provide an addendum to the fourth quarter report to describe estimates used in deriving any expenditures reported in any category.

Respondents: TANF Agencies.

ANNUAL BURDEN ESTIMATES

Instrument	Number of respondents	Number of responses per respondent	Average burden hours per response	Total burden hours
ACF–196	51	4	8	1,632

Estimated Total Annual Burden Hours: 1,632.

Additional Information: Copies of the proposed collection may be obtained by writing to the Administration for Children and Families, Office of Planning, Research and Evaluation, 370 L'Enfant Promenade, SW., Washington, DC 20447, Attn: ACF Reports Clearance Officer. All requests should be identified by the title of the information collection. Email address: infocollection@acf.hhs.gov.

OMB Comment: OMB is required to make a decision concerning the collection of information between 30 and 60 days after publication of this document in the **Federal Register**. Therefore, a comment is best assured of having its full effect if OMB receives it within 30 days of publication. Written comments and recommendations for the proposed information collection should be sent directly to the following: Office of Management and Budget, Paperwork Reduction Project, Email: *OIRA_SUBMISSION@OMB.EOP.GOV,*

Attn: Desk Officer for the Administration for Children and Families.

Robert Sargis,

Reports Clearance Officer.

[FR Doc. 2012–9759 Filed 4–23–12; 8:45 am]

BILLING CODE 4184–01–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.

Small-Molecule Modulators of Lipid Storage for Treatment of Obesity, Atherosclerosis, Metabolic Syndrome and Lipid Storage Diseases

Description of Technology: Lipid droplets are key organelles involved in lipid homeostasis. In normal physiology, these droplets are formed in response to elevated fatty acid levels, and are broken down when needed for energy production. Imbalances in lipid homeostasis trigger compensatory alterations in metabolism that can lead to diseases such as obesity, atherosclerosis, and metabolic syndrome. There are also a number of inherited lipid storage diseases that result in harmful buildup of various lipids, such as Gaucher disease, Fabry disease, and others. Reducing the accumulation of lipid droplets is a promising potential strategy for treatment of such disorders.

This technology describes three novel structural classes of small-molecule compounds that significantly reduce the accumulation of lipid droplets. These compounds hold promise for the treatment of diseases associated with aberrant lipid deposition.

Potential Commercial Applications:

- Treatment of inherited metabolic diseases such as Gaucher disease, Fabry disease, and Tay Sachs disease.
- Treatment of obesity and metabolic disease.
- Treatment of atherosclerosis.

Competitive Advantages: Modulation of lipid droplet accumulation is a novel mechanism for treatment of lipid storage diseases.

Development Stage:

- Early-stage
- In vitro data available

Inventors: Matthew Boxer et al. (NCATS).

Intellectual Property: HHS Reference No. E–277–2011/0—U.S. Provisional Patent Application No. 61/562,894 filed 22 Nov 2011.

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

A Broadly Neutralizing Human Anti-HIV Monoclonal Antibody (10E8) Capable of Neutralizing Most HIV–1 Strains

Description of Technology: This Human Anti-HIV Monoclonal Antibody (10E8) has great potential to provide passive protection from infection, as a therapeutic vaccine, or as a tool for the development of vaccine immunogens. 10E8 is one of the most potent HIV-neutralizing antibodies isolated thus far and it can potently neutralize up to 98% of genetically diverse HIV–1 strains. 10E8 is specific to the membrane-proximal external region (MPER) of the HIV envelope protein, GP41. It is anticipated that 10E8 could be used in combination with another human anti-HIV–1 monoclonal antibody to provide an antibody response that neutralizes nearly all strains of HIV–1. Additionally, 10E8 is a useful tool for the design of vaccine immunogens that can elicit an adaptive immune response to produces 10E8 like antibodies. This technology also includes monoclonal antibodies from the same germ line as 10E8.

Potential Commercial Applications:

- Passive protection to prevent HIV infection
- Passive protection to prevent mother-to-infant HIV transmission
- Topical microbicide to prevent HIV infection
- Gene-based vectors for anti-gp41 antibody expression
- Therapeutic for the elimination of HIV infected cells that are actively producing virus

Competitive Advantages:

- One of the most potent Human broadly-neutralizing anti HIV antibodies isolated to date
- Broad reactivity and high affinity to most HIV–1 strains
- Activity is highly complementary to existing broadly neutralizing antibodies, such as CD4 binding site antibodies
- Capable of neutralizing all HIV–1 strains, if used in combination with another anti-HIV monoclonal antibody

Development Stage:

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

Publication: In press.

Intellectual Property: HHS Reference No. E–253–2011/0—US Provisional Application No. 61/556,660 filed 07 Nov 2011.

Related Technologies:

- HHS Reference No. E–123–2005/1—PCT Application No. PCT/US2006/005613 filed 17 Feb 2006
- HHS Reference No. E–291–2008/0—US Application No. 13/057,414 filed 03 Feb 2011

Licensing Contact: Cristina Thalhammer-Reyero, Ph.D., MBA; 301–435–4507; *thalhamc@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 vaccine immunogens capable of eliciting a 10E8-like adaptive immune response. For collaboration opportunities, please contact Bill Ronnenberg at 301–451–3522 or *wronnenberg@niaid.nih.gov*.

