I. Background

Section 512 of the Federal Food, Drug and Cosmetic Act (21 U.S.C. 360b) establishes the requirements for new animal drug approval. FDA regulations in part 514 (21 CFR part 514) specify the information you must submit as part of your new animal drug application (NADA) and the proper format for the NADA submission. As part of your NADA submission, you must include a ''detailed description of the collection of samples and the analytical procedures to which they are subjected'' (§ 514.1(b)(5)(vii). This should include a description of practicable methods of analysis which have adequate sensitivity to determine the amount of the new animal drug in the final dosage form (§ 514.1(b)(5)(vii)(*a*). This draft guidance provides recommendations for describing methods for analyzing new animal drugs in Type C medicated feeds. This draft guidance applies to instrumental methods only (e.g., High Pressure Liquid Chromatography, Gas Chromatography. For guidance on other methods (e.g., microbiological methods) you should contact the center.

II. Paperwork Reduction Act of 1995

This draft guidance refers to previously approved collections of information found in FDA regulations. These collections of information are subject to review by the Office of Management and Budget (OMB) under the Paperwork Reduction Act of 1995 (44 U.S.C. 3501–3520). The collections of information in § 514.1 have been approved under OMB control numbers 0910–0032 and 0910–0154.

III. Significance of Guidance

This Level 1 draft guidance is being issued consistent with FDA's good guidance practices regulation (21 CFR 10.115). This draft guidance, when finalized, will represent the agency's current thinking on the topic. It does not create or confer any rights for or on any person and does not operate to bind FDA or the public. An alternate method may be used as long as it satisfies the requirements of applicable statutes and regulations.

IV. Comments

This draft guidance is being distributed for comment purposes only and is not intended for implementation at this time. Interested persons may submit to the Division of Dockets Management (see **ADDRESSES**) written or electronic comments regarding this draft guidance. Submit a single copy of electronic comments or 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. Received comments may be seen in the Division of Dockets Management between 9 a.m. and 4 p.m., Monday through Friday.

V. Electronic Access

Copies of the draft guidance document entitled ''Analytical Methods Description for Type C Medicated Feeds'' may be obtained from the CVM Home Page (*http://www.fda.gov/cvm*) and from the Division of Dockets Management Web site (*http://www.fda.gov/ohrms/dockets/default.htm*).

Dated: June 21, 2006.

Jeffrey Shuren,

Assistant Commissioner for Policy.

[FR Doc. 06–5860 Filed 6–27–06; 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, 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.

Agonist Epitopes for Renal Cell Carcinoma

Description of Technology: Approximately 30,000 patients are diagnosed with renal cell carcinoma (RCC) each year in the United States, and an estimated 12,000 patients die of this disease. Most patients are diagnosed with advanced local disease or metastatic disease. Metastatic RCC carries a poor prognosis with median survivals in the range of 10–12 months. Drugs that inhibit VEGF receptor tyrosine kinases such as Sorafenib and Sunitinib have recently been approved by the FDA to treat metastatic RCC. Although a significant percentage of patients will achieve a partial response or disease stabilization with these agents, complete responses are rare and disease progression eventually ensues. RCC is unusual among solid tumors as it appears to be susceptible to immunotherapy. Cytokines such as IL–2 and IFN-alpha nonspecifically stimulate the immune system resulting in disease regression. Unfortunately, these drugs achieve success in only a minority (15–20%) of the metastatic RCC patient population. Therefore, new methods are needed to improve on immune-based therapies and expand the curative potential of therapies for patients with RCC.

The present invention discloses peptides and antigen epitopes specific for RCC for use in the diagnosis, vaccination, or adoptive infusion of antigen specific T cells to treat patients with metastatic RCC. The immunogenic peptide, which binds to the HLA–A11 epitope, was identified in a patient with metastatic RCC that under went an investigational allogeneic hematopoietic stem cell transplant. Cancer regression occurred post-transplant consistent with a graft-vs-tumor effect. A T-cell line, expanded from the patient's blood cells at the time of tumor regression, was isolated and subsequently shown to kill the patients RCC cells in vitro. Expression and sequencing studies revealed that the patient's T-cells recognize an antigen epitope derived from a human endogenous retrovirus (HERV). Further, pre-clinical studies using quantitative real-time PCR found that this HERV was expressed in eight of 14 RCC tumor cell lines with no HERV expression in patient fibroblasts, hematopoietic cells or in c-DNAs analyzed from 48 different normal tissues. Plans are underway to investigate the immunogenic potential of this peptide to induce expansion of T-cells that are cytotoxic to RCC cells in vitro and in pre-clinical animal models.

