(NIH) National Institute on Drug Abuse (NIDA), and in partnership with the Food and Drug Administration (FDA). Using annual interviews and the collection of bio-specimens from adults, the study is designed to establish a population-based framework for monitoring and evaluating the behavioral and health impacts of regulatory provisions by FDA as it meets its mandate under the Family Smoking Prevention and Tobacco Control Act (FSPTCA) to regulate tobacco-product advertising, labeling, marketing, constituents, ingredients, and additives. These regulatory changes are expected to influence tobacco-product risk perceptions, exposures, and use patterns in the short term, and to reduce tobacco-related morbidity and mortality in the long term. By measuring and accurately reporting tobacco product use behaviors and health effects associated with these regulatory changes, this study will provide an empirical evidence base to inform the development, implementation, and evaluation of tobacco-product regulations in the U.S.

Frequency of Response: Annually. *Affected Public:* Individuals or households. *Type of Respondents:* Youth (ages 12–17) and Adults (ages 18+). The annual reporting burden for the field test is presented in Table 1, and the annual reporting burden for the baseline data collection is presented in Table 2. The annualized cost to respondents for the field test is estimated at: $24,495; and the annualized cost to respondents for the baseline data collection is: $1,947,567. There are no Capital Costs to report. There are no Operating or Maintenance Costs to report.

TABLE 1—PATH STUDY FIELD TEST HOUR BURDEN ESTIMATES

Type of respondents	Estimated number of respondents	Estimated number of responses per respondent	Average burden hours per response	Estimated total annual burden hours requested
Adults—Household Screener	1,295	1	22/60	479
Adults—Individual Screener	840	1	6/60	84
Adults—Extended Interview	590	1	1 26/60	844
Adults—Tobacco Use Form	590	1	2/60	18
Youth—Extended Interview	100	1	55/60	92
Adult—Parent Interview	100	1	24/60	40
Total	3,515	1	.....	1,557

TABLE 2—PATH STUDY BASELINE HOUR BURDEN ESTIMATES

Type of respondents	Estimated number of respondents	Estimated number of responses per respondent	Average burden hours per response	Estimated total annual burden hours requested
Adults—Household Screener	100,983	1	22/60	37,364
Adults—Individual Screener	63,000	1	6/60	6,300
Adults—Extended Interview	42,730	1	1 26/60	61,104
Adults—Tobacco Use Form	42,730	1	2/60	1,282
Youth—Extended Interview	16,857	1	55/60	15,508
Adult—Parent Interview	16,857	1	24/60	6,743
Total	283,157	1	.....	128,301

Request for Comments: Written comments and/or suggestions from the public and affected agencies are invited on one or more of the following points: (1) Whether the proposed collection of information is necessary for the proper performance of the function of the agency, including whether the information will have practical utility; (2) The accuracy of the agency's estimate of the burden of the proposed collection of information, including the validity of the methodology and assumptions used; (3) Ways to enhance the quality, utility, and clarity of the information to be collected; and (4) Ways to minimize the burden of the collection of information on those who are to respond, including the use of appropriate automated, electronic, mechanical, or other technological collection techniques or other forms of information technology.

FOR FURTHER INFORMATION CONTACT: To request more information on the proposed project or to obtain a copy of the data collection plans and instruments, contact Kevin P. Conway, Ph.D., Deputy Director, Division of Epidemiology, Services, and Prevention Research, National Institute on Drug Abuse, 6001 Executive Blvd., Room 5185; 301–443–8755; email *PATHprojectofficer@mail.nih.gov*.

Comments Due Date: Comments regarding this information collection are best assured of having their full effect if received within 60-days of the date of this publication.

Dated: May 11, 2012.

Helio Chaves,

Deputy Executive Officer (OM Director), NIDA.

[FR Doc. 2012–12017 Filed 5–17–12; 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.

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.

Java Applet for Modeling Human Metabolism and Energy Expenditure for Adaptive Dieting and Exercise Regimens

Description of Technology: Known methods for predicting weight loss fail to account for slowing of metabolism as weight is lost and therefore overestimate the degree of weight loss. While this limitation of the 3500 Calorie per pound rule has been known for some time, it was not clear how to dynamically account for the metabolic slowing. The invention provides a Java applet for modeling of human metabolism to improve the weight change predictions. The model has been validated using previously published human data and the model equations have been published. A web-based implementation of the published dynamic model has been created to allow users to perform simulations for planning weight loss interventions in adults and accounts for individual differences in metabolism and body composition.

