[FR Doc. 04–1263 Filed 1–21–04; 8:45 am]
BILLING CODE 4160–01–C

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 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.

Inhibitors of Formation of Protease Resistant Prion Protein

Bruce Chesebro, Byron Caughey, Joelle Chabry, Susette Priola (NIAID). U.S. Patent 6,211,149 issued on 03 Apr 2001 (DHHS Reference No. E–189–1998/0–US–02); U.S. Patent 6,355,610 issued on 12 Mar 2002 (DHHS Reference No. E–189–1998/0–US–03); U.S. Patent Application No. 10/096,080 filed 11 Mar 2002 (DHHS Reference No. E–189–1998/0–US–04).

Licensing Contact: Michael Ambrose; 301/594–6565; *ambrosem@mail.nih.gov*.

Protease-resistant prion proteins are actively associated with various transmissible spongiform encephalopathies (TSEs). These include Creutzfeldt-Jakob disease in humans and Bovine spongiform encephalopathy (''mad cow disease'') in cattle.

The present invention discloses proprietary peptides and potential pharmaceutical compositions using such peptides that inhibit the formation of protease-resistant prion protein aggregates. These aggregates develop into amyloid deposits in the brain of affected patients, leading to the development of the spongiform encephalopathy. The peptides, when used in vitro inhibit such aggregation. Furthermore, when used in pharmaceutical compositions and medically relevant dosages, may be used for therapies for TSEs.

Inhibitors of Amyloid Formation

Winslow S. Caughey, Byron Caughey, Lynne D. Raymond, Motohiro Horiuchi (NIAID). U.S. Patent 6,632,808 issued on 14 Oct 2003 (DHHS Reference No. E–205–1998/0–US–03).

Licensing Contact: Michael Ambrose; 301/594–6565; *ambrosem@mail.nih.gov*.

This invention discloses methods, compounds and compositions for therapeutic treatment of amyloidogenic diseases, like Alzheimer's disease, type 2 diabetes and, particularly, transmissible spongiform encephalopathies (prion diseases) such as CJD, Kuru in humans and BSE (''Mad Cow Disease'') in cattle.

The invention is based on the findings that cyclic tetrapyrroles and derivatives inhibit the formation of protease-resistant prion protein (PrP-res) the pathologic, amyloidogenic protein aggregates of the prion diseases. These methods and compounds have the potential for the development of pharmaceutical therapies for the treatment and prevention of progression of such TSEs.

Inhibition of Diseases Associated With Amyloid Formation

Byron Caughey, Richard E. Race (NIAID).

U.S. Patent 5,276,059 issued on 04 Jan 1994 (DHHS Reference No. E–107–1992/0–US–01).

Licensing Contact: Michael Ambrose; 301/594–6565; *ambrosem@mail.nih.gov*.

Amyloid deposition in brain samples is diagnostic for several serious and fatal diseases. These include Alzheimer's disease as well as several transmissible spongiform encephalopathies (prion diseases) such as CJD and BSE (''Mad Cow Disease''). Together, these diseases having amyloid depositions are termed amyloidogenic diseases.

This invention covers and discloses the method and compositions of using Congo Red in the treatment of such amyloidogenic diseases. Congo Red is shown to inhibit the accumulation of PrP-res, the amyloidogenic and pathologic protein or the transmissible spongiform encephalopathies. The potential therapeutics covered by this invention includes Congo Red and its derivatives.

Dated: January 14, 2004.

Steven M. Ferguson,

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

[FR Doc. 04–1258 Filed 1–21–04; 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.

Codon-Optimization of the HIV–1 Vif Gene

Klaus Strebel, Stephan Bour, Kim-Lien Nguyen (NIAID); DHHS Reference No. E–041–2004/0—Research Tool/Biological Material; Licensing Contact: Michael Ambrose; 301/594–6565; *ambrosem@mail.nih.gov*.

Expression of the HIV–1 Vif protein in the absence of other viral factors such a Tat and Rev is extremely inefficient due to the presence of inhibitory sequences on its mRNA. This invention uses codon optimization to remove such inhibitory sequences without altering the amino acid sequence of the protein. The modified vif gene in the resulting pcDNA -hVIF vector is expressed under the control of the CMV promoter. In this, the protein functions as wild type and is more amendable to high-level expression in mammalian cells.

Currently this vector is used in on-going studies of HIV infection and its