DEPARTMENT OF HEALTH AND HUMAN SERVICES

Food and Drug Administration

[Docket No. FDA–2009–N–0163]

Agency Information Collection Activities; Announcement of Office of Management and Budget Approval; Draft Guidance, Emergency Use Authorization of Medical Products

AGENCY: Food and Drug Administration, HHS.

ACTION: Notice.

SUMMARY: The Food and Drug Administration (FDA) is announcing that a collection of information entitled "Draft Guidance, Emergency Use Authorization of Medical Products" has been approved by the Office of Management and Budget (OMB) under the Paperwork Reduction Act of 1995.

FOR FURTHER INFORMATION CONTACT: Jonna Capezzuto, Office of Information Management, Food and Drug Administration, 1350 Piccard Dr., PI50–400B, Rockville, MD 20850, 301–796–3794, e-mail: *Jonnalynn.capezzuto@fda.hhs.gov.*

SUPPLEMENTARY INFORMATION: In the **Federal Register** of October 6, 2009 (74 FR 51285), the Agency announced that the proposed information collection had been submitted to OMB for review and clearance under 44 U.S.C. 3507. An Agency may not conduct or sponsor, and a person is not required to respond to, a collection of information unless it displays a currently valid OMB control number. OMB has now approved the information collection and has assigned OMB control number 0910–0595. The approval expires on January 31, 2013. A copy of the supporting statement for this information collection is available on the Internet at *http://www.reginfo.gov/public/do/PRAMain.*

Dated: October 21, 2010.

Leslie Kux,

Acting Assistant Commissioner for Policy.

[FR Doc. 2010–27160 Filed 10–26–10; 8:45 am]

BILLING CODE 4160–01–P

DEPARTMENT OF HEALTH AND HUMAN SERVICES

Food and Drug Administration

[Docket No. FDA–2010–N–0121]

Agency Information Collection Activities; Announcement of Office of Management and Budget Approval; The Mammography Quality Standards Act Requirements

AGENCY: Food and Drug Administration, HHS.

ACTION: Notice.

SUMMARY: The Food and Drug Administration (FDA) is announcing that a collection of information entitled "The Mammography Quality Standards Act Requirements" has been approved by the Office of Management and Budget (OMB) under the Paperwork Reduction Act of 1995.

FOR FURTHER INFORMATION CONTACT: Daniel Gittleson, Office of Information Management, Food and Drug Administration, 1350 Piccard Dr., PI50–400B, Rockville, MD 20850, 301–796–5156, e-mail: *Daniel.Gittleson@fda.hhs.gov.*

SUPPLEMENTARY INFORMATION: In the **Federal Register** of June 15, 2010 (75 FR 33811), the Agency announced that the proposed information collection had been submitted to OMB for review and clearance under 44 U.S.C. 3507. An Agency may not conduct or sponsor, and a person is not required to respond to, a collection of information unless it displays a currently valid OMB control number. OMB has now approved the information collection and has assigned OMB control number 0910–0309. The approval expires on October 31, 2013. A copy of the supporting statement for this information collection is available on the Internet at *http://www.reginfo.gov/public/do/PRAMain.*

Dated: October 21, 2010.

Leslie Kux,

Acting Assistant Commissioner for Policy.

[FR Doc. 2010–27159 Filed 10–26–10; 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.

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.

Immunotoxin for the Treatment of Neuroblastoma Relapse

Description of Technology: Immunotoxins are proteins which have two distinct domains: (1) An antibody or antibody binding fragment which is capable of recognizing a single specific cell surface protein and (2) a toxin domain which is capable of inducing cell death. Immunotoxins are currently being pursued as therapeutics because they specifically kill diseased cells while leaving essential, healthy cells alone. This increases the effectiveness of the therapy while reducing the appearance of side-effects. A particular immunotoxin that is being studied in clinical trials consists of an anti-CD22 antibody binding fragment and a mutated *Pseudomonas* exotoxin A. Although this immunotoxin is being explored primarily as a treatment for hematological malignancies, it can be used to treat any condition where CD22 is overexpressed on the cell membrane of diseased cells.

