41 inhibits HIV envelope mediated cell fusion at nanomolar concentrations with an IC50 of 16 nM. It is proposed that NCCG-gp41 targets the exposed C-terminal region of the gp41 ectodomain in its pre-hairpin intermediate state, thereby preventing the formation of the fusogenic form of the gp41 ectodomain that comprises a highly stable trimer of hairpins arranged in a six-helix bundle. NCCG-gp41 has potential as (a) an HIV therapeutic agent that inhibits cell entry; (b) as an AIDS vaccine and; (c) as a component of a high throughput screening assay for small molecule inhibitors of HIV envelope mediated cell fusion. Antibodies have been raised against NCCG-gp41 that inhibit HIV envelope mediated cell fusion.

This invention is further described in: J.M. Louis *et al.*, ''Design and properties of NCCG-gp41, a chimeric gp41 molecule with nanomolar HIV fusion inhibitory activity,'' J. Biol. Chem. (2001 Aug 3) 276(31):29485–29489; C.A. Bewley *et al.*, ''Design of a novel peptide inhibitor of HIV fusion that disrupts the internal trimeric coiled-coil of gp41,'' J. Biol. Chem. (2002 Apr 19) 277(16):14238–14245; J.M. Louis *et al.*, ''Covalent trimers of the internal N-terminal trimeric coiled-coil of gp41 and antibodies directed against them are potent inhibitors of HIV envelope-mediated cell fusion,'' J. Biol. Chem. (2003 May 30) 278(22):20278–20285; J.M. Louis *et al.*, ''Characterization and HIV–1 fusion inhibitory properties of monoclonal Fabs obtained from a human non-immune phage library selected against diverse epitopes of the ectodomain of HIV–1 gp41,'' J. Mol. Biol. (2005 Nov 11) 353(5):945–951.

Dated: December 19, 2005.

Steven M. Ferguson,

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

[FR Doc. E5–8121 Filed 12–29–05; 8:45 am]

BILLING CODE 4140–01–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, HHS.

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.

Molecular Cloning and Characterization of SNAPIN: A Synaptic Vesicle Protein Implicated in Neurotransmitter

Dr. Zu-hang Sheng *et al.* (NINDS),
HHS Reference No. E–182–1999/0—Research Tool,
Licensing Contact: Marlene Shinn-Astor; 301/435–4426; *shinnm@mail.nih.gov.*

Neurotransmitter release is dependent on a binding complex (designated as SNAR) of three proteins, synaptic-vesicle-associated protein synaptobrevin/VAMP, syntaxin and SNAP–25 (snaptosome-associated protein-25) with results in a calcium

dependent fusion between synaptic vesicles and the presynaptic terminal. SNAPIN, a neuron specific protein found predominately on synaptic vesicles, binds to the SNAR complex, most likely to the SNAP–25. Although the complete function of SNAPIN has not been determined, it appears to regulate a step between vesicle docketing and neurotransmitter release through its ability to potentiate the interaction of synaptotagmin with the SNAREs, which then leads to the final fusion step triggered by calcium influx into nerve terminals through voltage-dependent calcium channels.

A Mouse With a Targeted Mutation in the Uncoupling Protein-3 (upc3) Gene

Dr. Marc Reitman *et al.* (NIDDK),

HHS Reference No. E–031–1999/0—Research Tool,

Licensing Contact: Marlene Shinn-Astor; 301/435–4426; *shinnm@mail.nih.gov.*

The NIH announces the development of a transgenic mouse with a targeted mutation in the ucp3 gene. The ucp3 gene is implicated I the function of regulating energy metabolism. This regulatory function is thought to be accomplished by changing metabolic efficiency (causing energy expended as heat rather than used for ADP/ATP conversion) and/or by participating in fat metabolism. The mutation should inactivate the ucp3 function and the mouse provided a testing vehicle for the above hypotheses.

If in fact ucp3 is involved in energy efficiency and/or fat metabolism, then variation in its sequence or level of expression may explain some of human obesity. If ucp3 is involved in fever generation, it would be of interest in testing inactivating drugs.

