aimed at developing control strategies such as vaccines and therapeutic drugs.

**Inventors:** Gael M. Belliot, Kim Y. Green, Stanislav V. Sosnovtsev (NIAID)


**Licensing Status:** The cDNA clone for norovirus strain MD145–12 is available for licensing via a biological material license (BML).

**Licensing Contact:** Peter A. Soukas, J.D.; 301/435–4646; soukasp@mail.nih.gov

**Collaborative Research Opportunity:** The Laboratory of Infectious Diseases, NIAID, NIH, is seeking statements of capability or interest from parties interested in collaborative research to further develop, evaluate, or commercialize reagents derived from an infectious cDNA copy of the genome of porcine enteric calicivirus. Please contact Kim Y. Green at kgreen@niaid.nih.gov for more information.

**Description of Technology:** The noroviruses (formerly known as “Norwalk-like viruses”) are associated with gastroenteritis outbreaks, affecting large numbers of individuals each year. Emerging data are supporting their increasing recognition as important agents of diarrhea-related morbidity and mortality. The frequency with which noroviruses are associated with gastroenteritis as “food and water-borne pathogens” has led to the inclusion of caliciviruses as Category B Bioterrorism Agents/Diseases. Because the noroviruses cannot be propagated by any means in the laboratory, an important strategy in their study is the development of molecular biology-based tools and replication systems. This invention reports the isolation of the first recombinant, enzymatically-active proteinase and RNA dependent RNA polymerase (RdRp) complex for a human norovirus. This enzyme should facilitate studies aimed at developing therapeutic drugs for norovirus disease.

**Inventors:** Gaeil M. Belliot, Stanislav V. Sosnovtsev, Kyeong-Ok Chang, Kim Y. Green (NIAID).

**Publication:** The materials are further described in L Wei et al., “Proteinase-polymerase precursor as the active form of feline calicivirus RNA-dependent RNA polymerase,” J. Virol. 2001 Feb;75(3):1211–1219.


**Licensing Status:** The materials embodied in this invention are available nonexclusively through a biological materials license.

**Licensing Contact:** Peter A. Soukas, J.D.; 301/435–4646; soukasp@mail.nih.gov

**Collaborative Research Opportunity:** The Laboratory of Infectious Diseases, NIAID, NIH, is seeking statements of capability or interest from parties interested in collaborative research to further develop, evaluate, or commercialize an active human norovirus proteinase-polymerase enzyme. Please contact Kim Y. Green at kgreen@niaid.nih.gov for more information.

Dated: June 8, 2007.

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

[FR Doc. E7–11826 Filed 6–19–07; 8:45 am]

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.

**Methods for Prevention and Treatment of Polyomavirus Infection or Reactivation**

**Description of Technology:** Available for licensing and commercial development are methods of using two MAP kinase kinase (MEK) inhibitors, PD98059 and U0126, in the prevention and treatment of polyomavirus infection. Decrease in viral protein expression upon treatment with the MEK inhibitors has been demonstrated.
for two polyomavirus species, JC virus (JCV) and BK virus (BKV). It is believed that these MEK inhibitors may also be effective against other polyomavirus species in which TGF-β expression is elevated.

JCV is responsible for the demyelination of the central nervous system which is observed in cases of progressive multifocal leukoencephalopathy (PML). PML is most frequently seen in patients with HIV/AIDS, but is also a contributing factor in fatalities in patients with leukemia, lymphoma, and connective tissue diseases, in addition to individuals receiving immunosuppressive therapy for autoimmune disorders or prevention of transplant rejection.

BKV is associated with deadly clinical syndromes such as viruria and viremia, ureteral ulceration and stenosis, and hemorrhagic cystitis. BKV also causes polyomavirus-associated nephropathy in 1–10% of all renal transplant recipients.

Currently, no effective antiviral agents are available to treat these opportunistic infections. In all observed cases, activation of either JCV and BKV in immuno-compromised patients has resulted in mortality.

**Development Status:** In vitro data is currently available and inventors are actively developing the technology.

**Inventors:** Veersamy Ravichandran and Eugene Major (NINDS).


**Licensing Status:** Available for licensing.

**Licensing Contact:** Cristina Thalhammer-Reyero, Ph.D., M.B.A.; 301/435–4507; thalhamc@mail.nih.gov.

**Collaborative Research Opportunity:** The National Institute of Neurological Disorders and Stroke is seeking statements of capability or interest from parties interested in collaborative research to further develop, evaluate, or commercialize treatment and prevention of polyomavirus infections in immunocompromised patients. Please contact Melissa Maderia, Ph.D. at maderiam@mail.nih.gov for more information.

### Monoclonal Antibodies That Bind or Neutralize Hepatitis B Virus

**Description of Technology:** Hepatitis B virus (HBV) chronically infects over 300 million people worldwide. Many of them will die of chronic hepatitis or hepatocellular carcinoma. The present technology relates to the isolation and characterization of a novel neutralizing chimpanzee monoclonal antibody to HBV. The antibody was identified through a combinatorial antibody library constructed from bone marrow cells of a chimpanzee experimentally infected with HBV. The selected monoclonal antibody has been shown to react equally well with wild-type HBV and the most common neutralization escape mutant variants. Therefore, this monoclonal antibody with high affinity and broad reactivity may have distinct advantages over other approaches to immunoprophylaxis and immunotherapy of chronic HBV infection, as most of the monoclonal antibodies currently in use are not sufficiently and broadly reactive to prevent the emergence of neutralization escape mutants of HBV. This technology describes such antibodies, fragments of such antibodies retaining hepatitis B virus-binding ability, fully human or humanized antibodies retaining hepatitis B virus-binding ability, and pharmaceutical compositions including such antibodies. This invention further describes isolated nucleic acids encoding the antibodies and host cells transformed with nucleic acids. In addition, this invention provides methods of employing these antibodies and nucleic acids in the in vitro and in vivo diagnosis, prevention and therapy of HBV diseases.