Inventors: Richard W. Childs, *et al.* (NHLBI).

Publications: Details of the invention are published in:

1. I. Delgado-Espinoza, *et al.*, ''Nonmyeloablative transplantation for solid tumors: A new frontier for allogeneic immunotherapy,'' *Expert Rev Anticancer Ther.* 2004 Oct;4(5):865–75.

2. Y. Takahashi, *et al.*, ''Nonmyeloablative transplantation: An allogeneic-based immunotherapy for renal cell carcinoma,'' *Clin Cancer Res.* 2004 Sep 15;10(18 Pt 2):6353S–9S.

3. R.W. Childs, *et al.*, ''Regression of Metastatic Renal-Cell Carcinoma after Nonmyeloablative Allogeneic Peripheral-Blood Stem-Cell Transplantation,'' *N Engl J Med.* 2000 Sep 14;343:750–758.

4. Marco Bregni, Naoto T. Ueno, and Richard Childs. Meeting Report: The Second International Meeting on Allogeneic Transplantation in Solid Tumors (ATST). Bone Marrow Transplantation (Submitted 2006).

Patent Status: U.S. Provisional Application No. 60/783,350 filed 17 Mar 2005 (HHS Reference No. E–122–2006/0–US–01).

Licensing Status: Available for non-exclusive or exclusive licensing.

Licensing Contact: Michelle A. Booden, PhD; 301/451–7337; *boodenm@mail.nih.gov*.

Collaborative Research Opportunity: The Hematology Branch of the NHLBI is seeking statements of capability or interest from parties interested in collaborative research to further develop, evaluate, or commercialize therapeutic treatment approaches targeting this novel RCC antigen. Please contact Dr. Richard Childs at 301/594–8008 or *childsr@nhlbi.nih.gov* for more information.

Immunogenic Peptides and Methods of Use for Treating Prostrate and Uterine Cancers

Description of Technology: Cancer of the prostate is the most commonly diagnosed cancer in men and the second leading cause of cancer death in men. Despite the use of standard therapy, including surgery, radiotherapy, chemotherapy, and/or hormonal therapy more than 30,000 men will die from prostate cancer. Moreover, current therapy has limited success against metastatic androgen insensitive prostate cancer. A potential systemic treatment for all subclasses of prostate cancer is immunotherapy, either alone or in combination with standard radiation or chemotherapy.

Prostate Antigen Gene-4 (PAGE4) is an X chromosome-linked cancer-testis antigen that is highly expressed in prostate and uterine cancers. To this end, Drs. Jeffery Schlom, Kwong Tsang, and Ira Pastan have identified and characterized novel PAGE4 cytotoxic T-cell lymphocyte (CTL) epitopes and enhanced agonist epitopes. Preclinical studies performed by Dr. Schlom and colleagues indicate that the PAGE4 agonist epitopes bound HLA-A2 molecules at lower peptide concentrations, form more stable peptide HLA–A2 complexes, induce higher levels of production of INFγ, Granzyme B, TNFα, IL-2, and lymphotactin by PAGE4 specific T-cell lines, and T-cell lines generated against the agonist peptide were more efficient at lysing human tumor cells expressing native PAGE4. Thus, these agonist epitopes of PAGE4 could be incorporated into immunotherapy protocols, and may constitute an alternative and/or additional approach for the treatment of PAGE4 expressing prostate and uterine cancers.

Development Status: The Laboratory of Tumor Immunobiology plans to initiate clinical studies utilizing this technology and collaborative opportunities may be available.

Inventors: Jeffrey Schlom, Kwong-Yok Tsang, Ira Pastan (NCI).

Publications: Publications which may provide background information for this technology include:

1. C. Iavarone, *et al.*, ''PAGE4 is a cytoplasmic protein that is expressed in normal prostate and in prostate cancers,'' *Mol Cancer Ther.* 2002 Mar;1(5):329–335.

2. L. Prikler, *et al.*, ''Adaptive immunotherapy of the advanced prostate cancer—cancer testis antigen (CTA) as possible target antigens,'' *Aktuelle Urol.* 2004 Aug;35(4):326–330. [article in German].

