0262. Attn: Melissa Musotto, CMS–10156;
and,
OMB Human Resources and Housing Branch, Attention: Christopher Martin, New Executive Office Building, Room 10235, Washington, DC 20503.

Dated: June 1, 2005.

Jimmy Wickliffe,

CMS Paperwork Reduction Act Reports Clearance Officer, Office of Strategic Operations and Regulatory Affairs, Regulations Development Group.

[FR Doc. 05–11178 Filed 6–2–05; 8:45 am]

BILLING CODE 4120–03–P

DEPARTMENT OF HEALTH AND HUMAN SERVICES

Food and Drug Administration

[Docket No. 2005M–0005]

Medical Devices Regulated by the Center for Biologics Evaluation and Research; Availability of Safety and Effectiveness Summaries for Premarket Approval Applications

AGENCY: Food and Drug Administration, HHS.

ACTION: Notice.

SUMMARY: The Food and Drug Administration (FDA) is publishing a list of premarket approval applications (PMAs) that have been approved by the Center for Biologics Evaluation and Research (CBER). This list is intended to inform the public of the availability of safety and effectiveness summaries of approved PMAs through the Internet and FDA's Division of Dockets Management.

ADDRESSES: Submit written requests for copies of summaries of safety and effectiveness data to the Division of Dockets Management (HFA–305), Food and Drug Administration, 5630 Fishers Lane, rm. 1061, Rockville, MD 20852. Please include the appropriate docket number as listed in table 1 of this document when submitting a written request. See the **SUPPLEMENTARY INFORMATION** section for electronic access to the summaries of safety and effectiveness data.

FOR FURTHER INFORMATION CONTACT: Nathaniel L. Geary, Center for Biologics Evaluation and Research (HFM–17), Food and Drug Administration, 1401 Rockville Pike, suite 200N, Rockville, MD 20852–1448, 301–827–6210.

SUPPLEMENTARY INFORMATION:

I. Background

In the **Federal Register** of January 30, 1998 (63 FR 4571), FDA published a final rule that revised 21 CFR 814.44(d) and 814.45(d) to discontinue individual publication of PMA approvals and denials in the **Federal Register**, providing instead to post this information on the Internet at *http://www.fda.gov*. In addition, the regulations provide that FDA publish a quarterly list of available safety and effectiveness summaries of PMA approvals and denials that were announced during the quarter. FDA believes that this procedure expedites public notification of these actions because announcements can be placed on the Internet more quickly than they can be published in the **Federal Register**, and FDA believes that the Internet is accessible to more people than the **Federal Register**.

In accordance with section 515(d)(4) and (e)(2) of the Federal Food, Drug, and Cosmetic Act (the act) (21 U.S.C. 360e(d)(4) and (e)(2)), notification of an order approving, denying, or withdrawing approval of a PMA will continue to include a notice of opportunity to request review of the order under section 515(g) of the act. The 30-day period for requesting administrative reconsideration of an FDA action under § 10.33(b) (21 CFR 10.33(b)) for notices announcing approval of a PMA begins on the day the notice is placed on the Internet. Section 10.33(b) provides that FDA may, for good cause, extend this 30-day period. Reconsideration of a denial or withdrawal of approval of a PMA may be sought only by the applicant; in these cases, the 30-day period will begin when the applicant is notified by FDA in writing of its decision.

The following is a list of PMAs approved by CBER for which summaries of safety and effectiveness were placed on the Internet from October 1, 2004, through December 31, 2004. There were no denial actions during the period. The list provides the manufacturer's name, the product's generic name or the trade name, and the approval date.

TABLE 1.—LIST OF SAFETY AND EFFECTIVENESS SUMMARIES FOR APPROVED PMAS MADE AVAILABLE OCTOBER 1, 2004, THROUGH DECEMBER 31, 2004

PMA No./Docket No.	Applicant	Trade Name	Approval Date
BP 040046/02005M–0005	Bio-Rad Laboratories	Multispot HIV–1/HIV–2 Rapid Test	November 12, 2004

II. Electronic Access

Persons with access to the Internet may obtain the documents at *http://www.fda.gov/cber/products.htm*.

