for public review in the Division of Dockets Management (see ADDRESSES) between 9 a.m. and 4 p.m., Monday through Friday.

Dated: September 26, 2012.

Leslie Kux, 
Assistant Commissioner for Policy.

[FR Doc. 2012–24330 Filed 10–2–12; 8:45 am]
BILLING CODE 4160–01–P

DEPARTMENT OF HEALTH AND HUMAN SERVICES

Food and Drug Administration

Notice.

With regard to the withdrawal of approval of New Animal Drug Applications; Butorphanol; Doxapram; Triamcinolone; Tylosin

AGENCY: Food and Drug Administration, HHS.

ACTION: Notice.

SUMMARY: The Food and Drug Administration (FDA) is withdrawing approval of a new animal drug application (NADA) and three abbreviated new animal drug applications (ANADAs) at the sponsors’ request because the products are no longer manufactured or marketed.

DATES: Withdrawal of approval is effective October 15, 2012.

FOR FURTHER INFORMATION CONTACT: David Alterman, Center for Veterinary Medicine (HFV–212), Food and Drug Administration, 7519 Standish Pl., Rockville, MD 20855, 240–453–6643, email: david.alterman@fda.hhs.gov.

SUPPLEMENTARY INFORMATION: The following sponsors have requested that FDA withdraw approval of the NADA and ANADAs listed in table 1 of this document because the products are no longer manufactured or marketed.

Therefore, under authority delegated to the Commissioner of Food and Drugs and redelegated to the Center for Veterinary Medicine, and in accordance with § 514.116 Notice of withdrawal of approval of application (21 CFR 514.116), notice is given that approval of NADA 100–556 and ANADAs 200–435, 200–446, and 200–459, and all supplements and amendments thereto, is hereby withdrawn, effective October 15, 2012.

Elsewhere in this issue of the Federal Register, FDA is amending the animal drug regulations to reflect the voluntary withdrawal of approval of these applications.

Dated: September 27, 2012.

Bernadette Dunham, 
Director, Center for Veterinary Medicine.

[FR Doc. 2012–24330 Filed 10–2–12; 8:45 am]
BILLING CODE 4160–01–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.

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.

FOR FURTHER INFORMATION CONTACT: 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.

A Novel Immortalized Human Adrenal Cell Line With Inactive Protein Kinase A for Studies on cAMP Signaling and Endocrine Tumorigenesis

Description of Technology: The first known immortalized cell line with a naturally-occurring inactivating mutation in PRKAR1A, the regulatory subunit type 1A (R1alpha) of protein kinase A (PKA), which is associated with tumor formation.

PKA isoform balance is critical for the control of cAMP signaling and related cell cycle and proliferation changes. Aberrant CAMP signaling has been linked to adrenocortical and other, mostly endocrine, tumors. Inactivating mutations in the PRKAR1A gene are known causes of the Carney Complex—an autosomal dominant multiple neoplasia syndrome associated with skin, heart, and other myxomas and a variety of endocrine tumors.

Potential Commercial Applications:

• Studies on multiple tumor formation associated with Carney Complex.

• Characterization of cAMP-mediated mechanisms of endocrine tumor formation.

• Studies of a large variety of cAMP-mediated processes in normal physiology and disease.

Competitive Advantages: First known immortalized cell line with a naturally-occurring inactivating mutation in the PRKAR1A gene.

Development Stage: In vitro data available.

Inventor: Constantine A. Stratakis (NICHD).

Publication: Nesterova M, et al. An immortalized human cell line bearing a PRKAR1A-inactivating mutation: effects of overexpression of the wild-type

<table>
<thead>
<tr>
<th>NADA/ANADA No.</th>
<th>Trade name (drug)</th>
<th>Applicant</th>
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**Licensing Contact:** Patrick McCue, Ph.D.; 301–435–5560; mccuepat@mail.nih.gov.

**Collaborative Research Opportunity:** The Eunice Kennedy Shriver National Institute of Child Health and Human Development is seeking statements of capability or interest from parties interested in collaborative research to further develop, evaluate or commercialize this technology. For collaboration opportunities, please contact Joseph Conrad III, Ph.D. at jmconrad@mail.nih.gov.

**Modulation of Regulatory T-Cell and B-Cell Lymphocytes for the Treatment of Autoimmune and Other Disease Indications**

**Description of Technology:** A method of modulating the immune response by affecting the activity of the regulatory lymphocytes through targeting of the Hepatitis A Virus receptor 1 (HAVCR1) receptor. This methodology can be developed for the treatment of autoimmune diseases, allergies, prevention of transplant rejection, and incorporated into therapeutic strategies for cancer.

Regulatory lymphocytes, such as regulatory T-cells (Tregs) and B-cells (Bregs), play a significant role in suppressing and controlling immune responses to antigens, including allergens and self-antigens that induce autoimmune diseases. The Tregs and Bregs also control the immune responses to microbial pathogens thereby limiting excessive damage to tissue. HAVCR1 is expressed on these regulatory lymphocytes and functions as a master regulator of these cells.

**Potential Commercial Applications:**
- Treatment of Autoimmune Diseases.
- Treatment of Allergies.
- Prevention of Rejection of Allogenic Transplants.
- Cancer Therapy.
- Immunotherapies.
- Stimulate Response to Vaccines (adjuvant).