Patent Status: U.S. Provisional Application No. 60/776,506 filed 24 Feb 2006 (HHS Reference No. E–104–2006/0–US–01).

Licensing Status: Available for non-exclusive or exclusive licensing.

Licensing Contact: Michelle A. Booden, PhD; 301/451–7337; *boodenm@mail.nih.gov*.

Collaborative Research Opportunity: The NCI Laboratory of Tumor Immunobiology is seeking statements of capability or interest from parties interested in collaborative research to further develop, evaluate, or commercialize cancer vaccine technology encompassing PAGE4. Please contact Denise M. Crooks, PhD, at 301/451–3943 and/or *crooksd@mail.nih.gov* for more information.

Novel Human IGF–1 Specific IGF–I and IGF–II Cross-Reactive Human Monoclonal Antibodies as Potential Anti-Tumor Agents

Description of Technology: Cancer is one of the leading causes of death in United States and it is estimated that there will be approximately 600,000 deaths caused by cancer in 2006. A major drawback of the current chemotherapy-based therapeutics is the cytotoxic side-effects associated with them. Thus there is a dire need to develop new therapeutic strategies with fewer side-effects. Monoclonal antibody-based therapies have taken a lead among the new cancer therapeutic approaches.

The type 1 insulin-like growth factor (IGF) receptor (IGF1R) is over-expressed by many tumors and mediates proliferation, motility, and protection from apoptosis. Agents that inhibit IGF1R expression or function can potentially block tumor growth and metastasis. Its major ligands, IGF–I, and IGF–II are over-expressed by multiple tumor types. Previous studies indicate that inhibition of IGF–I, and/or IGF–II binding to its cognizant receptor negatively modulates signal transduction through the IGF pathway and concomitant cell proliferation and growth. Therefore, use of humanized or fully human antibodies against IGFs represents a valid approach to inhibit tumor growth.

The present invention discloses the identification and characterization of three (3) novel fully human monoclonal antibodies designated m705, m706, and m708, which are specific for insulin-like growth factor (IGF)–I. Two (2) of the three (3) antibodies, m705 and m706 are specific for IGF–I and do not cross react with IGF–II and insulin while, m708 cross reacts with IGF–II. These antibodies can be used to prevent binding of IGF–I to its concomitant receptor IGFIR, consequently, modulating diseases such as cancer. Additional embodiments describe methods for treating various human diseases associated with aberrant cell growth and motility including breast, prostate, and leukemia carcinomas. Thus, these novel IGF–I antibodies may provide a therapeutic intervention for multiple carcinomas.

Development Status: The technology is in the pre-clinical stage; animal studies are currently under way.

Inventors: Dimiter S. Dimitrov and Zhongyu Zhu (NCI).

Publications:

1. A manuscript from the IGF–I work is in preparation (Copy can be provided with Confidential Disclosure Agreement).

2. Y. Feng, Z. Zhu, X. Xiao, V. Choudhry, J.C. Barrett, D.S. Dimitrov, ''Novel human monoclonal antibodies to insulin-like growth factor (IGF)–II that potently inhibit the IGF receptor type I signal transduction function,'' *Mol Cancer Ther.* 2006 Jan; 5 (1):114–120.

Patent Status: U.S. Provisional Patent Application filed 07 Apr 2006 (HHS Reference No. E–336–2005/0–US–01).

Licensing Status: This technology is available for licensing under an exclusive or non-exclusive patent license.

Licensing Contact: Michelle A. Booden, PhD; 301/451–7337; *boodenm@mail.nih.gov*.

Collaborative Research Opportunity: The NCI Center for Cancer Research Nanobiology Program is seeking statements of capability or interest from parties interested in collaborative research to further develop, evaluate, or commercialize monoclonal antibodies to treat human diseases. Please contact Melissa Maderia at *maderiam@mail.nih.gov* or by phone at (301) 846–5465 for more information.

Immortal Human Prostate Epithelial Cell Cultures as a Prostate Cancer Model

Description of Technology: The National Institutes of Health has multiple immortalized, malignant, human, adult prostate epithelial cell lines available for license. They are useful as models in epithelial cell oncogenesis studies and in the diagnosis and treatment of prostate cancer.