Potential Commercial Applications

- Obesity.
- Weight Loss.

Competitive Advantages: Personalized predictions.

Development Stage: Prototype.

Inventors: Kevin Hall, Carson Chou, Dhruva Chandramohan (all of NIDDK).

Intellectual Property: HHS Reference No. E–160–2012/0—Research Tool.

Patent protection is not being pursued for this technology.

Licensing Contact: Michael Shmilovich, Esq.; 301–435–5019; *shmilovm@mail.nih.gov.*

Antagonist of A_3 Adenosine Receptor Fluorescent Probes for the Study of Diseases Such as Cancer, Autoimmune Conditions, Dry Eye and Other Indications that Involve A_3 Signaling

Description of Technology: Small molecule drugs, A_3AR-selective agonists, are currently in advanced clinical trials for the treatment of hepatocellular carcinoma, autoimmune inflammatory diseases, such as rheumatoid arthritis, psoriasis, and dry eye disease, and other conditions. This molecular probe may serve as a companion tool to identify and stratify patient populations based on the prevalence of the target A_3 adenosine receptors.

Potential Commercial Applications: Useful tools to study prevalence of this receptor on neutrophils which is predictive of response to the agonist drugs.

Competitive Advantages: Drug screening at this receptor is often done currently using radiolabeled agonists or antagonists of the human A_3AR of nanomolar affinity. This method would avoid the use of radioisotopes in this part of the research and development process.

Development Stage

- Early-stage.
- In vitro data available.

Inventors: Kenneth A. Jacobson, *et al.* (NIDDK).

Publication: Novel Fluorescent Antagonist as a Molecular Probe in A3 Adenosine Receptor Binding Assays Using Flow Cytometry, manuscript submitted for publication.

Intellectual Property: HHS, Reference No. E–073–2012/0—U.S. Provisional Application 61/590,596 filed 25 Jan 2012 (Note: a separate license may be required for the fluorescent portion of the molecule.)

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

Methods for Selection of Cancer Patients and Predicting Efficacy of Combination Therapy With Histone Deacetylase (HDAC) and mTOR Inhibitors

Description of Technology: Available for licensing is a novel gene signature of thirty-seven drug responsive genes that links changes in gene expression to the clinically desirable outcome of improved overall survival. Expression of these genes has been linked to prognosis in several cancers, including, but not limited to multiple myeloma, lung, breast, and melanoma. Patients identified by this signature would be predicted to benefit from combined HDAC inhibitor/mTOR inhibitor therapy. Additional information is available upon request.

Potential Commercial Applications

- Development of a clinical diagnostic test to identify cancer patients who would benefit most from mTOR and HDAC combination therapy.
- Use as a surrogate biomarker related to drug response.
- Development of therapeutics targeting several cancers, including multiple myeloma.

Competitive Advantages

- Implements a smaller gene set compared to current diagnostic gene signatures.
- Provides a basis for the development of a diagnostic for patient stratification or a response measurement related to the combined use of mTOR and HDAC inhibitors for cancer treatment.

Development Stage

- Early-stage.
- In vitro data available.
- In vivo data available (animal).

Inventors: Beverly Mock *et al.* (NCI).

Intellectual Property: HHS Reference No. E–013–2012/0—U.S. Provisional Application No. 61/558,402 filed 10 Nov 2011.

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

Collaborative Research Opportunity: The NCI Center for Cancer Research, Laboratory of Cancer Biology and Genetics, is seeking statements of capability or interest from parties interested in collaborative research to further develop, evaluate or commercialize Methods for Selecting Cancer Patients for HDACi/mTORi Combination Therapy. For collaboration opportunities, please contact John Hewes, Ph.D. at *hewesj@mail.nih.gov.*

GLI-Similar 3(GLIS3) Knock Out (KO) Mice as Models to Screen Therapeutics for Diabetes, Polycystic Kidney Disease, and Hypothyroidism

Description of Technology: GLI-similar (Glis) 1–3 proteins constitute a subfamily of the Krüppel-like zinc finger transcription factors that are closely related to the Gli family. Mutations in human GLIS3 have been implicated in a syndrome characterized by neonatal diabetes and congenital hypothyroidism (NDH) and in some patients accompanied by polycystic kidney disease, glaucoma, and liver fibrosis. To further identify and study the physiological functions of GLIS3,

