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.

Novel Inhibitor of NF-kappa B Pathway

Description of Technology: Many tumors and blood cell cancers show overactivation of the NF-kappa B signal transduction pathway. This overactivation is associated with cancer forming in the colon, liver and other epithelial sites. In addition, there is evidence that overactivation leads to tumor formation and metastasis. However, this pathway is key for normal immunity, so any inhibition of NF-kappa B overactivation must avoid diminishing the body's ability to fight infection.

This invention claims a compound that inhibits NF-kappa B activation without affecting other transcription factors such as AP–1 and SRE binding proteins. It appears to function by blocking IKK beta and is effective at low micromolar concentrations without affecting cell proliferation or cell survival. At this low concentration, NF-kappa B is reduced to basal levels so this novel compound has prospects for preventing or treating cancer without being detrimental to immunity. In addition, because NF-kappa B overactivation contributes to a variety of inflammatory disorders including colitis, diabetes, prostatitis, and pancreatitis this compound has therapeutic applications beyond cancer.

Applications:

• Therapeutic for the chemoprevention or treatment of cancers associated with the overactivation of NF-kappa B signaling pathway.

• Therapeutic for the treatment of inflammatory disorders related to NF-kappa B overactivation.

• Reagent for the diagnosis of conditions related to overexpression of NF-kappa B.

Advantages:

• Highly specific inhibitor that allows targeting NF-kappa B without inhibiting other transcription factors.

• Effective at preventing carcinogenesis without affecting normal cell proliferation and survival.

• Therapeutic for treatment of cancer that will not compromise the immune system.

Development Status: Early stage.

Market: Cancer is the second leading cause of death in the U.S. and it is estimated that 1.4 million Americans develop cancer in a year.

Inventors: Curtis J. Henrich *et al.* (NCI).

Publications: None related to invention have been published.

Patent Status: U.S. Provisional Application No. 61/098,977 filed 22 Sep 2008 (HHS Reference No. E–295–2008/0–US–01).

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

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

Collaborative Research Opportunity: The National Cancer Institute (SAIC-Frederick) is seeking statements of capability or interest from parties interested in collaborative research to further develop, evaluate, or commercialize around development of analogs and/or further investigations of mechanism of action of the compound. Please contact John D. Hewes, Ph.D. at 301–435–3121 or *hewesj@mail.nih.gov* for more information.

Method for Predicting and Detecting Tumor Metastasis

Description of Technology: Detecting cancer prior to metastasis greatly increases the efficacy of treatment and the chances of patient survival. Although numerous biomarkers have been reported to identify aggressive tumor types and predict prognosis, each biomarker is specific for a particular type of cancer, and no universal marker that can predict metastasis in a number of cancers have been identified. In addition, due to a lack of reliability, several markers are typically required to determine the prognosis and course of therapy.

The inventors discovered a novel CPE splice variant designated CPE-Δ N and found its expression levels increase according to the presence of cancer and metastasis wherein this variant is upregulated in tumors and further increased in metastatic cancer. This data has been demonstrated both in vitro and in vivo experiments and in liver, breast, prostate, colon, and head and neck cancers. Metastatic liver cells treated with CPE-Δ N siRNA reversed the cells from being metastatic and arrested cells from further metastasis. Thus, this novel CPE isoform is a biomarker for predicting metastasis and its inhibitors have an enormous potential to increase patient survival.

Applications:

• Method to prognose multiple types of cancer and determine likelihood of metastasis.

• Method to prevent and treat cancer with CPE inhibitors.

• Method to determine the stage of cancer development.

• CPE-Δ N pharmaceutical compositions.

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

Market:

• Global cancer market is worth more than eight percent of total global pharmaceutical sales.

• Cancer industry is predicted to expand to $85.3 billion by 2010.

Inventors: Y. Peng Loh *et al.* (NICHD).

Patent Status: U.S. Provisional Application No. 61/080,508 filed 14 Jul 2008 (HHS Reference No. E–234–2008/0–US–01).

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

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

Collaborative Research Opportunity: The National Institute of Child Health and Human Development, Laboratory of Development Neurobiology, is seeking statements of capability or interest from parties interested in collaborative research to further develop, evaluate, or commercialize Method for Predicting and Detecting Tumor Metastasis. Please contact John D. Hewes, Ph.D. at 301–435–3121 or *hewesj@mail.nih.gov* for more information.

Dated: December 23, 2008.

Richard U. Rodriguez,

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

[FR Doc. E8–31238 Filed 12–31–08; 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.

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.

Knockout of Aryl Hydrocarbon Receptor (AhR) and Its Binding Partner Aryl Hydrocarbon Receptor Nuclear Translocator (Arnt) Each in Separate Mouse Models

Description of Technology: The technology relates to two separate knockout mouse models of related transcription factors that bind each other. The aryl hydrocarbon receptor (AhR) and the aryl hydrocarbon receptor nuclear translocator (Arnt) protein are transcription factors that play an important role in mediating the effects of man-made environmental toxins. They also play a role in mammalian development and physiological homeostasis. Members of the PAS domain/bHLH family of transcription factors, they are obligate dimerization partners with each other and other members of this family, such as hypoxia-inducible factor 1alpha (HIF1alpha). These transcription factors have been shown to be important in a number of specific tissues including ovary, vascular endothelium, keratinocytes, T-cells, and liver.