Cytochromes P450 CYP2J and CYP2C Polyclonal Antibodies and Recombinant Proteins

Description of Technology: The National Institutes of Health announces polyclonal antibodies against mouse cytochrome P450s CYP2J and CYP2C. Cytochrome P450s catalyze the metabolism of a wide range of exogenous compounds, including drugs, industrial chemicals, environmental pollutants, and carcinogens. The 2C family of cytochrome P450 metabolizes an extensive number of drugs which include tolbutamide, S-Warfarin, mephenytoin, diazepam and taxol. Many of the P450 enzymes are also active in the NADPH-dependent oxidation of arachidonic acid to various eicosanoids found in several species. The 2J family is expressed at high levels in the heart and has been shown to metabolize both arachidonic acid and linoleic acid. The CYP2J and CYP2C subfamily members have a wide tissue distribution and may be useful as model systems for studies of cardiovascular disease, drug metabolism and toxicity.

Recombinant proteins of mouse cytochrome P450s CYP2C and CYP2J have also been expressed and can be used as controls in immunoblotting, as well as for metabolism studies.

Potential Commercial Applications:

- These antibodies can be used to study the expression of the P450s in various tissues by immunohistochemistry and immunoblotting.
- The recombinant proteins can be used as controls in immunoblotting as well as for metabolism studies.

Competitive Advantages: The CYP2J and CYP2C subfamily members have a wide tissue distribution and may be useful as model systems for studies of cardiovascular disease, drug metabolism and toxicity.

Development Stage: In vitro data available.

Inventors: Darryl C. Zeldin (NIEHS) et al.

Publications: Manuscripts in preparation.

Intellectual Property: HHS Reference No. E–153–2011/0—Research Material. Patent protection has not been pursued for this technology.

Licensing Contact: Fatima Sayyid, M.H.P.M.; 301–435–4521; *Fatima.Sayyid@nih.hhs.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 antibodies against mouse cytochrome P450s CYP2J and CYP2C. For collaboration opportunities, please contact Elizabeth M. Denholm, Ph.D. at denholme@niehs.nih.gov.

Monoclonal Antibodies Targeting Human DNA Polymerase beta, a DNA Repair Enzyme

Description of Technology: Available for licensing are monoclonal antibodies targeting human DNA polymerase beta (Pol B). Pol B is a constitutively expressed ''housekeeping'' enzyme that plays a role in base excision repair (BER), a cellular defense mechanism that repairs DNA base damage and loss. Aberrant Pol B expression is associated with genomic instability indicating that Pol B is required for DNA maintenance, replication and recombination.

These antibodies can be utilized to elucidate BER's mechanism of action and Pol B's structure and function. Moreover, the antibodies can be used to detect Pol B in samples with a variety of techniques including immunoblotting, ELISA, immunoprecipitation, and immunohistochemistry.

Potential Commercial Applications:

- Research tool to elucidate the mechanism of base excision repair
- Research reagents

Competitive Advantages: Can be utilized in a variety of applications to study Pol B.

Development Stage:

- Early-stage
- In vitro data available

Inventors: Samuel Wilson and Rajendra Prasad (NIEHS).

Publications:

1. Srivastava DK, et al. Phorbol ester abrogates up-regulation of DNA polymerase beta by DNA-alkylating agents in Chinese hamster ovary cells. J Biol Chem. 1995 Jul 7;270(27):16402–8. [PMID 7608211]
2. Singhal R, et al. DNA polymerase beta conducts the gap-filling step in uracil-initiated base excision repair in a bovine testis nuclear extract. J Biol Chem. 1995 Jan 13;270(2):949–57. [PMID 7822335]
3. Prasad R, et al. Specific interaction of DNA polymerase beta and DNA ligase I in a multiprotein base excision repair complex from bovine testis. J Biol Chem. 1996 Jul 5;271(27):16000–7. [PMID 8663274]
4. Piersen C, et al. Evidence for an imino intermediate in the DNA polymerase beta deoxyribose phosphate excision reaction. J Biol Chem. 1996 Jul 26;271(30):17811–5. [PMID 8663612]
5. Sobol R, et al. Regulated over-expression of DNA polymerase beta mediates early onset cataract in mice. DNA Repair (Amst). 2003 May 13;2(5):609–22. [PMID 12713817]
6. Poltoratsky V, et al. Down-regulation of DNA polymerase beta accompanies somatic hypermutation in human BL2 cell lines. DNA Repair (Amst). 2007 Feb 4;6(2):244–53. [PMID 17127106]

Intellectual Property: HHS Reference No. E–036–2008/0—Research Tool. Patent protection is not being pursued for this technology.

Licensing Contact: Jennifer Wong; 301–435–4633; *wongje@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 these monoclonal antibodies. For collaboration

opportunities, please contact Elizabeth M. Denholm, Ph.D. at *denholme@niehs.nih.gov*.

