

- Texas; Court of Federal Claims Number 02–1279V
401. Jeanne and Brian Rippentrop on behalf of Anthony Thomas Rippentrop; Dallas, Texas; Court of Federal Claims Number 02–1280V
402. Dana and Lee Halvorson on behalf of Robyn Leigh Halvorson; Dallas, Texas; Court of Federal Claims Number 02–1281V
403. Margaret Springer on behalf of Jonah Springer; Boston, Massachusetts; Court of Federal Claims Number 02–1284V
404. Vera A. Easter on behalf of Jordan Delaney Easter; Dallas, Texas; Court of Federal Claims Number 02–1285V

Dated: January 28, 2003.

Elizabeth M. Duke,
Administrator.

[FR Doc. 03–2658 Filed 2–4–03; 8:45 am]

BILLING CODE 4165–15–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, DHHS.

ACTION: Notice.

SUMMARY: The inventions listed below are owned by agencies 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.

Fibroblast Growth Factor 3 (FGFR3) Receptor Knockin Mice

Dr. Chuxia Deng (NIDDK), DHHS Reference No. E–060–2003/0—Research Tool.

Licensing Contact: Marlene Shinn-Astor; 301/435–4426; shinnm@od.nih.gov.

Missense mutations in Fibroblast Growth Factor Receptor 3 (FGFR3) result in several human skeletal dysplasias, including the most common form of dwarfism, achondroplasia.

The NIH announces the generation of FGFR3 knockin mice, which have a Gly369Cys mutation, inserted into the mouse genome. Phenotypic analysis of the mice reveals that the FGF/FGFR3 signals affect both chondrogenesis and osteogenesis by regulating Stat proteins and cell-cycle inhibitors, and the activities of chondrocytes, osteoclasts, and osteoblasts during endochondral ossification. These mice provide a new animal model to study functions of FGF/FGFR3 signals in achondroplasia patients, which could lead to new drug discovery and therapeutic treatments.

Compositions and Methods for Inhibiting Group B Streptococcal-Induced Pulmonary Hypertension in Neonates

Rodney L. Levine et al. (NHLBI), DHHS Reference No. E–259–2002/0 filed Oct. 15, 2002.

Licensing Contact: Susan Ano; 301/435–5515; anos@od.nih.gov.

Group B streptococcus (GBS), the most common cause of sepsis and meningitis in human newborns, often results in respiratory distress. The underlying cause of this distress is pulmonary hypertension, historically believed to be induced by increased production of thromboxane A2 as stimulated by GBS. The technology described here reveals that the phospholipids cardiolipin and phosphatidylglycerol are causative agents of GBS-induced pulmonary hypertension. Furthermore, the technology describes administration of these phospholipids or immunogenic fragments thereof in an appropriate fashion to elicit an immune response, including administration as conjugates to hapten to enhance the binding selectivity of the resulting antibodies. Additionally, administration of antibodies to these phospholipids for the same purpose is related. The phospholipids or immunogenic fragments can also be administered in a dose-dependent manner to increase blood pressure in pulmonary arteries. Kits for administration of these phospholipids and/or anti-phospholipid antibodies are also described. The standard treatment for GBS infection is the penicillin class of antibiotics, which increases the synthesis and excretion of the two phospholipids revealed in this technology to cause pulmonary hypertension. Thus, the current technology offers a potential improvement over existing treatments.

In the course of the research that led to the above discovery, a method of separating recombinantly expressed membrane-bound proteins and membrane-associated endotoxin in gram-negative prokaryote expression systems was also developed.

p-Toluenesulfonhydrazide Derivatization for Separation and Measurement of Endogenous Estrogen Metabolites by High-Pressure Liquid Chromatography-Electrospray-Mass Spectrometry

Dr. Xia Xu (NCI), U.S. Provisional Patent Application 60/372,848 filed Apr. 15, 2002.

Licensing Contact: Brenda Hefti; 301/435–4632; heftib@od.nih.gov.

The current invention relates to a method for measuring endogenous estrogen levels, and this technology may be generalizable to all endogenous ketolic steroids, including estrogens, androgens, and phytoestrogens.

Specifically, the current invention is a derivatization technique that forms estrogen-p-toluenesulfonhydrazones, which can be separated and then measured using high-pressure liquid chromatography-electrospray-mass spectrometry (HPLC–ESI–MS). This method offers a number of improvements over current methods. It is more sensitive, it is faster, it is more accurate, and it requires a smaller sample size.

FXR/BAR Knockout Mouse Model

Frank Gonzalez (NCI), DHHS Reference No. E–323–2001/0—Research Tool.

Licensing Contact: Marlene Shinn-Astor; 301/435–4426; shinnm@od.nih.gov.

Cholesterol lowering drugs are being prescribed more and more as a way to combat high cholesterol levels associated as a precursor to heart disease. The NIH announces a new knockout mouse model that lacks the nuclear receptor FXR/BAR (bile acid receptor). The receptor controls the synthesis and transport of bile salts, which are degradation products of cholesterol. These mice could, therefore, be used to test new targets for cholesterol lowering drugs that use a new mechanism which is distinct from the current statin drugs that control HMG CoA reductase.

Telomerase Immortalized Hepatocyte Cell Lines

Xin W. Wang and Curtis Harris (NCI), DHHS Reference No. E–251–2000/0—US–01 filed Dec. 14, 2000.

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