The cell lines were generated from primary adenocarcinomas of the prostate. Long-term cultures were established by immortalizing cells with human papillomavirus (HPV) transforming proteins. The cultures were characterized and single-cell clones with unique genetic characteristics were selected based on allelic loss of heterozygosity (LOH). Tissue-matched normal cell lines are available also, useful for the appropriate controls.

The invention also encompasses polyclonal and monoclonal antibodies directed to the cell lines, which may be useful as immunotherapeutics.

Applications: (1) Screening tool to identify novel genes unique to or overexpressed in prostate cancer; (2) Raising of prostate cancer-reactive antibodies, useful as immunotherapeutics or diagnostics; (3) Screen for compounds that kill tumor cells and represent potential therapeutic agents; (4) Identification of prostate cancer antigens to develop recombinant prostate cancer vaccines.

Inventors: Susan L. Topalian, W. Marston Linehan, Robert K. Bright, Cathy D. Vocke (NCI).

Publication: R.K. Bright, *et al.*, ''Generation and genetic characterization of immortal human prostate epithelial cell lines derived from primary cancer specimens,'' *Cancer Res.* 1997 Mar 5;57(5):995–1002.

Patent Status: U.S. Patent 6,982,168 issued on 07 May 2003 (HHS Reference No. E–053–1996/0–US–03).

Licensing Status: Available for non-exclusive internal use and biological material license.

Licensing Contact: Michelle A. Booden, PhD; 301/451–7337; *boodenm@mail.nih.gov*.

Collaborative Research Opportunity: The NCI Center for Cancer Research, Surgery Branch, is seeking statements of capability or interest from parties interested in collaborative research to further develop, evaluate, or commercialize this technology. Please contact Brian W. Bailey, PhD, at 301/451–2158 or *bbailey@mail.nih.gov* for more information.

Dated: June 21, 2006.

David R. Sadowski,

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

[FR Doc. 06–5867 Filed 6–27–06; 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.

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Treatment of Inflammatory Bowel Disease (IBD) Using NF–KB Decoy Polynucleotides

Warren Strober (NIAID), Ivan Fuss (NIAID), Atsushi Kitani (NIAID), and Stefan Fichtner-Feigl (NIAID)

U.S. Patent Application No. 11/125,919 filed 10 May 2005 (HHS Reference No. E–108–2005/0–US–01); PCT International Application filed 10 May 2006 (HHS Reference No. E–108–2005/0–PCT–02)

Licensing Contact: Susan Carson, D. Phil; 301/435–5020; *carsonsu@mail.nih.gov*.

Inflammatory Bowel Diseases (IBDs; Crohn's disease and ulcerative colitis) are chronic inflammatory disorders affecting almost 1 million people in the developed world at an estimated annual cost of one billion dollars in lost work days. Current treatments include corticosteroids, 5-aminosalicylates and immunomodulators but novel and more effective therapies without adverse side effects continue to be needed. NIH researchers have previously shown that a variety of immunomodulators affecting the Th1 and Th2 T cell responses which underlie Inflammatory Bowel Diseases can be used to treat IBD disease models and have now extended this work by inhibiting NF–KB transcriptional activity in a variety of animal models using decoy oligodeoxynucleotides (decoy ODNs).

Dr. Strober and colleagues at the National Institute of Allergy and Infectious Diseases (NIAID) have shown that intrarectal (i.r.) or intraperitoneal (i.p.) administration of decoy ODNs encapsulated in a viral envelope (HVJ–E) prevented and treated a model of acute trinitrobenzene sulfonic acid-induced (TNBS-induced) colitis, a model for Crohn's disease, as assessed by clinical course and the effect on Th1 cytokine production. NF–KB decoy ODNs were also shown to be an effective treatment of a model of chronic TNBS-colitis, inhibiting both the production of IL–23/Il–17 and the development of fibrosis that characterizes this model. Treatment of TNBS-induced inflammation by i.r. administration of NF–KB decoy ODNs did not inhibit NF–KB in extraintestinal organs and resulted in CD4+ T cell apoptosis, suggesting that such treatment is highly focused and durable. Additionally, NF–KB decoy ODNs also prevented and treated oxazolone-colitis, a mouse model for ulcerative colitis, and thus affected a Th2-mediated inflammatory process. In each case, decoy administration led to inflammation clearing effects, suggesting a therapeutic potency