Neuroblastomas are malignant cancers that start in nerve tissue and primarily affect infants and children. Although frontline treatments for neuroblastoma are often effective, relapse frequently occurs in high risk cases. The most common form of relapse in neuroblastoma patients is caused by Neuroblastoma tumor initiating cells (NB–TIC). Therefore, if NB–TIC could be eliminated, high risk neuroblastoma patients could have a therapeutic option for preventing a relapse.

This invention concerns the discovery that NB–TIC expresses CD22. As a result, NB–TIC are susceptible to treatment with an anti-CD22 immunotoxin. By combining frontline

neuroblastoma treatments with anti-CD22 immunotoxins, both the primary neuroblastoma and cells capable of initiating a relapse can be eliminated. As a result, even high risk neuroblastoma patients should have an increased chance of surviving neuroblastoma.

Application: Treatment and prevention of neuroblastoma relapse.

Advantages:

• Increased therapeutic effectiveness with decreased non-specific killing of essential, healthy cells.

• Neuroblastoma relapse commonly begins in the bone marrow, an environment which is accessible to immunotoxins.

• Combined treatment addresses both the tumor and the cause of relapse, leading to more efficient treatments than frontline therapeutics alone.

Development Status: Preclinical stage of development for treatment of neuroblastoma relapse; immunotoxins have clinical data associated with treatment of hematological malignancies.

Inventors: Thiele (NCI) *et al.*

Patent Status: U.S. provisional application 61/356,202 (E–204–2010/0–US–01).

For more information, see:

• U.S. Patent 7,355,012—"Mutated Anti-CD22 Antibodies with Increased Affinity to CD22—Expressing Leukemia Cells".

• PCT Patent Application WO 2007/016150—"Mutated Pseudomonas Exotoxins with Reduced Antigenicity".

• PCT Patent Application WO 2009/032954—"Deletions in Domain II of Pseudomonas Exotoxin A That Reduce Non-Specific Toxicity".

Licensing Status: Available for licensing.

Licensing Contact: David A. Lambertson, PhD; 301–435–4632; *lambertsond@mail.nih.gov.*

Collaborative Research Opportunity: The Center for Cancer Research, Pediatric Oncology Branch and Laboratory of Molecular Biology, is seeking statements of capability or interest from parties interested in collaborative research to further develop, evaluate, or commercialize recombinant anti-CD22 immunotoxins for the treatment of neuroblastoma. Please contact John Hewes, Ph.D. at 301–435–3121 or *hewesj@mail.nih.gov* for more information.

Mouse Model of Thyroid Cancer

Description of Technology: This technology describes a mouse model of thyroid cancer where the phosphatidylinositol 3-kinase (PI3K)–AKT/protein kinase B-signaling pathway is overactivated. These mice have a knock-in dominantly negative mutant thyroid hormone receptor β gene (TRβPV mutant) that spontaneously develops thyroid cancer and distant metastasis similar to human follicular thyroid cancer. The thyroids of TRβPV mice exhibit extensive hyperplasia, which progresses to capsular invasion, vascular invasion, anaplasia, and ultimately, metastasis to distant organs. Consequently, this mouse model could be used as a preclinical model to understand genetic changes during cancer development and to identify potential molecular targets for the diagnosis, prevention, and treatment of cancer. For example, the inventors have used the TRβPV mice to show that the peroxisome proliferator-activated receptor γ (PPARγ) could function as a tumor suppressor *in vivo* and that the activation of the PI3K–AKT signaling contributes to thyroid carcinogenesis and could be a potential therapeutic target in follicular thyroid carcinoma.

Applications:

• Identifying potential molecular targets for cancer diagnosis, prevention, and treatment.

• Testing kinase inhibitors and other novel drugs being discovered for the treatment of thyroid cancer.

• Tools to understand the genetic changes during cancer development.

Advantages: This model provides the opportunity to study the alterations in gene regulation that occur during the progression and metastasis of thyroid carcinogenesis, not just the genes that control initial carcinogenesis.

Development Status: The technology is currently in the pre-clinical stage of development.

Inventors: Sheue-yann Cheng (NCI).

Patent Status: HHS Reference No. E–208–2009/0—Research Tool. Patent protection is not being pursued for this technology.

