sale to the consumer. The term ''leafy greens'' as used in this draft guidance includes raw agricultural commodities and fresh-cut/value-added products. Examples of leafy greens include iceberg lettuce, romaine lettuce, leaf lettuce, butter lettuce, baby leaf lettuce (immature lettuce or leafy greens), escarole, endive, spring mix, spinach, cabbage, kale, arugula, and chard. Leafy greens do not include herbs such as cilantro and parsley.

This draft guidance is based primarily on leafy greens industry guidelines issued in 2006 (Ref. 1), along with agency experience and information from other recent public and private programs. The leafy greens industry has since updated and supplemented its 2006 guidelines with additional recommendations on the production and harvest of leafy greens that include quantitative metrics and measures to assist industry in implementing the guidelines (Ref. 2). This draft guidance does not include these more specific and quantitative metrics and measures. We are considering the extent to which more specific measures, including metrics, should be utilized to help verify the implementation and efficacy of the Federal recommendations and industry practices. We are also evaluating the extent to which metrics can be applied to diverse geographic areas within the United States and internationally. FDA invites comment on whether such information should be incorporated into the guidance, when finalized.

FDA is issuing this draft guidance as Level 1 draft guidance consistent with FDA's good guidance practices regulation (21 CFR 10.115). The draft guidance, when finalized, will represent the agency's current thinking on the microbiological hazards presented by fresh and fresh-cut leafy greens products and the recommended control measures for such hazards in production and harvesting, postharvest operations, processing, distribution, and retail and food service handling of such produce. It does not create or confer any rights for or on any person and does not operate to bind FDA or the public. An alternative approach may be used if such approach satisfies the requirements of the applicable statutes and regulations.

II. Paperwork Reduction Act of 1995

Under the Paperwork Reduction Act of 1995 (the PRA) (44 U.S.C. 3501–3520), Federal agencies must obtain approval from the Office of Management and Budget (OMB) for each collection of information they conduct or sponsor. ''Collection of information'' is defined in 44 U.S.C. 3502(3) and 5 CFR 1320.3(c) and includes agency requests or requirements that members of the public submit reports, keep records, or provide information to a third party. Section 3506(c)(2)(A) of the PRA (44 U.S.C. 3506(c)(2)(A)) requires Federal agencies to publish notice in the **Federal Register** soliciting public comment on each proposed collection of information before submitting the collection to OMB for approval. To comply with this requirement, FDA will publish a 60-day notice on the proposed collection of information in a future issue of the **Federal Register**.

III. Comments

Interested persons may submit to the Division of Dockets Management (see **ADDRESSES**) written or electronic comments regarding this document. 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. The draft guidance and received comments may be seen in the Division of Dockets Management between 9 a.m. and 4 p.m., Monday through Friday.

IV. Electronic Access

Persons with access to the Internet may obtain the draft guidance at either *http://www.fda.gov/FoodGuidances* or *http://www.regulations.gov*.

V. References

The following references have been placed on display in the Division of Dockets Management, 5630 Fishers Lane, rm. 1061, Rockville, MD 20852 and may be seen by interested persons between 9 a.m. and 4 p.m., Monday through Friday.

1. Gorny, J., et al., editors, ''Commodity Specific Food Safety Guidelines for the Lettuce and Leafy Greens Supply Chain'' (1st ed.); International Fresh-cut Produce Association, Produce Marketing Association, United Fresh Fruit and Vegetable Association, Western Growers Association; April 25, 2006. Accessed online at *http://www.fda.gov/Food/FoodSafety/Product-SpecificInformation/FruitsVegetablesJuices/GuidanceComplianceRegulatoryInformation/ucm168630.htm*.

2. See ''Commodity Specific Food Safety Guidelines for the Production and Harvest of Lettuce and Leafy Greens''; Produce Marketing Association, United Fresh Fruit and Vegetable Association, and Western Growers Association; last revised June 13, 2008. Accessed online at *http://www.caleafygreens.ca.gov/trade/documents/LGMAAcceptedGAPs06.13.08.pdf*. (FDA has verified the Web site address, but FDA is not responsible for any subsequent changes to the Web site after this document publishes in the **Federal Register**.)

