PHYSICIAN ENCOUNTER DATA TRAINING SCHEDULE 2000—Continued

Date	Location
September 7, 2000, Chicago, IL	Hyatt Regency Woodfield, 1800 East Golf Road, Schaumburg, IL 60173, (847) 605–1234.
September 13, 2000, Tampa, FL	Hyatt Regency Westshore on Tampa Bay, 6200 Courtney Campbell Causeway, Tampa, FL 33607, (813) 874–1234.
September 20, 2000, San Diego, CA	San Diego Marriott Hotel and Marina, 333 West Harbor Drive, San Diego, CA 32101–7700, (619) 234–1500.

FOR FURTHER INFORMATION CONTACT: Marcy Perkins, Restuccio Healthcare Group, Encounter Data Representative, (901) 385–0123 (telephone); (901) 385–1821 (fax); or e-mail us with your questions at encounterdata@ritecode.com. Information is also available on our homepage at http://www.hcfa.gov/events.

SUPPLEMENTARY INFORMATION:

Background

The Balanced Budget Act of 1997 (BBA) (Public Law 105–33) established the Medicare+Choice (M+C) program. Under the BBA, we must implement a risk adjustment methodology that accounts for variations in per capita costs based on health status and other demographic factors for payment to M+C organizations (M+COs). Risk adjustment implementation began January 1, 2000.

The BBA gives us the authority to collect inpatient hospital data for discharges on or after July 1, 1997, and additional data for services occurring on or after July 1, 1998. Pending OMB approval, M+COs must submit physician encounter data beginning October 1, 2000.

The agenda for the half-day training sessions will include the following topics:

•; Overview of comprehensive risk adjustment models and implementation timeline.

• Review of M+C National Standard Format (M+C NSF).

• Coding tips and resources for obtaining additional coding information.

• Data requirements for physician encounter data.

• Question-and-answer period.

Registration

Registration for these training sessions is required and will be on a first-come, first-served basis, limited to two attendees per organization. A waiting list will be available for additional requests. Registration can be accomplished via the Internet at http://www.hcfa.gov/events or by completing a paper form available at the aforementioned Internet address. A confirmation notice will be sent to attendees upon finalization of registration.

Attendees will be provided with training materials at the time of the training session. There will be two training sessions per day. The morning session will be from 8:30 a.m. to 11:30 a.m.; the afternoon session will be from 1:30 p.m. to 4:30 p.m. Individuals can attend only one session (either morning or afternoon).

Authority: Sections 1851 through 1859 of the Social Security Act (42 U.S.C. 1395w-21 through 1395w-28).

(Catalog of Federal Domestic Assistance Program No. 93.773, Medicare—Hospital Insurance; and Program No. 93.774, Medicare—Supplementary Medical Insurance Program)

Dated: July 25, 2000.

Nancy-Ann Min DeParle,

Administrator, Health Care Financing Administration.

[FR Doc. 00–19158 Filed 7–27–00; 8:45 am]

BILLING CODE 4120–01–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, DHHS.

ACTION: Notice.

SUMMARY: The inventions listed below are owned by agencies 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 contacting Susan S. Rucker, J.D., at the Office of Technology Transfer, National Institutes of Health, 6011 Executive Boulevard, Suite 325, Rockville, Maryland 20852–3804; telephone: 301/496–7056 ext. 245; fax: 301/402–0220; e-mail: ruckers@od.nih.gov. A signed Confidential Disclosure Agreement will be required to receive copies of the patent applications.

Establishment of Cellular Manipulations Which Enhance Oligo-Mediated Gene Targeting

MM Seidman and A Majumdar (both of NIA)

Serial No. 60/191,996 filed 24 Mar 2000

This application relates to gene targeting, illustrated by the use of triplex-forming oligonucleotides (TFO's). In particular, the application describes and claims methods for improving the efficiency of the modification of gene sequence (including mutation and/or recombination) through the use of cells which have been cultured so as to synchronize their cell cycles. According to the method described and claimed in the application gene targeting reagents, as demonstrated by, but not limited to, triple helix forming oligonucleotides, are introduced into cultured, synchronized cells. Gene targeting applications are useful in research applications for the generation of transgenic animals and plants, including animals used as model systems, such as knockout mice, and animals or plants used for production of the product of the transgene of interest. In addition, efficient methods of gene targeting may also be useful in improving or carrying out gene therapy applications.

AAV5 Vector for Transducing Brain Cells and Lung Cells

JA Chiorini (NHLBI/NIDCR), RM Kotin (NHLBI)

Serial No. 09/533,427 filed 22 Mar 2000

The invention described and claimed in this patent application is related to the delivery of heterologous nucleic acids or genes to particular target cells. In particular, the application relates to methods of delivering a heterologous nucleic acid or gene of interest to particular target cells using an Adeno-

Associated Virus of serotype 5 (AAV5). The particular target cells identified include the alveolar cells of the lung and cerebellar and ependymal cells of the brain. The methods described herein may be useful in carrying out gene therapy related to diseases of the brain or central nervous system and the respiratory tract.