Dated: April 11, 2005.

Jesse Goodman,

Director, Center for Biologics Evaluation and Research.

[FR Doc. 05–11072 Filed 6–2–05; 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.

Method of Diagnosing Cancer Using beta-Catenin Splice Variants

Mark J. Roth and Konrad Huppi (NCI); U.S. Provisional Application No. 60/652,154 filed 10 Feb 2005 (DHHS Reference No. E–018–2005/0–US–01); *Licensing Contact:* Susan S. Rucker; (301) 435–4478; *ruckersu@mail.nih.gov.*

This application relates to methods for early detection, diagnosis, and prognosis of cancers and their associated preneoplastic lesions. The methods are useful in evaluating the status of preneoplastic lesions as well as tumor tissue. Because of this, the methods can be used to track the progression and therapeutic response of disease in cell and tissue samples of normal, dysplasia or cancerous epithelium procured by routine cytology, i.e., exfoliated/brush or fine needle aspiration, or surgical methods.

The methods are particularly useful with respect to adenocarcinomas and squamous cell carcinomas. In particular, the methods described and claimed in the application are useful with respect to preneoplasias and carcinomas involving the upper aerodigestive tract.

The methods involve the measurement of levels of one or more pairs of transcripts or the protein products of these pairs of transcripts or the cellular localization of the transcripts or proteins. The primary transcripts or protein products useful in this method are those of the beta-Catenin gene (CTNNB1). In particular, the levels of the 16A and 16B CTNNB1 transcripts or protein products are of importance in carrying out the methods of this patent application. Other gene transcripts or protein products that may be used in conjunction with CTNNB1 16A and 16B to provide additional information are WAF1 (p21) and cMYC.

The methods can be practiced using fresh or frozen cell and/or tissue specimens and techniques such as laser capture microdissection (LCM) RT–PCR.

In addition to licensing, the technology is available for further development through collaborative research opportunities with the inventors.

Method of Diagnosing and Treating Cancer Using beta-Catenin Splice Variants

Mark J. Roth and Konrad Huppi (NCI); U.S. Provisional Application No. 60/667,084 filed 30 Mar 2005 (DHHS Reference No. E–018–2005/1–US–01); *Licensing Contact:* Susan S. Rucker; (301) 435–4478; *ruckersu@mail.nih.gov.*

This application relates to methods for treatment of cancers and preneoplastic lesions. The treatment methods may also be used in conjunction with the diagnostic/prognostic methods disclosed in related provisional patent application 60/652,154 (NIH Ref: E–018–2005/0–US–01).

The methods are particularly useful with respect to adenocarcinomas and squamous cell carcinomas. In particular, the methods described and claimed in the application are useful with respect to preneoplasias and carcinomas involving the upper aerodigestive tract.

The methods employ small interfering RNA molecules (siRNAs) as a means to alter the expression of one or more particular CTNNB1 transcripts. In particular, preferred siRNA molecules alter the expression of the CTNNB1 transcripts 16A and/or 16B. The siRNA molecules may be single-stranded (ss) or double-stranded (ds). The siRNA molecules may be delivered using a construct, which is capable of expressing the siRNA molecule upon delivery to the target cell.

In addition to licensing, the technology is available for further development through collaborative research opportunities with the inventors.

Framework Residue Substituted Humanized COL–1 Antibodies and Their Use

Syed Kashmiri (NCI), Eduardo Padlan (NIDDK), and Jeffrey Schlom (NCI); U.S. Provisional Application No. 60/640,672 filed 30 Dec 2004 (DHHS Reference No. E–339–2004/0–US–01); *Licensing Contact:* Michelle A. Booden; (301) 451–7337; *boodenm@mail.nih.gov.*

Carcinoembryonic antigen (CEA) has been found to be an important marker of colorectal cancer. CEA is expressed in 85 percent of all gastric cancers and may function as a metastatic potentiator of such cancers. In addition, it has been shown that CEA is up regulated when certain cancers are treated with standard chemotherapy drugs. A treatment modality that focuses specifically on CEA could be an effective way of treating many carcinomas, including colorectal, gastric, pancreatic, lung and breast cancers.