Treatment of Acute and Chronic Neurological Disorders Using GLP–1, Exendin-4 and Analogs

Description of Technology: Glucagon-like peptide-1 (GLP–1) and related peptides, including exendin-4 and liraglutide, are incretin mimetics that enhance glucose-dependent insulin secretion following food ingestion as a regulator of glucose homeostasis. Exendin-4 and liraglutide are used clinically in the safe and effective treatment of type 2 diabetes to enhance insulin secretion and maintain a euglycemic state. These actions are primarily mediated at the level of the GLP–1 receptor in the pancreas; however, these compounds are known to enter the brain where the GLP–1 receptor also is expressed.

Researchers at the NIH have discovered the novel use of GLP–1 and exendin-4 analogs in the treatment of acute and chronic neurological disorders and neurodegenerative diseases. Studies conducted in extensive cell culture and in mouse models using these analogs have demonstrated significant neurotrophic and neuroprotective actions in models of several disorders, including Alzheimer's disease, Parkinson's disease, Huntington's disease, ALS, stroke, head trauma and peripheral neuropathy. These studies have now been extensively published and independently validated by other scientific groups. Furthermore, clinical studies are ongoing to evaluate the use of GLP–1 receptor agonists for the treatment of early Alzheimer's disease, Parkinson's disease and diabetic neuropathy by several groups within the US and Europe.

Potential Commercial Applications: Therapeutics for:

- Neurodegenerative diseases—Alzheimer's, Huntington's, Parkinson's, ALS
- Stroke
- Head trauma (traumatic brain injury)
- Peripheral neuropathies

Competitive Advantages:

- Compounds reduce neuronal cell death, amyloid deposition and neuroinflammation while promoting neurogenesis.
- Compounds in this class have already been shown to be safe and effective for other indications.
- Extensive in vitro and animal data are available, and clinical studies are ongoing.
- There are extensive publications in the literature, both from the inventors and independent groups.

Development Stage:

- Pre-clinical
- Clinical
- In vitro data available
- In vivo data available (animal)
- In vivo data available (human)

Inventors: Nigel Greig, Harold Halloway, Maire Doyle, Josephine Egan (all of NIA).

Publications:

1. Li Y, et al. Exendin-4 ameliorates motor neuron degeneration in cellular and animal models of amyotrophic lateral sclerosis. PLoS One. 2012;7(2):e32008. [PMID 22384126]
2. Li Y, et al. Enhancing the GLP–1 receptor signaling pathway leads to proliferation and neuroprotection in human neuroblastoma cells. J Neurochem. 2010 Jun;113(6):1621–1631. [PMID 20374430]
3. Li Y, et al. GLP–1 receptor stimulation reduces amyloid-beta peptide accumulation and cytotoxicity in cellular and animal models of Alzheimer's disease. J Alzheimers Dis. 2010;19(4):1205–1219. [PMID 20308787]
4. Li Y, et al. GLP–1 receptor stimulation preserves primary cortical and dopaminergic neurons in cellular and rodent models of stroke and Parkinsonism. Proc Natl Acad Sci USA. 2009 Jan 27;106(4):1285–1290. [PMID 19164583]
5. Martin B, et al. Exendin-4 improves glycemic control, ameliorates brain and pancreatic pathologies and extends survival in a mouse model of Huntington's disease. Diabetes. 2009 Feb;58(2):318–328. [PMID:18984744]
6. Perry T, et al. Evidence of GLP–1-mediated neuroprotection in an animal model of pyridoxine-induced peripheral sensory neuropathy. Exp Neurol. 2007 Feb;203(2):293–301. [PMID 17125767]
7. Perry T, Greig NH. Enhancing central nervous system endogenous GLP–1 receptor pathways for intervention in Alzheimer's disease. Curr Alzheimers Res. 2005 Jul;2(3):377–385. [PMID 15974903]
8. Greig NH, et al. New therapeutic strategies and drug candidates for neurodegenerative diseases: p53 and TNF-alpha inhibitors, and GLP–1 receptor agonists. Ann NY Acad Sci. 2004 Dec;1035:290–315. [PMID 15681814]
9. Perry TA, Greig NH. A new Alzheimer's disease interventive strategy: GLP–1. Curr Drug Targets. 2004 Aug;5(6):565–571. [PMID 15270203]

Listing of additional related publications available upon request.

Intellectual Property: HHS Reference No. E–049–2001/0—

- U.S. Patent 7,576,050 issued 18 Aug 2009
- U.S. Patent Application No. 12/317,042 filed 18 Dec 2008
- Foreign counterparts in Australia, Canada, Europe, India, and Japan

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

Collaborative Research Opportunity: The National Institute on Aging, Drug Design and Development Section, is seeking statements of capability or interest from parties interested in collaborative research to further develop, evaluate or commercialize this technology. For collaboration opportunities, please contact Vio Conley at *conleyv@mail.nih.gov*.

Dated: April 18, 2012.

Richard U. Rodriguez,

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

[FR Doc. 2012–9776 Filed 4–23–12; 8:45 am]

BILLING CODE 4140–01–P

DEPARTMENT OF HEALTH AND HUMAN SERVICES

National Institutes of Health

Government-Owned Inventions; Availability for Licensing: Mouse Models

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 for the technologies 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–