cumulative list of orphan drug and biological designations. This list includes the name of the drug or biological, the specific disease/condition for which the drug or biological is designated, and information about the sponsor such as the name, address, telephone, and contact.

At the end of each calendar year, the agency publishes a cumulative list of orphan drug and biological designations current through the calendar year. The list that is the subject of this notice is the cumulative list of orphan drug and biological designations through December 31, 2000, and, therefore, brings the March 1, 2000 (65 FR 11066) publication up to date. This list is available upon request from the Dockets Management Branch (address above). Those requesting a copy should specify Docket No. 84N–0102, which is the docket number for this notice. In addition, the list is updated monthly and is available upon request from OPD or the FDA's Dockets Management Branch (address above). The current list is also available on the Internet at http://www.fda.gov/orphan.

The orphan designation of a drug or biological applies only to the sponsor who requested the designation. Each sponsor interested in developing a drug or biological for an orphan indication must apply for orphan designation in order to obtain exclusive marketing rights. Any request for designation must be received by FDA before the submission of a marketing application for the proposed indication for which designation is requested (21 CFR 316.23). Copies of the orphan drug regulations (21 CFR part 316) (57 FR 62076, December 29, 1992) and explanatory background materials for use in preparing an application for orphan designation may be obtained from OPD (address above).

The names of the drugs and biologicals shown in the cumulative list of orphan designations may change upon marketing approval/licensing, reflecting the established, proper name approved by FDA. Because drugs and biologicals not approved/licensed for marketing are investigational, the appropriate established, proper name has not necessarily been assigned.

Dated: March 27, 2001.

Ann M. Witt,

Acting Associate Commissioner for Policy.

[FR Doc. 01–8061 Filed 4–2–01; 8:45 am]

BILLING CODE 4160–01–S

DEPARTMENT OF HEALTH AND HUMAN SERVICES

Food and Drug Administration

Consumer Briefing on Bovine Spongiform Encephalopathy (BSE) and Transmissible Spongiform Encephalopathies (TSE)

AGENCY: Food and Drug Administration, HHS.

ACTION: Notice of meeting.

The Food and Drug Administration (FDA) is announcing the following consumer meeting: Consumer Briefing on Bovine Spongiform Encephalopathy (BSE) and Transmissible Spongiform Encephalopathies (TSE). This briefing is the first in a series of consumer briefings on the consumer protection priorities discussed by the agency and consumers at the December 13, 2000, Consumer Roundtable on Consumer Protection Priorities meeting. These consumer briefings enable the agency and consumers to sustain a dialogue on FDA priorities of high consumer interest in the spirit of openness, transparency, and participation. This consumer briefing will provide an update on FDA's efforts to ensure the safety of products that may contain or are manufactured with bovine-derived ingredients.

Date and Time: The briefing will be held on April 16, 2001, 1 p.m. to 4:30 p.m. Registration will open at 12 noon.

Location: The briefing will be held at Holiday Inn Capitol, Columbia II, 550 C St., SW., Washington, DC.

Contact: Karen R. Mahoney, Office of Consumer Affairs (HFE–88), Food and Drug Administration, 5600 Fishers Lane, Rockville, MD 20857, 301–827–4393, FAX 301–827–2866, e-mail: Kmahoney@oc.fda.gov.

Registration: Preregistration is required as space is very limited. Send registration information (including name, title, organization/firm name, address, telephone, fax number and e-mail) to the contact person by April 13, 2001. Preregistered consumer attendees will be given first priority for seating.

If you need any special accommodations due to disability, please contact Karen R. Mahoney (address above) by April 13, 2001.

Transcripts: Transcripts of the meeting may be requested in writing from the Freedom of Information Office (HFI–35), Food and Drug Administration, 5600 Fishers Lane, rm. 12A–16, Rockville, MD 20857, approximately 15 working days after the meeting at a cost of 10 cents a page.

SUPPLEMENTARY INFORMATION: The consumer briefing is an opportunity for the agency to meet with consumers and to discuss issues and concerns as well as how FDA and consumers can work together to keep consumers informed and involved.

Procedure: The briefing is open to the public. There will be an open public session at the conclusion of the briefing where interested persons can respond to the topics and issues discussed during the briefing.

Dated: March 27, 2001.

Ann M. Witt,

Acting Associate Commissioner for Policy.

[FR Doc. 01–8062 Filed 4–2–01; 8:45 am]

BILLING CODE 4160–01–S

DEPARTMENT OF HEALTH AND HUMAN SERVICES

National Institutes of Health

National Institute of Diabetes and Digestive and Kidney Diseases (NIDDK): Opportunity for Cooperative Research and Development Agreements (CRADAs) To Identify Novel Candidate Genes for Obesity and Insulin Resistance Using Global Gene Expression Profiling

AGENCY: National Institutes of Health, Public Health Service, DHHS.

ACTION: Notice.

SUMMARY: The National Institute of Diabetes and Digestive and Kidney Diseases (NIDDK) of the National Institutes of Health (NIH) announces the opportunity for Cooperative Research and Development Agreements (CRADAs) to identify novel candidate genes for obesity and insulin resistance using global gene expression profiling. The NIH seeks potential Collaborator(s) wishing to provide expertise in (1) identification of genes that may contribute to the development of obesity; (2) identification of genes that may contribute to the development of insulin resistance; (3) characterization of potentially novel sub-pathways of insulin signaling mechanisms; and (4) identification of genes regulated by free-fatty acid.