The present invention relates to humanized monoclonal antibodies that bind to CEA. Specifically, these antibody variants have amino acid substitutions in the heavy chain framework that reduces the likelihood of human anti-mouse antibodies (HAMA).

The original murine COL–1 antibody has been shown to be reactive to CEA without cross reactivity with other potential antigens of the CEA family: specifically Antigens NCA–1 and normal fecal antigen Ag1. The increased specificity to CEA and reduced human immunogenicity of these COL–1 humanized variants makes these antibodies attractive therapeutic and/or diagnostic compounds.

The COL–1 antibody is described in the following background publications:

(i) Gonzales NR, Padlan EA, De Pascalis R, Schuck P, Schlom J, and Kashmiri SV. SDR grafting of a murine antibody using multiple human germline templates to minmize its immunogenicity. Mol. Immunol. 41(9): 863–872, 2004.

(ii) De Pascalis R, Iwahashi M, Tamura M, Padlan EA, Gonzales NR, Santos AD, Giuliano M, Schuck P, Schlom J, and Kashmiri SV. Grafting of ''abbreviated'' complementarity-determining regions containing specificity-determining residues essential for ligand contact to engineer a less immunogenic humanized monoclonal antibody. J. Immunol. 169: 3076–3084, 2002.

(iii) Gonzales NR, Padlan EA, De Pascalis R, Schuck P, Schlom J, Kashmiri SV. Minimizing immunogenicity of the SDR-grafted humanized antibody CC49 by genetic manipulation of the framework residues. Mol. Immunol. 40:337–349, 2003.

In addition to licensing, the technology is available for further development through collaborative research opportunities with the inventors.

Inhibiting IL–13 Receptor-Expressing Cancer Cells With Anti-IL–13 Receptor Immunotoxin and Alkylating Agents

Raj Puri and Syed Husain (FDA); U.S. Provisional Application No. 60/621,035 filed 20 Oct 2004 (DHHS Reference No. E–302–2003/0–US–01); *Licensing Contact:* Brenda Hefti; (301) 435–4632; *heftib@mail.nih.gov.*

The present invention relates to methods of inhibiting the growth of cancer cells expressing the IL–13 receptor. Most generally, the patent application claims immunotoxins consisting of anti-IL–13 antibodies bound to toxins such as pseudomonas exotoxin or diphtheria toxin, or a cytotoxic fragment thereof, used in combination with alkylating agents. This combination appears to have significant advantages over use of either agent alone in the treatment of malignant gliomas, head and neck cancers, adenocarcinomas of the colon, stomach of skin, and Hodgkin's disease.

Regulation of RNA Stability

Wi Lai *et al.* (NIEHS); U.S. Provisional Application No. 60/451,976 filed 06 Mar

2003 (DHHS Reference No. E–314–2002/0–US–01); PCT Application No. PCT/US04/06703 filed 05 Mar 2004, which published as WO 2004/081179 A2 on 10 Feb 2005 (DHHS Reference No. E–314–2002/0–PCT–02); *Licensing Contact:* Jesse S. Kindra; (301) 435–5559; *kindraj@mail.nih.gov.*

This invention relates to the discovery that tristetraprolin (TTP) can promote the poly(A)RNase (PARN) mediated deadenylation of polyadenylated substrates containing AU-rich elements (AREs). As one aspect of the invention, the inventors have developed a cell free system that may be used for the purposes of assessing the effects of the various system components or their derivatives (*i.e.* AREs, PARN, or TTP) on the deadenylation process or the effects of various test agents on the deadenylation process. Aspects of this work have been published as follows: Lai *et al.*, 2003, Tristetraprolin and Its Family Members Can Promote the Cell-Free Deadenylation of AU-Rich Element-Containing mRNAs by Poly(A) Ribonuclease, MCB 23(11):3798–3812.

This technology is available for licensing on an exclusive or a non-exclusive basis.

In addition to licensing, the technology is available for further development through collaborative research opportunities with the inventors.

