and other device-oriented information. The CDRH Web site may be accessed at *http://www.fda.gov/cdrh*. A search capability for all CDRH guidance documents is available at *http://www.fda.gov/cdrh/guidance.html*. Guidance documents are also available on the Division of Dockets Management Internet site at *http://www.fda.gov/ohrms/dockets*.

V. Comments

Interested persons may submit to the Division of Dockets Management (see **ADDRESSES**), written or electronic comments regarding the guidance at any time. Submit a single copy of electronic comments to *http://www.fda.gov/dockets/ecomments*. Submit two paper copies of any mailed comments, except that individuals may submit one paper copy. Comments are to be identified with the docket number found in brackets in the heading of this document. Comments received may be seen in the Division of Dockets Management between 9 a.m. and 4 p.m., Monday through Friday.

Dated: February 25, 2004.

Beverly Chernaik Rothstein,

Acting Deputy Director for Policy and Regulations, Center for Devices and Radiological Health.

[FR Doc. 04–4982 Filed 3–4–04; 8:45 am]

BILLING CODE 4160–01–S

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 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.

Cloning and Characterization of an Avian Adeno-Associated Virus and Uses Thereof

Ioannis Bossis (NIDCR).

U.S. Provisional Application No. 60/472,066 filed 19 May 2003 (DHHS Reference No. E–105–2003/0–US–01).

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

Currently, adeno-associated virus (AAV) represents the gene therapy vehicle of choice because it has many advantages over current strategies for therapeutic gene insertion. AAV is less pathogenic than other virus types; stably integrates into dividing and non-dividing cells; integrates at a consistent site in the host genome; and shows good specificity towards various cell types for targeted gene delivery.

To date, eight AAV isolates have been isolated and characterized, but new serotypes derived from other animal species may add to the specificity and repertoire of current AAV gene therapy techniques.

This invention describes vectors derived from an avian AAV. These vectors have innate properties related to their origin that may confer them with a unique cellular specificity in targeted human gene therapy. Therefore, vectors derived from this avian AAV are likely to find novel applications for gene therapy in humans and fowl.

This research has been described, in part, in Bossis and Chiorini (2003) J. Virol. 12:6799–6810.

Activation of Recombinant Diphtheria Toxin Fusion Proteins by Specific Proteases Highly Expressed on the Surface of Tumor Cells

Stephen Leppla, Shi-Hui Liu, Manuel Osorio, and Jennifer Avallone (NIDCR).

DHHS Reference No. E–331–2002/0–US–01 filed 06 May 2003.

Licensing Contact: Brenda Hefti; 301/435–4632; *heftib@mail.nih.gov.*

This invention relates to diphtheria toxin fusion proteins comprising a diphtheria toxin (DT) cell-killing component and a cell-binding component such as granulocyte macrophage colony-stimulating factor (GM–CSF), interleukin 2 (IL–2), or epidermal growth factor (EGF). Receptors for the latter three materials are present on many types of cancer cells; therefore, these fusion proteins bind preferentially to these cancer cells. A key feature is that these toxins are altered so as to require activation by a cell-surface protease that is overexpressed on many types of cancers. Examples of such proteases include matrix metalloproteinases and urokinase plasminogen activator. Consequently, these novel cytotoxins kill tumors expressing receptors for either GM–CSF, IL–2, or EGF along with the cell-surface protease. Because killing requires the presence of both a receptor and a cancer-cell enriched protease, and few normal tissues contain both, there is less toxicity to normal cells. Thus, a larger amount of the agent may be used for cancer therapy without inducing side effects. In other words, these cytotoxins have a higher therapeutic index than toxins that are targeted to cells using a single strategy.

Dominant Negative Deletion Mutants of C-Jun and Their Use in the Prevention and Treatment of Cancer

NH Colburn, Z Dong, PH Brown, MJ Birrer (NCI).

U.S. Patent Application No. 08/213,433 filed 10 Mar 94 (DHHS Reference No. E–240–1993/0–US–01).

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

A number of mutants of the c-jun oncogene have been developed, which may be particularly useful in the prevention and treatment of cancer. Numerous studies have shown that tumor promotion is a long-term process that is partially reversible and that requires chronic exposure to a tumor promoter, and that subsequent progression of tumors through invasive and metastatic stages is also a long term process. In recent years, numerous cellular oncogenes have been implicated in the transactivation of genes associated with cellular growth and differentiation. One such cellular oncogene, c-jun, encodes a phosphoprotein that is a component of the dimeric transcriptional activator AP–1 along with c-Fos or other Jun or Fos family proto-oncoproteins. Several genes that may be involved in tumor promotion or progression have been shown to be dependent on AP–1 transactivation, including collagenase and stromelysin (transin). AP–1 inhibiting dominant negative deletion mutants of the c-jun gene have been developed that, when given to a mammal, may prevent or reverse carcinogenesis during early or late stages. For the treatment of cancer, a deletion mutant of the c-jun gene or the protein product may inhibit the elevated AP–1 transactivation that frequently characterizes tumor progression and may consequently prevent or reverse the development or further progression of

tumors. This invention also includes a method for determining whether a tumor promoter induces transformation via a pathway that depends on induction or elevation of AP–1 transcriptional activity and AP–1 target gene expression.

Deazaflavin Compounds and Methods of Use Thereof

Alan Weissman *et al.* (NCI).

U.S. Provisional Application No. 60/447,610 filed 13 Feb 2003 (DHHS Reference No. E–231–2002/0–US–01).