In summary, this mouse model provides a model for evaluating the role of ucp3 in obesity, energy efficiency, and selective use of energy sources (i.e., fat vs. carbohydrates), body temperature regulation, such as fever, or other forms of stimulated thermogenesis (e.g., by diet of dietary fat). For example, a drug candidate thought to act via ucp3 should have no effect in these mice.

Dated: December 19, 2005.

Steven M. Ferguson,

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

[FR Doc. E5–8122 Filed 12–29–05; 8:45 am]

BILLING CODE 4140–01–P

DEPARTMENT OF HEALTH AND HUMAN SERVICES

National Institutes of Health

National Cancer Institute; Notice of Closed Meeting

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

The meeting will be closed to the public in accordance with the provisions set forth in section 552b(c)(4) and 552b(c)(6), Title 5 U.S.C., as amended. The grant applications and the discussions could disclose confidential trade secrets or commercial property such as patentable material, and personal information concerning individuals associated with the grant applications, the disclosure of which would constitute a clearly unwarranted invasion of personal privacy.

Name of Committee: National Cancer Institute Special Emphasis Panel; Molecular Oncology 2.

Date: February 21–22, 2006.

Time: 4 p.m. to 6 p.m.

Agenda: To review and evaluate grant applications.

Place: Marriott Bethesda North Hotel and Conference Ctr., 5700 Marinelli Road, North Bethesda, MD 20852.

Contact Person: Shamala K. Srinivas, PhD, Scientific Review Administrator, Grants Review Branch, Division of Extramural Activities, National Cancer Institute, National Institutes of Health, 6116 Executive Boulevard, Room 8133, Bethesda, MD 20892, 301–594–1224.

(Catalogue of Federal Domestic Assistance Program Nos. 93.392, Cancer Construction; 93.393, Cancer Cause and Prevention Research; 93.394, Cancer Detection and Diagnosis Research; 93.395, Cancer Treatment Research; 93.396, Cancer Biology Research; 93.397, Cancer Centers Support; 93.398, Cancer Research Manpower; 93.399, Cancer Control, National Institutes of Health, HHS)

Dated: December 22, 2005.

Anna Snouffer,

Acting Director, Office of Federal Advisory Committee Policy.

[FR Doc. 05–24651 Filed 12–29–05; 8:45 am]

BILLING CODE 4140–01–M

DEPARTMENT OF HEALTH AND HUMAN SERVICES

National Institutes of Health

National Center for Complementary & Alternative Medicine; Notice of Closed Meeting

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

The meeting will be closed to the public in accordance with the provisions set forth in sections 552b(c)(4) and 552b(c)(6), Title 5 U.S.C., as amended. The grant applications and the discussions could disclose confidential trade secrets or commercial property such as patentable material, and personal information concerning individuals associated with the grant applications, the disclosure of which would constitute a clearly unwarranted invasion of personal privacy.

Name of Committee: National Center for Complementary and Alternative Medicine Special Emphasis Panel; Clinical Science.

Date: February 13–14, 2006.

Time: 8 a.m. to 5 p.m.

Agenda: To review and evaluate grant applications.

Place: Bethesda Marriott Suites, 6711 Democracy Boulevard, Bethesda, MD 20817.

Contact Person: Jeanette M Hosseini, Scientific Review Administrator, National Center for Complementary and Alternative Medicine, 6707 Democracy Blvd, Suite 401, Bethesda, MD 20892. (301) 594–9096.

Dated: December 20, 2005.

Anna Snouffer,

Acting Director, Office of Federal Advisory Committee Policy.

[FR Doc. 05–24662 Filed 12–29–05; 8:45 am]

BILLING CODE 4140–01–M

DEPARTMENT OF HEALTH AND HUMAN SERVICES

National Institutes of Health

National Eye Institute; Notice of Meeting

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

The meeting will be open to the public as indicated below, 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.

The meeting will be closed to the public in accordance with the provisions set forth in sections 552b(c)(4) and 552b(c)(6), Title 5 U.S.C., as amended. The grant applications and/or contract proposals and the