Tristetraprolin (TTP) Knockout Mice

Perry Blackshear *et al* (NIEHS).
DHHS Reference No. B–015–1999/0—Research Material.
Licensing Contact: Michelle A. Booden; 301/451–7337; *boodenm@mail.nih.gov.*

National Institutes of Health researchers have developed knockout mice that do not express Tristetraprolin (TTP). TTP is an AU-rich element (ARE) binding protein and the prototype of a family of CCCH zinc finger proteins. AREs were identified as conserved sequences found in the 3' untranslated region (3' UTR) of a variety of transiently expressed genes including early respsonse genes, proto-oncogenes, and other growth regulatory genes. AREs function as instability sequences that target ARE-containing transcripts for rapid mRNA decay. TTP functions by binding directly to the ARE sequence contained in the TNF-alpha mRNA, which destabilizes and mediates rapid decay of the TNF-alpha mRNA. More recent studies demonstrate TTP's ability to downregulate IL–2 gene expression.

TTP knockout mice appear normal at birth but soon develop inflammatory arthritis, dermatitis, cachexia, autoimmunity, and myeloid hyperplasia. Almost all aspects of these phenotypes can be prevented with repeated injections of antibodies to TNF. Moreover, macrophages isolated from these mice exhibit increased production of TNF-alpha and increased amounts of TNF-alpha mRNA.

This transgenic mouse model will be valuable in advancing our understanding of the mechanisms controlling mRNA turnover in immune homeostasis as well as autoimmune diseases. This model will also permit the development of screening assays to elucidate the functions and binding partners for other members of the CCCH zinc finger family as well as compounds capable of inhibiting aberrant TNF-alpha and IL–2 biosynthesis. Lastly, this model will advance understanding of the pathogenetic role for IL–2 and/or TNF in various autoimmune and inflammatory diseases. The mice will be made available on a non-exclusive basis under a Biological Materials License Agreement.

Background scientific detail may be found in Immunol. 2005 Jan 15; 174(2):953–61; Arthritis Res Ther. 2004; 6(6):248–64; and Science. 1998 Aug 14; 281(5379):1001–5.

Dated: May 23, 2005.

Steven M. Ferguson,

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

[FR Doc. 05–11096 Filed 6–2–05; 8:45 am]

BILLING CODE 4140–01–P

DEPARTMENT OF HEALTH AND HUMAN SERVICES

National Institutes of Health

National Center on Minority Health and Health Disparities; 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 Center on Minority Health and Health Disparities Special Emphasis Panel, NCMHD Endowment.

Date: June 27–28, 2005.

Time: 3 p.m. to 5 p.m.

Agenda: To review and evaluate grant applications.

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

Contact Person: Merlyn M. Rodrigues, PhD, MD, Director, Office of Extramural Activities, National Center On Minority Health and Health Disparities, National Institutes of Health, 6707 Democracy Blvd. Suite 800, Bethesda, MD 20894, (301) 402–1366, *rodrigm1@mail.nih.gov.*

Dated: May 25, 2005.

LaVerne Y. Stringfield,

Director, Office of Federal Advisory Committee Policy.

[FR Doc. 05–11095 Filed 6–2–05; 8:45 am]

BILLING CODE 4140–01–M

DEPARTMENT OF HEALTH AND HUMAN SERVICES

National Institutes of Health

National Institute of Diabetes and Digestive and Kidney Disorders; 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 purpose of this meeting is to evaluate requests for preclinical development resources for potential new therapeutics for Type 1 diabetes. The outcome of the evaluation will be a decision whether NIDDK should support the request and make available contract resources for development of the potential therapeutic to improve the treatment or prevent the development of Type 1 diabetes and its complications. The research proposals and the discussions could disclose confidential trade secrets or commercial property such as patentable material, and personal information concerning individuals associated with the proposed research projects, the disclosure of which would constitute a clearly unwarranted invasion of personal privacy.

Name of Committee: National Institute of Diabetes and Digestive and Kidney Disorders Special Emphasis Panel; Type 1 Diabetes—Rapid Access to Intervention Development.

Date: June 21, 2005.

Time: 3 p.m.–4 p.m.

Agenda: To evaluate requests for preclinical development resources for