Publications:

1. Furuya F, Lu C, Willingham MC, Cheng SY. Inhibition of phosphatidylinositol 3-kinase delays tumor progression and blocks metastatic spread in a mouse model of thyroid cancer. Carcinogenesis. 2007 Dec;28(12):2451–2458. [PubMed: 17660507]

2. Kato Y, Ying H, Zhao L, Furuya F, Araki O, Willingham MC, Cheng SY. PPARgamma insufficiency promotes follicular thyroid carcinogenesis via activation of the nuclear factor-kappaB signaling pathway. Oncogene. 2006 May 4;25(19):2736–2747. [PubMed: 16314832]

3. Suzuki H, Willingham MC, Cheng SY. Mice with a mutation in the thyroid hormone receptor beta gene spontaneously develop thyroid carcinoma: a mouse model of thyroid carcinogenesis. Thyroid. 2002 Nov;12(11):963–969. [PubMed: 12490073]

4. Kaneshige M, Kaneshige K, Zhu X, Dace A, Garrett L, Carter TA, Kazlauskaite R, Pankratz DG, Wynshaw-Boris A, Refetoff S, Weintraub B, Willingham MC, Barlow C, Cheng S. Mice with a targeted mutation in the thyroid hormone beta receptor gene exhibit impaired growth and resistance to thyroid hormone. Proc Natl Acad Sci U S A. 2000 Nov 21;97(24):13209–13214. [PubMed: 11069286]

Licensing Status: Available for licensing.

Licensing Contact: Whitney A. Hastings; 301–451–7337; *hastingw@mail.nih.gov.*

Chemokine-Tumor Antigen Fusion Proteins as Cancer Vaccines

Description of Technology: Available for licensing is a tumor vaccine construct comprising a chemoattractant (such as human chemokines CCL7 and CCL20) fused to a tumor antigen (including human mucin-1, a transmembrane protein that is aberrantly expressed in cancer; or single chain antibody expressed by B cell malignancy, or melanoma antigen gp100 expressed in human melanomas). The majority of tumor antigens are believed to be poorly immunogenic because they represent oncogene gene products or other cellular genes which are normally present in the host. As a result, poor immunogenicity has been a major obstacle to successful immunotherapy with tumor vaccines. Administration of this fusion chemokine and tumor antigen protein, or a nucleic acid encoding this fusion protein, elicits a tumor specific cellular and humoral immune response thereby providing a potent cancer vaccine.

Applications: Cancer immunotherapy.

Development Status: Proof of the concept and pre-clinical development have been successfully completed.

Market: The global cancer market is forecasted to reach US$40 billion by 2012. Cancer vaccine research is coming to fruition, with a number of products now in Phase III trials and 15 therapeutic cancer vaccines realistically expected to launch by 2013. The therapeutic vaccine market has the potential to mirror the growth seen in the monoclonal antibody market, and reach sales in excess of US$5 billion by 2012.

Inventors: Larry Kwak (NCI) and Arya Biragyn (NIA) (both NCI at time of invention).

Related Publications:

1. Coscia M, Biragyn A. Cancer immunotherapy with chemoattractant peptides. Semin Cancer Biol. 2004 Jun;14(3):209–218. [PubMed: 15246057].

2. Biragyn A, Belyakov IM, Chow YH, Dimitrov DS, Berzofsky JA, Kwak LW. DNA vaccines encoding human immunodeficiency virus-1 glycoprotein 120 fusions with proinflammatory chemoattractants induce systemic and mucosal immune responses. Blood. 2002 Aug 15;100(4):1153–1159. [PubMed: 12149191].

3. Schiavo R, Baatar D, Olkhanud P, Indig FE, Restifo N, Taub D, Biragyn A. Chemokine receptor targeting efficiently directs antigens to MHC class I pathways and elicits antigen-specific CD8+ T-cell responses. Blood. 2006 Jun 15;107(12):4597–4605. [PubMed: 16514063].

4. Biragyn A, Ruffini PA, Coscia M, Harvey LK, Neelapu SS, Baskar S, Wang JM, Kwak LW. Chemokine receptor-mediated delivery directs self-tumor antigen efficiently into the class II processing pathway in vitro and induces protective immunity in vivo. Blood. 2004 Oct 1;104(7):1961–1969. [PubMed: 15191951].

5. Qin H, Nehete PN, He H, Nehete B, Buchl S, Cha SC, Sastry JK, Kwak LW. Prime-boost vaccination using chemokine-fused gp120 DNA and HIV envelope peptides activates both immediate and long-term memory cellular responses in rhesus macaques. J Biomed Biotechnol. 2010;2010:860160. [PubMed: 20454526].