**Competitive Advantages:** Can be used to target multiple disease states.

**Development Stage:**
- Early-stage.
- Pre-clinical.
- In vitro data available.

**Inventors:** Gerardo Kaplan, Mohanraj Manangeeswaran, Jerome Jacques, Krishnamurthy Konduru (all of FDA).


**Licensing Contact:** Kevin W. Chang, Ph.D.; 301–435–5018; changke@mail.nih.gov.

**Collaborative Research Opportunity:** The Center for Biologics Evaluation and Research, Laboratory of Emerging Pathogens, is seeking statements of capability or interest from parties interested in collaborative research to further develop, evaluate or commercialize targeting of HAVCR1 to control Treg and Breg function in human diseases. For collaboration opportunities, please contact Gerardo Kaplan at gerardo.kaplan@fda.hhs.gov.

**A Method To Expand a Population of Regulatory T Cells Optimal for the Treatment of Autoimmune Diseases**

**Description of Technology:** The transfusion of regulatory T cells (Tregs) has been used in the clinic to successfully prevent graft vs. host disease and is currently being evaluated in the treatment of other autoimmune diseases, such as organ graft rejection, type 1 diabetes and multiple sclerosis. Prior to transfusion, adoptive regulatory T cell transfer requires the expansion of regulatory T cells in culture; this results in a mixed population of regulatory T cells that limits the effectiveness of the transferred cells.

Scientists at the NIH have developed a method that promotes the expansion of regulatory T cells that are longer lived, more stable, and more suppressive of the autoimmune response. By supplementing T cell cultures with DNA oligonucleotides, the inventors were able to enrich the regulatory T cell population that enhanced the suppression of the autoimmune response. This method has the potential to more effectively generate regulatory T cells for the treatment of autoimmune diseases.

**Potential Commercial Applications:** Treatment of autoimmune diseases, such as Graft vs. Host Disease, Organ Graft Rejection Type 1 Diabetes, Multiple Sclerosis.

**Competitive Advantages:**
- More effective therapy when compared to traditional T cell expansion methods.
- Expansion method is inexpensive and similar to current methods.

**Development Stage:** In vitro data available.

**Inventors:** Yong Chan Kim and Ethan M. Shevach (NAID).


**Licensing Contact:** John Stansberry, Ph.D.; 301–435–5236; stansbej@mail.nih.gov.

**Peptides for Treatment of Tumor Necrosis Factor Alpha Mediated Inflammatory Disease**

**Description of Technology:** Tumor Necrosis Factor alpha (TNF-alpha) is a multifunctional cytokine that mediates inflammation, immune regulation, and cellular proliferation. This cytokine is converted to its active form by TNF-alpha converting enzyme (TACE). Pathological increases in TNF-alpha activity have been associated with a wide variety of inflammatory diseases, including inflammatory bowel disease, rheumatoid arthritis, and cancer. Inhibiting the conversion of TNF-alpha to its active form by inhibiting TACE represents a potential treatment for these diseases.

The current technology provides peptides, derived from an N-terminal fragment of the TACE protein, that inhibit TACE activity. Also described are methods of using these peptides to lower levels of active TNF-alpha. These peptides could be used as a treatment for TNF-alpha-mediated inflammatory diseases.

**Potential Commercial Applications:** Treatment of TNF-alpha mediated inflammatory diseases.

**Competitive Advantages:** Inhibition of TACE activity represents a novel mechanism to treat inflammatory disease.

**Development Stage:**
- Early-stage.
- In vitro data available.

**Inventors:** Stewart J. Levine et al. (NHLBI).

Inventors: Dimiter Dimitrov and Weizao Chen (NCI).

Publications:

In vivo data available (animal).

In vitro data available.

DEPARTMENT OF HEALTH AND HUMAN SERVICES

National Institutes of Health

National Institute of General Medical Sciences Notice of Closed Meeting

Pursuant to section 10(d) of the Federal Advisory Committee Act, as amended (5 U.S.C. App.), 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 Institute of General Medical Sciences Special Emphasis Panel; Phase III Antibiotic Clinical Trials.

Date: November 1, 2012.

Time: 11 a.m. to 12 p.m.

Agenda: To review and evaluate grant applications.

Place: National Institutes of Health, Natcher Building, 45 Center Drive, Room 3An18K, Bethesda, MD 20892.

Contact Person: Brian R. Fike, Ph.D., Scientific Review Officer, Office of Scientific Review, National Institute of General Medical Sciences, National Institutes of Health, 45 Center Drive, Room 3An18, Bethesda, MD 20892, 301–594–3097, pikbr@mail.nih.gov. (Catalogue of Federal Domestic Assistance Program Nos. 93.375, Minority Biomedical Research Support; 93.821, Cell Biology and Biophysics Research; 93.859, Pharmacology, Physiology, and Biological Chemistry Research; 93.862, Genetics and Developmental Biology Research; 93.88, Minority Access to Research Careers; 93.96, Special Minority Initiatives, National Institutes of Health, HHS)

Dated: September 27, 2012.

Melanie J. Gray, Program Analyst, Office of Federal Advisory Committee Policy.

[FR Doc. 2012–24253 Filed 10–2–12; 8:45 am]

BILLING CODE 4140–01–P

DEPARTMENT OF HEALTH AND HUMAN SERVICES

National Institutes of Health

Eunice Kennedy Shriver National Institute of Child Health & Human Development; Notice of Closed Meeting

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