Dated: July 28, 2009.

Jeffrey Shuren,

Associate Commissioner for Policy and Planning.

[FR Doc. E9–18451 Filed 7–31–09; 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.

Treatment of Cancer Using Metal Coordinating Compounds That Kill Multi-Drug Resistant Cancer Cells

Description of Invention: One of the major hindrances to successful cancer chemotherapy is the development of multi-drug resistance (MDR) in cancer cells. MDR is frequently caused by the increased expression or activity of ABC transporter proteins in response to the toxic agents used in chemotherapy. Research has generally been directed to overcoming MDR by inhibiting the activity of ABC transporters. However, compounds that inhibit ABC transporter activity often elicit strong and undesirable side-effects, restricting their usefulness as therapeutics.

In an alternative approach to reducing the debilitating effects of MDR during cancer therapy, scientists at the NIH have identified a family of compounds

whose activities are enhanced, rather than decreased, in MDR cancer cells. Particular embodiments of these ''MDR-selective compounds'' include certain metal coordinating compounds. Recent evidence suggests that these MDR-selective compounds can be used to kill cancer cells that overexpress ABC transporters or to re-sensitize multi-drug resistant cancer cells to chemotherapeutics. Furthermore, the effectiveness of these compositions in killing MDR cancer cells correlates directly with the level of ABC transporter expression. Importantly, MDR-selective compounds are not inhibitors of ABC transporters, thereby reducing the likelihood of undesirable side-effects during treatment. Thus, MDR-selective compounds represent a powerful strategy for treating multi-drug resistant cancers as a direct chemotherapeutic and as agents that can re-sensitize MDR cancer cells for treatment with additional chemotherapeutic agents.

Applications

- Treatment of cancers associated with multi-drug resistance, either alone or in combination with other therapeutics.
- Re-sensitization of multi-drug resistant cancer cells to chemotherapeutic agents.

Advantages

- MDR-selective compounds capitalize on one of the most common drawbacks to cancer therapies (MDR) by using it as an advantage for treating cancer.
- The compositions do not inhibit the function of ABC transporters, reducing the chance of side-effects during treatment.
- The effects of MDR-selective compounds correlate with the level of ABC transporter expression, allowing healthy cells which do not express high levels of ABC transporters to better survive treatment.

Development Status: Preclinical stage of development.

Patent Status: U.S. Provisional Application No. 61/182,511 (HHS Reference No. E–157–2009/0–US–01).

Inventors: Gergely Szakacs *et al.* (NCI).

For More Information, See

- C Hegedus *et al.* Interaction of ABC multidrug transporters with anticancer protein kinase inhibitors: substrates and/or inhibitors? Curr Cancer Drug Targets. 2009 May;9(3):252–272.
- MD Hall *et al.* Synthesis, activity, and pharmacophore development for isatin-beta-thiosemicarbazones with selective activity toward multidrug-resistant cells. J Med Chem. 2009 May 28;52(10):3191–3204.
- U.S. Patent Application Publication 20080214606 A1 (U.S. Patent Application 11/629,233).

Licensing Status: Available for licensing.

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

Collaborative Research Opportunity: The Institute of Enzymology is seeking statements of capability or interest from parties interested in collaborative research to further develop, evaluate, or commercialize MDR-selective compounds. Please contact John D. Hewes, Ph.D. at 301–435–3121 or *hewesj@mail.nih.gov* for more information.

Non Toxic Peptide Treatment for Dyslipidemic and Vascular Disorders

Description of Invention: Dyslipidemia and vascular disorders such as hyperlipidemia, hypercholesterolemia, HDL deficiency, coronary heart disease, atherosclerosis, or thrombic stroke, have become major health concerns in recent years. Various approaches to treating these diseases have led to mixed success with some undesirable side effects. Long term administration of some regimens aimed at reducing cholesterol levels in cells can lead to persistent hypertriglyceridemia; a condition that is characterized by chronically high triglycerides in the blood. Other approaches, such as using peptides to stimulate the efflux of lipids from cells, are also associated with high toxicity, which has limited their use.

