Although this technology is still in an early stage, our researchers have obtained solid evidence of the involvement of Smad3 in these processes by use of a Smad3 null mouse model which they have developed. Based on these results, it is believed that antisense Smad3 or small molecule inhibitors of Smad3 will have clinical applications in wound healing, in improving growth and reducing unwanted fibrosis of autologous skin grafts for treatment of burn patients, and in treatment of radiation fibrosis and other fibrotic diseases associated with chronic inflammation. In addition, the discovery of inhibitors to Smad3 signaling may lead to radiation dose escalation and accelerated tumor cell death while reducing the side effects associated with radiation therapy.

Use of Smad3 Inhibitor in the Treatment of Fibrosis Dependent on Epithelial to Mesenchymal Transition as in the Eye and Kidney

Anita B. Roberts (NCI)

PCT Application No. PCT/US04/03563 Filed 16 Jan 2004 (DHHS Reference No. E–062–2003/3–PCT–01)

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

Fibroid scar tissue has been associated with wound healing of the epithelial layer following tissue damage created by surgery or other means. Examples of which include the opaque scar tissue associated with cataract surgery and the fibroid scar tissue produced in several kidney diseases such as is seen in unilateral ureteral obstruction.

Smad2 and Smad3 are highly homologous cytoplasmic proteins which function to mediate signals from Transforming Growth Factor Beta (TGF–B) and activin receptors to promoters of target genes found in the nucleus. The NIH announces a technology wherein Smad 3 is now implicated in TGF–B-dependent transdifferentiation of epithelial cells to mesenchymal cells (EMT), which blocks the endpoint of fibrosis at an early stage of differentiation of epithelial cell precursors into interstitial fibroblasts. In particular, fibrosis was blocked following wounding of the lens of the eye and damage created to the kidney. It is believed that an inhibitor of Smad 3 could be used to block fibrosis following cataract surgery and lens implantation in patients, as well as slowing the progression of end-stage renal disease.

Dated: September 22, 2004.

Steven M. Ferguson,

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

[FR Doc. 04–22148 Filed 9–30–04; 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.

Bovine Adeno-Associated Viral (BAAV) Vector and Uses Thereof

John Chiorini et al. (NIDCR)

U.S. Provisional Application No. 60/526,786 Filed 04 Dec 2003 (DHHS Reference No. E–329–2003/0–US–01)

Licensing Contact: Jesse Kindra; (301) 435–5559; *kindraj@mail.nih.gov*.

Adeno-associated viruses (AAVs) are common in humans, but no disease has been associated with AAV infections. This, as well as several other properties, has made AAVs potentially useful for gene therapy. Bovine AAV (BAAV) is serologically distinct from AAVs isolated from humans and may not be neutralized by circulating antibodies in patients receiving gene therapy. Moreover, BAAV has a unique tropism for various cell lines when compared to other AAVs. For instance, recombinant BAAV transduced murine submandibular salivary glands about ten times more efficiently than AAV–2. Therefore, BAAV may be a useful addition to the repertoire of gene transfer tools because of its unique serological identity, cell tropism, and efficient gene transfer in vivo.

The present invention describes the isolation, subcloning and sequencing of BAAV and provides a vector comprising BAAV viral particles, or a vector comprising subparts of the vectors. The invention also provides a method of delivering a nucleic acid to a cell subject. We note that this vector may also have future application(s) in the cattle industry.

This invention has been described in Schmidt *et al.* 2004. J. Virol. 78:6509–16.

Treatments for Inhibiting Development and Progression of Nevi and Melanoma Having *BRAF* Mutations

Paul S. Meltzer (NHGRI)

PCT Application No. PCT/US03/32989 Filed 16 Oct 2003 (DHHS Reference No. E–021–2003/0–PCT–01)

Licensing Contact: Charmaine Richman; (301) 451–7337; *richmanc@mail.nih.gov*.

The technology encompasses activating mutations in the *BRAF* gene that promote nevi and melanoma proliferation. These mutations produce an activated form of B–Raf, a serine/threonine kinase participant in the Ras/Raf/MEK/ERK MAPK pathway. In one example of the activating *BRAF* mutations, a 1796 T → A transversion produces a V599E mutated form of B–Raf. This mutated form of B–Raf possesses a tenfold greater basal kinase activity and induces focus formation in NIH3T3 cells 138 times more efficiently than does wild type B–Raf. Methods of diagnosing *BRAF* mutations in a subject, methods of treating nevi and melanoma in subjects having *BRAF* mutations, methods of selecting treatments, methods of screening for agents that influence B–Raf activity, and methods of influencing the expression of *BRAF* or *BRAF* variants are also claimed. Nucleotide sequences for use in the described methods are also provided, as are protein-specific binding agents, such as antibodies, that bind specifically to at least one epitope of a B–Raf variant protein preferentially compared to wild type B–Raf.