The NIDDK seeks capability statements from parties interested in entering into a potential CRADA to identify novel candidate genes for obesity and insulin resistance using global gene expression profiling. Collaborator applicants developing capability statements may also include proposals to provide funding for possible commercial uses of interest to the Collaborator. The availability of private sector support may increase the feasibility of particular aspects of the

final design, but the primary criterion for selecting potential Collaborator(s) is the scientific merit of proposals for developing a plan to identify novel candidate genes for obesity and insulin resistance using global gene expression profiling.

The control of clinical trials shall reside entirely with the Institute and the scientific participants of the trial. In the event that any adverse effects are encountered which, for legal or ethical reasons, may require communication with the FDA, the relevant collaborating institutions will be notified. Neither the conduct of the trial nor the results should be represented as an NIDDK endorsement of the drug under study.

DATES: Only written CRADA capability statements received by the NIDDK on or before May 1, 2001 will be considered during the initial design phase, confidential information must be clearly labeled. Potential Collaborators may be invited to meet with the Selection Committee at the Collaborator's expense to provide additional information. The Institute may issue an additional notice of CRADA opportunity during the design phase if circumstances change or if the design alters substantially.

FOR FURTHER INFORMATION CONTACT: Capability statements should be submitted to Dr. Michael W. Edwards, Office of Technology Development, National Institute of Diabetes and Digestive and Kidney Diseases, National Institutes of Health, BSA Building, Suite 350 MSC 2690, 9190 Rockville Pike, Bethesda, MD 20814–3800; Tel: 301/496–7778, Fax: 301/402–0535; Email: me1s@nih.gov.

SUPPLEMENTARY INFORMATION: Substantial evidence indicates that susceptibility to type 2 diabetes is largely genetically determined, especially in certain ethnic groups in which the prevalence of diabetes may be 10 times that of the general U.S. population. NIDDK has performed genomic linkage scans in subject populations and are planning to positionally clone diabetes susceptibility genes. In general, diabetes is not inherited as a simple Mendelian trait. Multiple genes with small to moderate effects are likely to contribute to the development of the diabetes. In most populations, obesity and insulin resistance precede and predict the development of type 2 diabetes. These traits are themselves highly heritable, suggesting that they have a substantial genetic basis. Genes influencing these metabolic precursors of type 2 diabetes may be fewer in number and, therefore, easier to identify than those contributing to the overall syndrome. An extensive study in the subject population has indicated several chromosomal regions that provide evidence for linkage not only to diabetes but also to pre-diabetic phenotypes. We plan to perform gene expression profiling experiments to identify susceptibility genes for obesity and insulin resistance that may serve as possible targets of intervention.

Capability Statements

A Selection Committee will utilize the information provided in the ''Collaborator Capability Statements'' received in response to this announcement to help in its deliberations. It is the intention of the NIDDK that all qualified Collaborators have the opportunity to provide information to the Selection Committee through their capability statements. The Capability Statement should not exceed 10 pages and should address the following selection criteria:

(1) The statement should provide specific details of the method to be utilized in the development of novel candidate genes for obesity and insulin resistance using global gene expression profiling.

(2) The statement should include a detailed plan demonstrating the ability to provide sufficient capacity using global gene expression profiling.

(3) The statement may include outline outcome measures of interest to the Collaborator. The specifics of the proposed outcome measures and the proposed support should include but not be limited to the following: global gene expression profiling expertise, specific funding commitment to support the advancement of scientific research, personnel, services, facilities, equipment, or other resources that would contribute to the conduct of the commercial development.

(4) The statement must address willingness to promptly publish research results and ability to be bound by PHS intellectual property policies (see CRADA: http://ott.od.nih.gov/NewPages/crada.pdf).

Dated: March 23, 2001.

Jack Spiegel,

Director, Division of Technology Development and Transfer Office of Technology Transfer.

[FR Doc. 01–8085 Filed 4–2–01; 8:45 am]

BILLING CODE 4140–01–M

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.

Methods and Compositions for Analysis of M3 Muscarinic Acetylcholine Receptors

Jurgen Wess, Masahisa Yamada (NIDDK), DHHS Reference No. E–291–00/0 filed 30 Oct 2000, Licensing Contact: John Rambosek; 301/496–7056 ext. 270; e-mail: rambosej@od.nih.gov

This invention discloses transgenic mice that have the M3 Muscarinic Acetylcholine Receptor deleted by gene knockout technology. These mice were developed in order to better understand the physiological relevance of the M3 receptor. Unexpectedly, these knockout mice have a phenotype that includes significant reduction in food intake, weight loss, peripheral fat deposits, as well as very low serum leptin and insulin levels. It was also found that the M3 receptor is highly expressed in the hypothalamus, a region of the brain known to be critically involved in regulation of food uptake. The mice also show physiological changes (increased levels of hypothalmic agouti-related peptide mRNA and decreased expression of propiomelanocortin mRNA) consistent with those observed in fasted animals. However, the knockout mice also have changes