This technology uses peptide and peptide analogues with multiple amphipathic alpha helical domains that have the dual ability to promote lipid efflux from cells and stimulate lipoprotein lipase activity, without inducing toxicity. It consists of motifs that mimick apolipoprotein A–I (apoA–I), the most abundant protein constituent of high density lipoproteins (HDLs) that is capable of inducing cellular lipid efflux, and motif resembling apolipoprotein C–II (apoC–II), a known activator of lipoprotein lipase. Peptides constructed with these structural domains are capable of stimulating lipid efflux and activating lipoprotein lipase, leading to a reduced incidence of hypertriglyceridemia. Unlike previous methods, some amphipathic peptides cause transient hypertriglyceridema in mice that lasts for less than 8 hours. Mice treated with these modified peptides have shown preserved liver function as they have failed to express increased levels of biomarkers for liver damage and prevent hypertriglyceridemia. Furthermore, treated mice show a reduced level of pro-atherogenic lipoproteins. This technology demonstrates specific control of lipid efflux and transport; a desirable property that gives it a significant advantage for treating or preventing a vast range of vascular diseases and their dyslipidemic precursors.

This technology also encompasses a method for identifying non-cytotoxic peptides that promote lipid efflux from cells and activates lipoprotein lipase.

Applications and Advantages

- Peptide treatment of dyslipidemic and vascular disorders.
- Transient hypertriglyceridemia with no reported toxicity.
- Method of identifying therapeutic non-cytotoxic peptides.

Development Status: Pre-clinical.

Inventor: Alan T. Remaley and Marcelo Amar (NHLBI).

Publication: AT Remaley, F Thomas, JA Stonik, SJ Demosky, SE Bark, EB Neufeld, AV Bocharov, TG Vishnyakova, AP Patterson, TL Eggerman, S Santamarina-Fojo, HB Brewer. Synthetic amphipathic helical peptides promote lipid efflux from cells by an ABCA1-dependent and an ABCA1-independent pathway. J Lipid Res. 2003 Apr;44(4):828–836.

Patent Status: U.S. Provisional Application No. 60/045,213 filed 15 Apr 2008 (HHS Reference No. E–138–2008/0–US–01); PCT Application No. PCT/US2009/040560 filed 14 Apr 2009 (HHS Reference No. E–138–2008/0–PCT–02).

Licensing Status: Available for licensing.

Licensing Contact: Fatima Sayyid, M.H.P.M.; 301–435–4521; *sayyidf@mail.nih.gov*.

Methods for Treating or Ameliorating Fibrosis by Inhibiting the Interaction Between IL–21 Receptor (IL–21R) and IL–21

Description of Invention: This invention includes methods for treating or ameliorating fibrosis by inhibiting the interaction between IL–21 Receptor (IL–21R) and IL–21 using either anti-IL21R monoclonal antibodies (or binding fragments of anti-IL–21R mAbs), anti-IL21 monoclonal antibodies (or binding fragments of anti-IL–21 mAbs) or soluble IL–21R (or binding fragments of IL–21R). It is believed that the TH2 immune response, induced by IL–21, plays a major role in the pathogenesis of tissue fibrosis. Antagonism of IL–21R by anti-IL–21R monoclonal antibodies or the sequestration of IL–21 by soluble IL–

21R or anti-IL–21 monoclonal antibodies has been demonstrated to reduce TH2 immune responses associated with fibrosis in animal models.

The causes of chronic tissue fibrosis are diverse and the market for a therapeutic that targets fibrosis is large. Fibrosis is associated with diverse causes which include: genetic diseases (such as cystic fibrosis); autoimmune diseases (such as scleroderma); chronic viral infections (such as hepatitis), parasitic infections (such as schistosomiasis); and occupational exposures to causative agents (such as asbestosis). Additionally, many cases of tissue fibrosis are idiopathic.

Application: The treatment or amelioration of tissue fibrosis.

Inventors: Thomas A. Wynn (NIAID); Deborah A Young; Mary Collins; and Michael J. Grusby.

Relevant Publication: J Pesce *et al.* The IL–21 receptor augments Th2 effector function and alternative macrophage activation. J Clin Invest 2006 Jul;116(7):2044–2055.

Patent Status: U.S. patent application no. 11/402,885 (priority date April 14, 2005) and international patent applications including European patent application No. EP06/0750009 (HHS Reference No. E–250–2005).