Important publications: *Oncogene* (2004) 23, 4060–4067; *Nature* (2002) 417(6892), 949–54.

Lentivirus Vector System

Suresh K. Arya (NCI)

U.S. Provisional Application No. 60/115,247 Filed 07 Jan 1999 (DHHS Reference No. E–231–1998/0–US–01)

PCT Application No. PCT/US/00/00390 Filed 06 Jan 2000, which published as WO 00/40741 on 13 Jul 2000 (DHHS Reference No. E–231–1998/0–PCT–02)

U.S. Patent Application No. 09/869,588 Filed 28 Jun 2001 (DHHS Reference No. E–231–1998/0–US–03)

U.S. Patent Application No. 10/731,988 Filed 09 Dec 2003 (DHHS Reference No. E–231–1998/0–US–04)

Licensing Contact: Jesse Kindra; (301) 435–5559; *kindraj@mail.nih.gov*.

This application relates to the field of gene therapy. More particularly the application describes a vector system useful in gene therapy. The vectors employed in this system are lentiviral vectors, particularly retroviral vectors based on HIV2. Retroviral vectors based on HIV2, unlike most other retroviral vectors such as MuLV, are capable of infecting non-proliferating cells thereby making them useful in situations where other retroviral vectors are not. The vector system uses a two vector approach to minimize the possibility of HIV infection and comprises a transfer vector, for carrying the foreign gene of interest, and a packaging vector. The transfer vector carries a specific modification that demonstrates an improved ability to package and express the gene of interest when compared to a control. In the experimental system this increase was 25 fold. This improved packaging and expression ability is one means to address current low viral titers which are problematic in the gene therapy field.

This research has been published, in part, in Human Gene Therapy 1998 June 10; 9(9): 1371–86.

Food Quality Indicator Device

Dwight W. Miller, Jon G. Wilkes, Eric D. Conte (FDA)

U.S. Provisional Application No. 60/052,674 Filed 17 Jul 1997 (DHHS Reference No. E–093–1997/0–US–01)

PCT Application No. PCT/US98/14780 Filed 16 Jul 1998, which published as WO 99/04256 on 28 Jan 1999 (DHHS Reference No. E–093–1997/0–PCT–04)

U.S. Patent Application No. 09/116,152 Filed 16 Jul 1998 (DHHS Reference No. E–093–1997/0–US–02)

U.S. Patent Application No. 10/005,004 Filed 04 Dec 2001 (DHHS Reference No. E–093–1997/0–US–03)

Licensing Contact: George Pipia; (301) 435–5560; *pipiag@mail.nih.gov*.

Scientists at the U.S. Food and Drug Administration have invented an effective way to monitor food quality and freshness in real time. The major factor for food spoilage is the release of volatile gases due to the action of enzymes contained within the food or produced by microorganisms, such as bacteria, yeasts and molds growing in the food. The rate of release of such gases depends on food's storage history. In this technology, a reactive dye locked in a water-repellent material reacts with the gases released during food decomposition, and changes color. Thus a rapid and informed decision can be made about quality of food and its shelf life under the storage conditions used. Since the detection is based on biological processes that are the root cause for food spoilage, these indicators are much more reliable.

This technology provides an excellent alternative to the current methods for assessing food quality that cannot accurately estimate shelf life of food products due to unreliable storage history. This technology is also much less expensive than the current methods. These indicators have been successfully tested on seafood and meats and can be easily adapted to dairy products. This product is fully developed, market-tested and ready for full commercial rollout.

Dated: September 22, 2004.

Steven M. Ferguson,

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

[FR Doc. 04–22149 Filed 9–30–04; 8:45 am]

BILLING CODE 4140–01–P

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National Institutes of Health

Government-Owned Inventions; Availability for Licensing

AGENCY: National Institutes of Health, Public Health Service, DHHS.

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DNA-Based Vaccination of Retroviral Infected Individuals Undergoing Treatment

Barbara K. Felber *et al.* (NCI)

U.S. Provisional Application filed 09 Jul 2004 (DHHS Reference No. E–249–2004/0–US–01); PCT Application No. PCT/US01/45624 filed 01 Nov 2001, which published as WO02/36806 on 10 May 2002 (DHHS Reference No. E–308–2000/0–PCT–02); National Stage filed in EP, CA, AU, JP, and U.S. (DHHS Reference No. E–308–2000/0–US–07)

Licensing Contact: Susan Ano; 301/435–5515; *anos@mail.nih.gov*.

This technology describes DNA-based vaccine vectors that produce either secreted or intracellularly degraded antigens that can be administered to individuals receiving antiretroviral therapy (ART) against HIV. Because some of the virus is sequestered in reservoirs, thus evading ART, drug regimen does not result in complete clearance of the virus, and prolonged ART is associated with toxicity and development of virus resistance. These vectors have recently been shown to work unusually well in controlling viremia when administered as DNA vaccines to SIV-infected monkeys that