NIEHS investigators generated mice in which GLIS3 is ubiquitously knocked out (GLIS3–KO) or conditionally knocked out in a cell type-specific manner. GLIS3–KO mice develop polycystic kidney disease, hypothyroidism, and neonatal diabetes, as indicated by the development of hyperglycemia and hypoinsulinemia. The pancreatic endocrine cells, particularly insulin-producing pancreatic beta cells, are greatly diminished in these mice. The pancreas-selective knockout mice GLIS3(Pdx1-Cre) develop severe diabetes within 2–3 months, much later than the GLIS3–KO mice. The kidney-selective knockout of GLIS3 (GLIS3(Ksp-Cre) mice lack expression of GLIS3 in the collecting ducts and develop severe polycystic kidney disease within a period of 2–4 months. These mice can be used as models to screen therapeutics for diabetes, polycystic kidney disease, and hypothyroidism.

Potential Commercial Applications

- Therapeutic target in the management of diabetes, polycystic kidney disease, and hypothyroidism.
- Models to test therapeutic drugs for diabetes, polycystic kidney disease, and hypothyroidism.

Competitive Advantages

- Provides opportunity to discover upstream signals that regulate GLIS3 activity.
- Can be used in stem cell therapy in diabetes treatment.
- Excellent model to study the role of GLIS3 in neonatal diabetes.

Development Stage

- Early-stage.
- Pre-clinical.
- In vivo data available (animal).

Inventors: Anton M Jetten, Hong Soon Kang, Kristin Lichti-Kaiser (all of NIEHS).

Publications

1. Kang HS, *et al.* Transcription factor Glis3, a novel critical player in the regulation of pancreatic beta-cell development and insulin gene expression. Mol Cell Biol. 2009 Dec;29(24):6366–79. [PMID 19805515]

2. Kang HS, *et al.* Glis3 is associated with primary cilia and Wwtr1/TAZ and implicated in polycystic kidney disease. Mol Cell Biol. 2009 May;29(10): 2556–69. [PMID 19273592]

Intellectual Property: HHS Reference No. E–303–2011/0—Research Tool. Patent protection is not being pursued for this technology.

Related Technologies

- HHS Reference No. E–253–2010/0—An In-Vitro Cell System Useful for Identification of RORgamma Antagonists.
- HHS Reference No. E–222–2009/0—RORgamma (RORC) Deficient Mice Which Are Useful for the Study of Lymph Node Organogenesis and Immune Responses.

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

Collaborative Research Opportunity: The NIEHS is seeking statements of capability or interest from parties interested in collaborative research to further develop, evaluate or commercialize GLIS3 Knock Out Mice. For collaboration opportunities, please contact Elizabeth M. Denholm, Ph.D. at *denholme@niehs.nih.gov.*

Microarray for Detection and Subtyping of Human Influenza Viruses

Description of Technology: Available for licensing and commercial development are a novel influenza virus microarray and methods for using the microarray for the identification of existing and new types and subtypes of human influenza viruses. There are three types of influenza viruses, type A, B and C. Influenza types A or B viruses cause epidemics of disease almost every winter, with type A causes major pandemic periodically. Influenza type A viruses are further divided into subtypes based on two proteins on the surface of the virus. These proteins are called hemagglutinin (H) and neuraminidase (N). There are 16 known HA subtypes and 9 known NA subtypes of influenza A viruses. Each subtype may have different combination of H and N proteins. Although there are only three known A subtypes of influenza viruses (H1N1, H1N2, and H3N2) currently circulating among humans, many other different strains are circulating among birds and other animals and these viruses do spread to humans occasionally. There is a requirement for sensitive and rapid diagnostic techniques in order to improve both the diagnosis of infections and the quality of surveillance systems. This microarray platform tiles the genomes of all types/subtypes of influenza viruses, and is capable of correctly identifying all 3 types/subtypes of influenza viruses from an influenza vaccine sample.

Potential Commercial Applications

- Detection and identification of human influenza viruses.
- Efficient discovery of new subtypes of influenza viruses.
- Diagnosis of influenza outbreaks.

Competitive Advantages: Technology can detect multiple types and subtypes of influenza virus.

