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Director, Warm Springs Service Unit, Warm Springs Indian Health Center, P.O. Box 1209, Warm Springs, Oregon 97761.

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Director, Sells Service Unit, Santa Rosa Indian Health Center, HCO1, Box 8700, Sells, Arizona 85634.

Director, Sells Service Unit, Sells Indian Hospital, P.O. Box 548, Sells, Arizona 85634.

Director, Sells Service Unit, West Side Health Station, P.O. Box 548, Sells, Arizona 85634.

Appendix 2—Federal Archives and Records Centers

District of Columbia, Maryland Except U.S. Court Records for Maryland, Washington National Records Center, 4205 Suitland Road, Suitland, Maryland 20746-8001.

Connecticut, Maine, Massachusetts, New Hampshire, Rhode Island, and Vermont, Federal Archives and Records Center, Frederick C. Murphy Federal Center, 380 Trapelo Road, Waltham, Massachusetts 02452-6399.

Northeast Region, Federal Archives and Records Center, 10 Conte Drive, Pittsfield, Massachusetts 01201-8230.

Mid-Atlantic Region and Pennsylvania, Federal Archives and Records Center, 14700 Townsend Road, Philadelphia, Pennsylvania 19154-1096.

Alabama, Florida, Georgia, Kentucky, Mississippi, North Carolina, South Carolina, and Tennessee, Federal Archives and Records Center, 1557 St. Joseph Avenue, East Point, Georgia 30344-2593.

Illinois, Indiana, Michigan, Minnesota, Ohio and Wisconsin and U.S. Court Records for the mentioned States, Federal Archives and Records Center, 7358 South Pulaski Road, Chicago, Illinois 60629-5898.

Michigan, Except U.S. Court Records, Federal Records Center, 3150 Springboro Road, Dayton, Ohio 45439-1883.

Kansas, Iowa, Missouri and Nebraska, and U.S. Court Records for the mentioned States, Federal Archives and Records Center, 2312 East Bannister Road, Kansas City, Missouri 64131-3011.

New Jersey, New York, Puerto Rico, and the U.S. Virgin Islands, and U.S. Court Records for the mentioned States and territories, 200 Space Center Drive, Lee's Summit, Missouri 64064-1182.

Arkansas, Louisiana, Oklahoma and Texas, and U.S. Courts Records for the mentioned States, Federal Archives and Records Center, P.O. Box 6216, Ft. Worth, Texas 76115-0216.

Colorado, Wyoming, Utah, Montana, New Mexico, North Dakota, and South Dakota, and U.S. Courts Records for the mentioned States, Federal Archives and Records Center, P.O. Box 25307, Denver, Colorado 80225-0307.

Northern California Except Southern California, Hawaii, and Nevada Except Clark County, the Pacific Trust Territories, and American Samoa, and U.S. Courts Records for the mentioned States and territories, Federal Archives and Records Center, 1000 Commodore Drive, San Bruno, California 94066-2350.

Arizona, Southern California, and Clark County, Nevada, and U.S. Courts Records for the mentioned States, Federal Archives and Records Center, 23123 Cajalco Road, Perris, California 93570-7298.

Washington, Oregon, Idaho and Alaska, and U.S. Courts Records for the mentioned States, Federal Archives and Records Center, 6125 Sand Point Way NE, Seattle, Washington 98115-7999.

Dated: December 22, 2005.

Charles W. Grim,

Assistant Surgeon General, Director, Indian Health Service.

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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.

A Single Ribozyme To Catalyze Both Trimming and Transacting Catalysis—Potential Therapeutic for HPV Infection and Cervical Cancer

Joseph A. DiPaolo (NCI) *et al.*,

U.S. Provisional Application No. 60/675,076 filed 25 April 2005 (HHS Reference No. E-142-2005/0-US-01),

Licensing Contact: Robert M. Joynes; 301/594-6565; joynesr@mail.nih.gov.

This technology relates to a potential therapeutic for treating human papillomavirus (HPV) infection as well as cervical cancer. It is acknowledged that HPV is the primary agent associated with cervical cancer. The life cycle of HPVs progresses with epithelial differentiation and may persist for decades. The E6 and E7 oncogenes are responsible for two viral proteins that target p53 and Rb. The persistence of E6 and E7 in cervical carcinomas has led to them being recognized as the hallmark of cervical carcinomas and makes them excellent targets for therapy. Previously, we reported an engineered hairpin ribozyme (R434) that caused down-regulation of HPV-16 E6/E7 mRNA and inhibited growth of both HPV-16 immortalized cells and tumor cells. To increase efficiency of R434 we constructed a ribozyme expression

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.

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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