Licensing Status: Available for non-exclusive licensing.

Licensing Contact: Surekha Vathyam, Ph.D.; 301–435–4076; *vathyams@mail.nih.gov*.

Collaborative Research Opportunity: The National Institute of Allergy and Infectious Diseases is seeking statements of capability or interest from parties interested in collaborative research to further develop, evaluate, or commercialize this invention. Please contact Nicole Mahoney at 301–435–9017 or *mahoneyn@niaid.nih.gov* for more information.

Use of Discoidin Domain Receptor 1 (DDR1) and Agents That Affect the DDR1/Collagen Pathway

Description of Invention: Dendritic cells (DCs) are pivotal antigen-presenting cells for initiation of an immune response. Indeed, dendritic cells provide the basis for the production of an effective immune response to a vaccine, particularly for antigens wherein conventional vaccination is inadequate. DCs are also important in the production on an immune response to tumor antigens.

The present invention discloses methods of using the receptor tyrosine kinase discoidin domain receptor 1 (DDR1) to facilitate the maturation/differentiation of DCs or macrophages. Activating agents of DDR1 may be useful in the induction of highly potent, mature DCs or highly differentiated macrophages from DC precursors, such as monocytes. Use of this method may enhance the antigen presenting capabilities of the immune system, leading to a more effective overall immune response.

Inventor: Teizo Yoshimura (NCI).

Relevant Publications

1. H Kamohara *et al.* Discoidin domain receptor 1 isoform-a (DDR1a) promotes migration of leukocytes in three-dimensional collagen lattices. FASEB J. 2001 Dec;15(14):2724–2726.

2. W Matsuyama *et al.* Interaction of discoidin domain receptor 1 isoform b (DDR1b) with collagen activates p38 mitogen-activated protein kinase and promotes differentiation of macrophages. FASEB J. 2003 Jul;17(10):1286–1288.

Patent Status: U.S. Application No. 10/507,385 filed 09 Sep 2004 (HHS Reference No. E–083–2002/2–US–02).

Licensing Status: Available for licensing.

Licensing Contact: Betty B. Tong, Ph.D.; 301–594–6565; *tongb@mail.nih.gov*.

Dated: July 28, 2009.

Richard U. Rodriguez,

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

[FR Doc. E9–18504 Filed 7–31–09; 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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A Tumorigenic MEF/3T3 Tet-Off Mouse Fibroblast Cell Line Stably Transfected With a T7–Tagged Srp20 Expression Construct (pJR17)

Description of Technology: Alternative RNA splicing is a means by which the human genome can produce many more proteins from the genes available. It is emerging that aberrations in alternative RNA splicing contributes to the development of cancers. SRp20 is a cellular splicing factor that is involved in the process of alternative splicing of RNA. Investigators at the National Cancer Institute (NCI), National Institutes of Health (NIH) have discovered that SRp20 is overexpressed in many types of cancer and furthermore promotes the induction and maintenance of tumor cell growth. This was demonstrated in part by engineering a non-tumorigenic cell to become tumorigenic in mice by overexpressing SRp20.

Research Material available for licensing is a tumorigenic MEF/3T3 tet-off mouse fibroblast cell line stably transfected with a T7-tagged SRp20 expression construct (pJR17) that is under the transcriptional control of tetracycline.

Applications: Use in pre-clinical development of therapeutic approaches to cancer that target aberrant alternative RNA splicing.

Advantages: Transcriptional control of expression using Tet-off system; Availability of stably transfected cell line saves time and effort for other investigators.

Market: Research Tool.

Development Status: Ready to use.

Inventors: Zhi-Ming Zheng and Rong Jia (NCI).

Publications: Manuscript in preparation.

Patent Status: HHS Reference No. E–229–2009/0—Research Material. Patent protection is not being sought for this technology.

Licensing Status: Available for licensing.

Licensing Contact: Sabarni Chatterjee, Ph.D.; 301–435–5587; *chatterjeesa@mail.nih.gov.*

Collaborative Research Opportunity: The National Cancer Institute, Center for Cancer Research, HIV and AIDS Malignancy Branch, is seeking statements of capability or interest from