Development Stage

- Pre-clinical.
- In vitro data available.

Inventors: Xiaolin Wu, David J. Munroe, Cassio S. Baptista, Elizabeth Shannon (all of NCI).

Intellectual Property: HHS Reference No. E–208–2006/0—U.S. Patent Application No. 11/936,530 filed 07 Nov 2007.

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

M3 Muscarinic Receptor Knockout Mice (Chrm3 tm1Jwe) for the Study of Obesity and Other Metabolic Disorders

Description of Mouse: The five Muscarinic Acetylcholine (ACh) receptors are G-protein coupled receptors (M1R–M5R). M3 muscarinic ACh receptors are present in the central nervous system and the periphery.

M3R knockout mice are viable and fertile, and have no major morphological abnormalities. They have a lean phenotype due to a combination of reduced caloric intake and increased energy expenditure. Because of their lean phenotype, M3R knockout mice have improved glucose tolerance and increased insulin sensitivity. Pharmacological blockade of central M3Rs may be a novel strategy for the treatment of obesity and associated metabolic disorders.

In the airway, vagally-mediated bronchoconstriction responses were abolished in M3R knockout mice in vivo, suggesting that M3R antagonists may be useful in the treatment of chronic obstructive pulmonary disease (COPD) and asthma. Studies with M3R knockout mice also have shown that the M3R is the major muscarinic receptor mediating ACh-induced glandular secretion from exocrine and endocrine glands, including the secretion of insulin from pancreatic beta cells.

Potential Commercial Applications: Animal model to study COPD and metabolism.

Competitive Advantages: M3R knockout mice are viable and fertile, and have no major morphological abnormalities.

Development Stage: Pre-clinical.

Developer of Mouse: Jürgen Wess, Ph.D. (NIDDK).

Publication: Yamada M, *et al.* Mice lacking the M3 muscarinic acetylcholine receptor are hypophagic and lean. Nature. 2001 Mar 8;410(6825):207–12. [PMID 11242080]

Intellectual Property: HHS Reference No. E–346–2004/2—Research Tool. Patent protection is not being pursued for this technology.

Related Technologies

- HHS Reference No. E–346–2004/0—M1 Muscarinic receptor KO (Chrm1tm1Jwe) Mice.
- HHS Reference No. E–346–2004/1—M2 Muscarinic receptor KO (Chrm2 tm1Jwe) Mice.

Licensing Contact: Jaime M. Greene, M.S.; 301–435–5559; *greenejaime@mail.nih.gov*

Use of E-Selectin Tolerization as Treatment for Immunological and Vascular-Related Disorders

Description of Technology: This technology relates to the mucosal delivery (e.g. intranasal) of an E-selectin fragment as a tolerization agent for the prevention and treatment of immunological and vascular-related disorders, including stroke and multiple sclerosis (MS) as well as rare or orphan diseases involving vascular modulated disorders.

E-selectin is an adhesion molecule that is expressed on endothelial cells lining blood vessels in response to certain localized cytokines, making the endothelial surface pro-coagulant, pro-inflammatory and/or immunoreactive. Such changes on the endothelial surface have been linked to the development of vascular-related disorders like stroke, as well as immune regulated diseases such as MS.

Intranasal administration of E-selectin, using a tolerizing dosing schedule, induces an immunological tolerance to E-selectin. T regulatory cells become targeted to activating blood vessel segments, where they release immunomodulatory cytokines such as IL–10. This release of cytokines suppresses local pro-coagulant, pro-inflammatory and immunoreactive effects. Thus, administration of E-selectin as a tolerizing agent will provide a targeted therapeutic approach, impacting only affected sites in the endothelium.

Potential Commercial Applications: Treatment of diseases biologically based on vascular initiated immune regulation. Such disorders include prevention of secondary stroke, MS, Alzheimer's, Parkinson's, rheumatoid arthritis, type 1 diabetes, and psoriasis.

Competitive Advantages

- Low doses utilized thus minimizing potential side effects.
- Animal data are available, with further studies currently on-going.
- Administration through the intranasal route represents a less invasive mode of delivery.
- FDA pre-IND meetings have been held and FDA communications are ongoing.

Development Stage

- Pre-clinical.
- In vitro data available.
- In vivo data available (animal).