Available for licensing is a knockout mouse line in which the AhR receptor has been knocked-out, and a mouse line containing a floxed allele of the Arnt gene. The Arnt mouse line can be used to disrupt the Arnt gene in different tissues by breeding the Arnt-floxed mice with transgenic mice in which the Cre recombinase is under the control of tissue-specific promoters. These mice may be used as a research tool for drug development where PAS/bHLH transcription factors are targeted.

Applications:

• Tool for drug studies targeting PAS/bHLH transcription factors.

• Tool to probe the role of the Arnt protein in a tissue-specific manner.

Inventors: Frank J. Gonzalez and Pedro M. Fernandez-Salguero (NCI).

Related Publications:

1. S Tomita, CJ Sinal, SH Yim, and FJ Gonzalez. Conditional disruption of the aryl hydrocarbon receptor nuclear translocator (Arnt) gene leads to loss of target gene induction by the aryl hydrocarbon receptor and hypoxia-inducible factor 1alpha. Mol Endocrinol. 2000 Oct;14(10):1674–1681.

2. SH Yim, Y Shah, S Tomita, HD Morris, O Gavrilova, G Lambert, JM Ward, and FJ Gonzalez. Disruption of the Arnt gene in endothelial cells causes hepatic vascular defects and partial embryonic lethality in mice. Hepatology. 2006 Sep;44(3):550–560.

3. P Fernandez-Salguero *et al.* Immune system impairment and hepatic fibrosis in mice lacking the dioxin-binding Ah receptor. Science 1995 May 5;268(5211):722–726.

Patent Status: HHS Reference Nos. E–046–2009/0 and E–047–2007/0—Research Tools. Patent protection is not being pursued for these technologies.

Licensing Status: This technology is available as a research tool under a Biological Materials License.

Licensing Contact: Steve Standley, Ph.D.; 301–435–4074; *sstand@mail.nih.gov.*

Collaborative Research Opportunity: The National Cancer Institute, Laboratory of Metabolism, Center for Cancer Research, is seeking statements of capability or interest from parties interested in collaborative research to further develop, evaluate, or commercialize this technology. Please contact John D. Hewes, Ph.D. at 301–435–3121 or *hewesj@mail.nih.gov* for more information.

Recombineering Vector

Description of Technology: Transgenic mouse models have become a common experimental tool for unraveling gene function. Bacterial artificial chromosome (BAC) mediated transgenesis has proven to be a highly reliable way to obtain accurate transgene expression for *in vivo* studies of gene expression and function. A rate-limiting step in characterizing large numbers of genes by this approach has been the speed and ease by which BACs can be modified. NIH investigators have developed a highly efficient recombineering vector that can be used for modifying BACs in bacteria. This new vector contains tetracycline and chloramphenical resistance as well as the ccdB gene that encodes a protein that interferes with *E. coli* DNA gyrase. This vector can be propagated in ccdB resistant *E. coli* strains but not in other strains (DH5a, Top10, DH10B, etc.) unless the ccdB is replaced by DNA inserts flanked by attB1 and attB2 sites. This vector was generated to modify BAC plasmids by RecA-mediated recombination.

The vector disclosed here bypasses the rate-limiting step in recombineering protocols; the efficient cloning of a modifying vector. It is well suited for efficient production of engineered BACs for use in a variety of *in vivo* studies.

Applications:

• The fusion of fluorescent protein or cre recombinase genes to a gene of interest.

• Generation of dominant negative mutations.

• Introduction of gene mutations that would mimic disease conditions.

• Insertion of lox sites for conditional deletion of transgenes.

• Generation of knock-out or knock-in constructs.

Inventors: Rafael C. Casellas and Susan E. Lim (NIAMS).

Patent Status: HHS Reference No. E–026–2009/0—Research Material. Patent protection is not being pursued for this technology.

Licensing Status: Available for Biological Material Licensing.

Licensing Contact: Suryanarayana (Sury) Vepa, Ph.D., J.D.; 301–435–5020; *vepas@mail.nih.gov.*

Collaborative Research Opportunity: The NIAMS/NIH Genomics and Immunity group is seeking statements of capability or interest from parties interested in collaborative research to further develop, evaluate, or commercialize the engineering of mouse transgenic constructs using the new vector and BAC recombineering. Please contact Rafael Casellas, Ph.D. at 301–402–7858 or e-mail to *casellar@mail.nih.gov* for more information.

Dated: December 22, 2008.

Richard U. Rodriguez,

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

[FR Doc. E8–31239 Filed 12–31–08; 8:45 am]

BILLING CODE 4140–01–P

DEPARTMENT OF HOMELAND SECURITY

Federal Emergency Management Agency

[Docket ID FEMA–2008–0017]

Voluntary Private Sector Accreditation and Certification Preparedness Program

AGENCY: Federal Emergency Management Agency, DHS.

ACTION: Public meeting notice.

SUMMARY: This notice announces the date, time, location, and discussion topics for a stakeholder meeting open to the public to engage in dialogue with Department of Homeland Security (DHS) leadership and program managers regarding the Voluntary Private Sector Preparedness Accreditation and Certification Program (PS–Prep).