The agenda for the public meeting will be made available on October 8, 2004, on the Internet at *http://www.fda.gov/cder/meeting/ICH_10192004.htm*.

Dated: September 23, 2004.

Jeffrey Shuren,

Assistant Commissioner for Policy.

[FR Doc. 04–22053 Filed 9–30–04; 8:45 am]

BILLING CODE 4160–01–S

DEPARTMENT OF HEALTH AND HUMAN SERVICES

Office of Inspector General

Program Exclusions; Correction

AGENCY: Office of Inspector General, HHS.

ACTION: Notice of program exclusions; correction.

SUMMARY: The HHS Office of Inspector GeneralPublished a document in the **Federal Register** of September 15, 2003, imposed exclusions. The document contained an incorrect exclusion type.

FOR FURTHER INFORMATION CONTACT: Jacqueline Freeman, (410) 786–5197.

Correction

In the **Federal Register** of September 15, 2004, in FR Doc. 20710, on page 55641, correct the exclusion date to read:

LABONTE, MARY ..... SCOTTSDALE, AZ	9/20/2004

Dated: September 21, 2004.

Katherine B. Petrowski,

Director, Exclusions Staff, Office of Inspector General.

[FR Doc. 04–22046 Filed 9–30–04; 8:45 am]

BILLING CODE 4150–04–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.

Cytonectin, Cytonectin Gene and Cytonectin Inhibitors and Binding Ligands and Their Use in the Diagnosis and Treatment of Disease

Soni J. Anderson et al. (NCI)

U.S. Provisional Application No. 60/553,977 Filed 18 Mar 2004 (DHHS Reference No. E–128–2004/0–US–01); U.S. Provisional Application No. 60/578,068 Filed 09 Jun 2004 (DHHS Reference No. E–128–2004/1–US–01)

Licensing Contact: Fatima Sayyid; (301) 435–4521; *sayyidf@mail.nih.gov*.

Cytonectin is a 35K molecular weight protein that displays ion-independent adherence properties, is expressed in a variety of organs and tissues and is evolutionarily conserved from human to rodent and avian species. Within the body it is thought to serve the function of ''super glue'' contributing to cell-cell interactions and 3-dimensional tissue structure and a physiologic ''do not attack'' signal molecule that prevents tissue destruction by cells of monocyte lineage including odontoclasts in secondary teeth. It also plays an important role in the pathology associated with cancer, arthritis, Alzheimer's and Parkinson's disease.

The present invention relates to cytonectin, to polynucleotides that encode cytonectin, to inhibitors and antibodies that bind to cytonectin and to the use of compositions in the diagnosis and treatment of cytonectin-related diseases and conditions.

Genetic Fingerprint of Acute Stroke

Alison E. Baird (NINDS)

U.S. Provisional Application No. 60/575,279 Filed 27 May 2004 (DHHS Reference No. E–306–2003/0–US–01)

Licensing Contact: Fatima Sayyid; (301) 435–4521; *sayyidf@mail.nih.gov*.

Stroke is the third leading cause of death and the leading cause of adult disability in developed countries. Despite the prevalence and burden of this disease, stroke precipitants and pathophysiological mechanisms in individual patients are often unknown. It is also difficult to accurately predict whether a stroke will lead to only minor neurological sequelae or more serious medical consequences. Although animal experiments in focally ischemic brain tissue have indicated that there are alterations in gene expression following a stroke, gene expression profiling has not yet been applied to clinical human stroke, primarily because brain tissue samples are inaccessible and rarely justified.

The present provisional patent application discloses methods of determining whether a subject had an ischemic stroke, methods of determining the prognosis of a subject who had an ischemic stroke, as well as methods of determining an appropriate treatment regimen for a subject who had an ischemic stroke.

Inhibition of Smad3 To Prevent Fibrosis and Improve Wound Healing

Anita B. Roberts et al. (NCI)

U.S. Patent Application No. 10/299,886 Filed 18 Nov 2002 (DHHS Reference No. E–070–2000/0–US–06), claiming priority to PCT Application No. PCT/US00/13725 Filed 19 May 2000 (DHHS Reference No. E–070–2000/0–PCT–01)

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

Millions of dollars are spent each year to heal chronic non-healing wounds and in the treatment of severe burn patients. The NIH announces a new technology that may lead to improved approaches to treatment of burn patients and the reduction of scarring and more rapid closure of both acute (surgical) and chronic wounds (*e.g.,* diabetic, decubitus, and venus statis ulcers).

Smad2 and Smad3 are highly homologous cytoplasmic proteins which function to transduce signals from Transforming Growth Factor–beta (TGF–beta) and activin receptors to promoters of target genes found in the nucleus. This new technology indicates that interference with specific signaling pathways downstream of TGF–beta may be more selective and have a better outcome than approaches aimed at blocking all effects of this pleiotropic cytokine.

Specifically, it is proposed that elimination or inhibition of Smad3 may interfere with fibrogenic mechanisms and reduce the accumulation of scar tissue associated with high dose radiation and wound healing, while increasing the rate of re-epithelialization of wounds.

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.

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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.