Inventors: John M. Hallenbeck, Maria Spatz, Hidetaka Takeda, Hideaki Wakita (all of NINDS)

Publications

1. Li X, *et al.* Intranasal delivery of E-selectin reduces atherosclerosis in ApoE-/- mice. PLoS One. 2011;6(6):e20620. Epub 2011 Jun 20. [PMID 21701687]
2. Hallenbeck J. How inflammation modulates central nervous system vessel activation and provides targets for intervention—a personal perspective. Ann N Y Acad Sci. 2010 Oct;1207:1–7. doi: 10.1111/j.1749–6632.2010.05785.x. [PMID 20955418]
3. Ishibashi S, *et al.* Mucosal tolerance to E-selectin promotes the survival of newly generated neuroblasts via regulatory T-cell induction after stroke in spontaneously hypertensive rats. J Cereb Blood Flow Metab. 2009 Mar;29(3):606–20. [PMID 19107136]
4. Wakita H, *et al.* Mucosal tolerization to E-selectin protects against memory dysfunction and white matter damage in a vascular cognitive impairment model. J Cereb Blood Flow Metab. 2008 Feb;28(2):341–53. [PMID 17637705]
5. Nakayama T, *et al.* Intranasal administration of E-selectin to induce immunological tolerization can suppress subarachnoid hemorrhage-induced vasospasm implicating immune and inflammatory mechanisms in its genesis. Brain Res. 2007 Feb 9;1132(1):177–84. [PMID 17188657]
6. Illoh K, *et al.* Mucosal tolerance to E-selectin and response to systemic inflammation. J Cereb Blood Flow Metab. 2006 Dec;26(12):1538–50. [PMID 16596122]
7. Chen Y, *et al.* Mucosal tolerance to E-selectin provides cell-mediated protection against ischemic brain injury. Proc Natl Acad Sci U S A. 2003 Dec 9;100(25):15107–12. [PMID 14645708]
8. Takeda H, *et al.* Induction of mucosal tolerance to E-selectin prevents ischemic and hemorrhagic stroke in spontaneously hypertensive genetically stroke-prone rats. Stroke. 2002 Sep;33(9):2156–63. [PMID 12215580]

Intellectual Property

- HHS Reference No. E–237–1999/0—
  —U.S. Patent No. 7,261,896 issued 28 Aug 2007.
  —U.S. Patent Application No. 11/820,326 filed 19 Jun 2007.
- HHS Reference No. E–237–1999/1—
  —U.S. Patent No. 7,897,575 issued 01 Mar 2011.
  —U.S. Patent Application No. 12,859,048 filed 18 Aug 2010.
- and Foreign counterparts in Australia, Canada, Europe, and Japan

Licensing Contact: Tara Kirby, Ph.D.; 301–435–4426; *tarak@mail.nih.gov.*

Collaborative Research Opportunity: The Stroke Branch, NINDS/NIH, is seeking statements of capability or interest from parties interested in collaborative research to further develop, evaluate or commercialize the applications of E-selectin tolerization in treatment of neurological based disease. For collaboration opportunities, please contact Laurie Arrants, NINDS at *arrantsl@ninds.nih.gov.*

Nucleic Acids and Methods for Expression of the Rat FcεRI beta Subunit, Which Plays a Critical Role in Allergy and the Immune Response

Description of Technology: FcεRI is the high-affinity receptor for the Fc region of immunoglobulin E (IgE), and plays an important role in the allergic response and inflammation. It controls the production of important immunomodulatory molecules, such as cytokines and histamine.

This technology describes nucleic acids encoding the beta subunit of rat FcεRI, as well as vectors and transgenic cells including such nucleic acids. Also described are methods of expressing functional rat FcεRI in a host cell. These may be useful in studies of allergy and the immune response.

Potential Commercial Applications: Research studies of allergy and the immune response.

Development Stage

- Early-stage.
- In vitro data available.

Inventors: Jean-Pierre Kinet and Henry Metzger (NIAMS).

Intell ectual Property: HHS Reference No. E–247–1988/4—U.S. Patent No. 6,165,744 issued 26 Dec 2000.

Licensing Contact: Tara L. Kirby, Ph.D.; 301–435–4426; *tarak@mail.nih.gov.*

Dated: May 14, 2012.

Richard U. Rodriguez,

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

[FR Doc. 2012–12041 Filed 5–17–12; 8:45 am]

BILLING CODE 4140–01–P