6. Qin H, Cha SC, Neelapu SS, Lou Y, Wei J, Liu YJ, Kwak LW. Vaccine site inflammation potentiates idiotype DNA vaccine-induced therapeutic T cell-, and not B cell-, dependent antilymphoma immunity. Blood. 2009 Nov 5;114(19):4142–4149. [PubMed: 19749091].

7. Singh A, Nie H, Ghosn B, Qin H, Kwak LW, Roy K. Efficient modulation of T-cell response by dual-mode, single-carrier delivery of cytokine-targeted siRNA and DNA vaccine to antigen-presenting cells. Mol Ther. 2008 Dec;16(12):2011–2021. [PubMed: 18813280].

Patent Status: U.S. Patent No. 6,562,347 issued 13 May 2003 (HHS Reference No. E–107–1998/0–US–03).

Licensing Contact: Patrick McCue, PhD; 301–435–5560; *mccuepat@mail.nih.gov*.

Collaborative Research Opportunity: The National Institute on Aging, Laboratory of Immunology, is seeking statements of capability or interest from parties interested in collaborative research to further develop, evaluate, or commercialize cancer vaccines that target skin antigen-resenting cells. Please contact Nicole Guyton at 301–435–3101 or *guytonn@mail.nih.gov* for more information.

Dated: October 21, 2010.

Richard U. Rodriguez,

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

[FR Doc. 2010–27179 Filed 10–26–10; 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.

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.

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IL–10 and IFNγ Peptide Inhibitors

Description of Invention: Available for licensing are several potent and selective inhibitors of IL–10 and IFN-γ signaling. Although cytokines play important roles in cancer and inflammation, there are no specific inhibitors of any cytokines to date. IL–10 and IFN-γ cytokine signaling play crucial roles in inflammation, cancer growth, and autoimmune diseases. The investigators have developed short peptides that potently and selectively interfere with dimerization of the cytokines and their binding to the corresponding receptor. Included in the patent application are also metabolically stable lipopeptides mimicking conserved regions of IL–10 and IFN-γ receptors that interfere with STAT3 and STAT1 phosphorylation and subsequent signaling. Lipopeptides potently inhibit STAT3 and STAT1-dependent growth of cancer cells. These compounds are promising drug candidates for the treatment of cancer and many infectious and inflammatory diseases.

Application: Cancer, viral infections and anti-inflammatory treatments.

Advantages:

- Potent, stable peptide inhibitors.
- Selective IL–10 and IFN-γ inhibitors.

Development Status: The technology is currently in the pre-clinical stage of development.

Market: The annual growth rate for the therapeutic peptide market is estimated at about 7.5%.

Inventors: Nadya Tarasova *et al.* (NCI).

Patent Status: U.S. Provisional Application No. 61/333,512 filed 11 May 2010 (HHS Reference No. E–167–2010/0–US–01).

Licensing Status: Available for licensing.

Licensing Contact: Jennifer Wong; 301–435–4633; *wongje@mail.nih.gov.*

Collaborative Research Opportunity: The Center for Cancer Research, Cancer and Inflammation Program, is seeking statements of capability or interest from parties interested in collaborative research to further develop, evaluate, or commercialize inhibitors of IL10, IFNγ and STAT3 signaling. Please contact John Hewes, Ph.D. at 301–435–3121 or *hewesj@mail.nih.gov* for more information.

Diagnostic and Prognostic HCC-Related Metabolites

Description of Invention: Metabolite profiling identifies and measures changes in cellular metabolites as a means to determine a direct correlation between gene expression and changes in biological function. Investigators at the National Cancer Institute have identified a unique set of metabolite biomarkers associated with hepatocellular carcinoma (HCC), early stage HCC, HCC patient outcome and HCC stem-cell subtype. Subsets of this metabolite/gene signature can distinguish HCC tumors from normal tissues with 88–97% accuracy, identify early stage HCC patients with 62–78% accuracy, wherein early stage is defined as TNM stage I, prognose negative patient outcome, and identify a HCC stem cell subtype with 70–77% accuracy. These metabolites and gene surrogates are elements of the PI3K and Myc signaling networks which can potentially be targeted for therapeutic purposes.