**Inventors:** Suzanne U. Emerson (NIAID), Robert H. Purcell (NIAID), et al.


**Licensing Contact:** Cheksha S. Clingman, Ph.D.; 301/435–5018; clingmac@mail.nih.gov.

### Endotracheal Tube Using Unique Leak Hole To Lower Dead Space

**Description of Technology:** Through injury or diseases, human or animal lungs may become too weak to sustain a sufficient flow of oxygen to the body and to pulmonary ventilation requires large amounts of expired carbon dioxide. The present invention is a tracheal tube ventilation apparatus which efficiently rids patients of expired gases and promotes healthier breathing. This is accomplished by creating one or more leak holes in the wall of the endotracheal tube above the larynx, such as in the back of the mouth (i.e., oropharynx), so that expired gases can leak out of the endotracheal tube. The described apparatus is a two stage tube where the first stage has a smaller diameter such that it fits within the confined area of the lower trachea and the second stage has a larger diameter, which fits properly within the larger diameter of the patient’s pharynx. The endotracheal tube is preferably wire reinforced and ultra-thin walled so as to reduce airway resistance. The invention substantially reduces endotracheal dead space and is expected to benefit those patients with both early and late stage acute respiratory failure, and reduce or obviate the need for mechanical pulmonary ventilation in many patients.

**Applications:** Tracheal tube ventilation; Efficiently rid patient of expired gases and thereby promote healthier breathing.

**Development Status:** System is well developed and operational.

**Inventor:** Theodor Kolobow (NHLBI).


**Licensing Status:** Available for non-exclusive or exclusive licensing.

**Licensing Contact:** Michael A. Shmilovich, Esq.; 301/435–5019; shmilovm@mail.nih.gov.

**Collaborative Research Opportunity:** The NHLBI/Pulmonary Critical Care Medicine Branch (PCCMB) is seeking statements of capability or interest from parties interested in collaborative research to further develop, evaluate, or commercialize innovative endotracheal tube technology. Please contact Marianne Lynch at 301–594–4094 or lynchnh@nhlbi.nih.gov for more information.

### Increased Protein Expression Vector for Vaccine Applications

**Description of Technology:** An expression vector with a unique promoter that results in higher level of protein expression than vectors currently in use is available for licensing from the NIH. The elevated
DEPARTMENT OF HEALTH AND HUMAN SERVICES

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**Vibrio Cholerae O139 Conjugate Vaccines**

Description of Technology: Cholera remains an important public health problem. Epidemic cholera is caused by two Vibrio cholerae serotypes O1 and O139. The disease is spread through contaminated water. According to information reported to the World Health Organization in 1999, nearly 8,500 people died and another 223,000 were sickened with cholera worldwide. This invention is a polysaccharide-protein conjugate vaccine to prevent and treat infection by Vibrio cholerae O139 comprising the capsular polysaccharide (CPS) of V. cholerae O139 conjugated through a dicarboxylic acid dihydrazide linker to a mutant diphtheria toxin carrier. In addition to the conjugation methods, also claimed are other methods of immunization against V. cholerae O139 using the conjugates of the invention. The inventors have shown that the conjugates of the invention elicited in mice high levels of serum antibodies to CPS, a surface antigen of Vibrio cholerae O139, that have vibriocidal activity. Clinical trials of the two most immunogenic conjugates have been planned by the inventors. The conjugate vaccine is aimed for long lasting immunity, especially in young children, and can be administered in concurrent with routine vaccines.

Inventors: Shousun Szu, Zuzana Kossaczka, John Robbins (NICHD).


Licensing Status: Available for exclusive or non-exclusive licensing.

Licensing Contact: Peter A. Soukas, J.D.; 301/435–4646; soukas@email.nih.gov

Collaborative Research Opportunity: The NICHD/LMDI is seeking statements of capability or interest from parties interested in collaborative research to further develop, evaluate, or commercialize Vibrio cholera O139 or O1 conjugate vaccines. Please contact John D. Hewes, Ph.D. at 301–435–3121 or hewes@mail.nih.gov for more information.

**CC Chemokine Receptor 5 DNA, New Animal Models and Therapeutic Agents for HIV Infection**

Description of Technology: Chemokine receptors are expressed by many cells, including lymphoid cells, and function to mediate cell trafficking and localization. CC chemokine receptor 5 (CCR5) is a seven-transmembrane, G protein-coupled receptor (GPCR) which regulates trafficking and effector functions of memory/effector T-lymphocytes, macrophages, and immature dendritic cells. Chemokine binding to CCR5 leads to cellular activation through pertussis toxin-sensitive heterotrimeric G proteins as well as G protein-independent signalling pathways. Like many other GPCRs, CCR5 is regulated by agonist-dependent processes which involve G protein coupled receptor kinase (GRK)-dependent phosphorylation, beta-arrestin-mediated desensitization and internalization.

Human CCR5 also functions as the main coreceptor for the fusion and entry of many strains of human immunodeficiency virus (HIV–1, HIV–2). HIV–1 transmission almost invariably involves such CCR5-specific variants (designated R5); individuals lacking functional CCR5 (by virtue of homozygosity for a defective CCR5 allele) are almost completely resistant to HIV–1 infection. Specific blocking of CCR5 (e.g. with chemokine ligands, anti-CCR5 antibodies, CCR5-blocking low MW inhibitors, etc.) inhibits entry/infected of target cells by R5 HIV strains. Cells expressing CCR5 and CD4 are useful for screening for agents that inhibit HIV by binding to CCR5. Such agents represent potential new