Licensing Contact: Jeffrey Walenta; 301/435–4633; *walentaj@mail.nih.gov.*

Recently, a new strategy for the treatment of cancer was validated by the FDA approval of a small molecule proteasome inhibitor. This treatment strategy, while being efficacious, achieved this result by generally inhibiting all proteasome activity. However, the ubiquitin-mediated process that instructs the proteasome to degrade specific proteins is exquisitely specific to the type of protein degraded. The exact mechanism of how the individual components of the ubiquitin-mediated process regulate the amount of a specific protein present in a cell is just beginning to be elucidated with certainty. Drugs specific to these components can regulate cellular level of important proteins.

This invention is a family of 7-nitro-5-deazaflavin compounds that inhibit MDM2 protein activity in a cell. The MDM2 protein is an E3 ubiquitin ligase that mediates the transfer of ubiquitin to the important tumor suppressor protein p53: p53 will initiate apoptosis in cancer cells. By minimizing ubiquitin transfer to p53—and its subsequent degradation—the 7-nitro-5-deazaflavin compounds can potentially increase the tumor suppressor properties of p53 by maintaining a higher concentration of the important tumor suppressor protein within the cell.

This invention could be an important next generation proteasome inhibitor as it can potentially inhibit the degradation of specific proteasome substrates.

Dated: February 27, 2004.

Steven M. Ferguson,

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

[FR Doc. 04–4915 Filed 3–4–04; 8:45 am]

BILLING CODE 4140–01–P

DEPARTMENT OF HEALTH AND HUMAN SERVICES

National Institutes of Health

National Cancer Institute; Notice of Closed Meeting

Pursuant to section 10(d) of the Federal Advisory Committee Act, as amended (5 U.S.C. appendix 2), notice is hereby given of the following meeting.

The meeting will be closed to the public in accordance with the provisions set forth in sections 552b(c)(4) and 552b(c)(6), title 5 U.S.C., as amended. The grant applications and the discussions could disclose confidential trade secrets or commercial property such as patentable material, and personal information concerning individuals associated with the grant applications, the disclosure of which would constitute a clearly unwarranted invasion of personal privacy.

Name of Committee: National Cancer Institute Initial Review Group; Subcommittee E—Cancer Epidemiology, Prevention & Control.

Date: April 13–14, 2004.

Time: 8 a.m. to 6 p.m.

*Agenda:*To review and evaluate grant applications.

Place: Holiday Inn Select Bethesda, 8120 Wisconsin Avenue, Bethesda, MD 20814.

Contact Person: Mary C. Fletcher, PhD, Scientific Administrator, Research Programs Review Branch, Division of Extramural Activities, National Cancer Institute, National Institutes of Health, 6116 Executive Boulevard, Rm 8115, Bethesda, MD 20892, (301) 496–7413.

Any interested person may file written comments with the committee by forwarding the statement to the Contact Person listed on this notice. The statement should include the name, address, telephone number and when applicable, the business or professional affiliation of the interested person.

(Catalogue of Federal Domestic Assistance Program Nos. 93.392, Cancer Construction; 93.393, Cancer Cause and Prevention Research; 93.394, Cancer Detection and Diagnosis Research; 93.395, Cancer Treatment Research; 93.396, Cancer Biology Research; 93.397, Cancer Centers Support; 93.398, Cancer Research Manpower; 93.399, Cancer Control, National Institutes of Health, HHS)

Dated: February 27, 2004.

LaVerne Y. Stringfield,

Director, Office of Federal Advisory Committee Policy.

[FR Doc. 04–4909 Filed 3–4–04; 8:45 am]

BILLING CODE 4140–01–M

DEPARTMENT OF HEALTH AND HUMAN SERVICES

National Institutes of Health

National Cancer Institute; Notice of Closed Meeting

Pursuant to section 10(d) of the Federal Advisory Committee Act, as amended (5 U.S.C. appendix 2), notice is hereby given of the following meeting.

The meeting will be closed to the public in accordance with the provisions set forth in sections 552b(c)(4) and 552b(c)(6), title 5 U.S.C., as amended. The contract proposals and the discussions could disclose confidential trade secrets or commercial property such as patentable material, and personal information concerning individuals associated with the contract proposals, the disclosure of which would constitute a clearly unwarranted invasion of personal privacy.

Name of Committee: National Cancer Institute Special Emphasis Panel; SBIR Topics 181, 184, 199.

Date: March 24, 2004.

Time: 8:30 a.m. to 5 p.m.

Agenda: To review and evaluate contract proposals.

Place: Bethesda Marriott, 5151 Pooks Hill Road, Bethesda, MD 20814.

Contact Person: C. Michael Kerwin, PhD, MPH, Scientific Review Administrator, Special Review and Logistics Branch, Division of Extramural Activities, National Cancer Institute, National Institutes of Health, 6116 Executive Boulevard, Room 8057, MSC 8329, Bethesda MD 20892–8329, 301–496–7421, *kerwinm@mail.nih.gov.*

(Catalogue of Federal Domestic Assistance Program Nos. 93.392, Cancer Construction; 93.393, Cancer Cause and Prevention Research; 93.394, Cancer Detection and Diagnosis Research; 93.395, Cancer Treatment Research; 93.396, Cancer Biology Research; 93.397, Cancer Centers Support; 93.398, Cancer Research Manpower; 93.399, Cancer Control, National Institutes of Health, HHS)

Dated: February 27, 2004.

LaVerne Y. Stringfield,

Director, Office of Federal Advisory Committee Policy.

[FR Doc. 04–4913 Filed 3–4–04; 8:45 am]

BILLING CODE 4140–01–M

DEPARTMENT OF HEALTH AND HUMAN SERVICES

National Institutes of Health

National Institute on Alcohol Abuse and Alcoholism; Notice of Closed Meetings

Pursuant to section 10(d) of the Federal Advisory Committee Act, as