This work has been published, in part, at Davidson BL, *et al.* PNAS, USA 97(7):3428–32 (March 28, 2000) and Zabner J, *et al.* J Virol. 74(8):3852–8 (April 2000).

In addition to this patent application, PHS owns additional intellectual property related to this technology. The patent application has been published as WO 99/61601 on December 2, 1999 and the research corresponding thereto has been published at Chiorini JA, *et al.* J. Virol. 73(5): 4293–98 (May 1999) and Chiorini JA, *et al.* J. Virol. 73(2): 1309–19 (Feb. 1999).

AAV4 Vector and Uses Thereof

JA Chiorini (NHLBI/NIDCR), RM Kotin, B Safer (both of NHLBI)
Serial No. 09/532,594 filed 22 Mar 2000

The invention described and claimed in this patent application relates to the delivery of heterologous nucleic acids or genes to particular target cells. In particular, the application relates to methods of delivering a heterologous nucleic acid or gene of interest to particular target cells using Adeno-Associated Virus of serotype 4 (AAV4). The particular target cells identified are the ependymal cells of the brain. The methods described herein may be useful in carrying out gene therapy for diseases of the brain or central nervous system.

This work has been published in part at Davidson, BL, *et al.* ''Recombinant adeno-associated virus type 2, 4, and 5 vectors: transduction of variant cell types and regions in the mammalian central nervous system'' PNAS USA 97(7):3428–32 (March 28, 2000).

In addition, PHS owns additional intellectual property related to this technology describing an AAV4-based vector system. The material contained in the patent application has been published as WO 98/11244 (March 19, 1998) and the research corresponding thereto has been published in J. Virology 71(9): 6823–33 (Sept 1997).

A Novel Pro-Apoptotic Protein, ARTS

S Larisch-Bloch, SJ Kim, RJ Lechleider, AB Roberts and Y Yi (all of NCI)
Serial No. 60/178,866 filed 29 Jan 2000

This application relates to the field of apoptosis, in particular the application relates to a novel gene product which is associated with induction of apoptosis by Transforming Growth Factor Beta (TGF–β). Apoptosis is a critical event in developmental processes and homeostasis; its dysregulation is often central to pathogenic mechanisms. Apoptotic aberrations contribute to the development of the transformed phenotype; both metastatic potential and tumor aggressiveness are associated with increased resistance to apoptosis. Certain chemotherapeutic agents act by increasing the sensitivity of cells to apoptosis and patients with mutations in genes regulating apoptosis are known to have a poor prognosis.

The application describes the cloning of a gene which encodes a splice variant of the known gene designated H5/PNUTL2/CDCrel–2a/2b and the isolation and characterization of its protein product. The newly identified protein, designated ARTS (*A*poptosis *R*elated *P*rotein in the *T*GF–β *S*ignaling Pathway), is a member of the septin family of proteins. It is localized to mitochondria and translocates to the nucleus where ARTS induces apoptosis in response to TGF–β. ARTS is the first septin shown to be essential for mediating *T*GF–β dependent apoptosis. Antisense ARTS nucleic acids are also contemplated. Because of its role in regulating the sensitivity of cells to TGF–β induced apoptosis ARTS derived products may provide a means for treating conditions where increased TGF–β induced apoptosis is desired (*e.g.,* cancer) and where decreased TGF–β induced apoptosis is desired (*e.g.,* neurodegenerative diseases).

This work has been published in part at Larisch-Bloch S *et al.* ''Selective loss of the transforming growth factor-beta apoptotic signaling pathway in mutant NRP–154 rat prostatic epithelial cells'' Cell Growth Differ 11(1):1–10 (Jan 2000).

Replication Deficient Retroviral Vector System and Methods of Using

WJ Ramsey (NHGRI)

Serial No. 60/101,425 filed 22 Sep 1998; PCT/US99/21393

The technology described and claimed in this application relates to the field of gene therapy. More particularly, the technology described and claimed in the application relates to a method for producing replication deficient, but infectious, retroviral vectors. This method of producing replication deficient retroviral vectors for gene therapy yields virus in high titer and is readily adaptable to large scale production. In the methods described herein a producer cell is transformed with an integrating proviral sequence which includes a pair of retroviral LTRs, a retroviral packaging signal and the gene of interest. A second viral vector, containing trans complementing functions, such as the gag, pol, and env genes, is then used to infect/transform the cells containing the integrated proviral sequence enabling the generation of a replication deficient vector. This second viral vector may be chimeric, *e.g.,* have an adenoviral backbone and retroviral trans complementing functions. Producer cells, which now contain the trans complementing vector and the integrated proviral vector are then cultured to obtain the replication deficient viral vector from the medium.

The PCT application has been published as WO 00/17736 (March 30, 2000). Related technology describing chimeric vectors for gene therapy is also available for licensing. It is described in USSN 09/058,686 filed 10 Apr 1998 (published as WO 98/46778 (Oct. 22, 1998).

Dated: July 19, 2000.

Jack Spiegel,

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

[FR Doc. 00–19150 Filed 7–27–00; 8:45 am]

BILLING CODE 4140–01–P

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