

The number of respondents in Table 1 of this document is the number of sponsors registered to make electronic submissions (25). The number of total annual responses is based on a review of the actual number of such submissions made between July 1, 2005, and June 30, 2006. 103 x hours per response (.20) = 20.6 total hours.

Dated: November 2, 2006.

Jeffrey Shuren,

Assistant Commissioner for Policy.

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DEPARTMENT OF HEALTH AND HUMAN SERVICES

Food and Drug Administration

[Docket No. 2006N-0434]

Agency Information Collection Activities; Proposed Collection; Comment Request; Guidance For Industry on How to Use E-Mail to Submit a Request for a Meeting or Teleconference to the Office Of New Animal Drug Evaluation

AGENCY: Food and Drug Administration, HHS.

ACTION: Notice.

SUMMARY: The Food and Drug Administration (FDA) is announcing an opportunity for public comment on the proposed collection of certain information by the agency. Under the Paperwork Reduction Act of 1995 (the PRA), Federal agencies are required to publish notice in the **Federal Register** concerning each proposed collection of information, including each proposed extension of an existing collection of information, and to allow 60 days for public comment in response to the notice. This notice solicits comments on extending Office of Management and Budget (OMB) approval of existing reporting requirements on electronic submission of requests for meetings, in person or via teleconference, to discuss

with animal drug sponsors studies to be conducted and how to meet the statutory requirements for drug approval under the Federal Food, Drug, and Cosmetic Act. Requests for meetings about new animal drug submissions were previously submitted on paper copy to the Center for Veterinary Medicine (CVM).

DATES: Submit written or electronic comments on the collection of information by January 8, 2007.

ADDRESSES: Submit electronic comments on the collection of information to: <http://www.fda.gov/dockets/ecomments>. Submit written comments on the collection of information to the Division of Dockets Management (HFA-305), Food and Drug Administration, 5630 Fishers Lane, rm. 1061, Rockville, MD 20852. All comments should be identified with the docket number found in brackets in the heading of this document.

FOR FURTHER INFORMATION CONTACT: Denver Presley, Jr., Office of the Chief Information Officer (HFA-250), Food and Drug Administration, 5600 Fishers Lane, Rockville, MD 20857, 301-827-1472.

SUPPLEMENTARY INFORMATION: Under the PRA (44 U.S.C. 3501-3520), Federal agencies must obtain approval from OMB for each collection of information they conduct or sponsor. "Collection of information" is defined in 44 U.S.C. 3502(3) and 5 CFR 1320.3(c) and includes agency requests or requirements that members of the public submit reports, keep records, or provide information to a third party. Section 3506(c)(2)(A) of the PRA (44 U.S.C. 3506(c)(2)(A)) requires Federal agencies to provide a 60-day notice in the **Federal Register** concerning each proposed collection of information, including each proposed extension of an existing collection of information, before submitting the collection to OMB for approval. To comply with this requirement, FDA is publishing notice

of the proposed collection of information set forth in this document.

With respect to the following collection of information, FDA invites comments on these topics: (1) Whether the proposed collection of information is necessary for the proper performance of FDA's functions, including whether the information will have practical utility; (2) the accuracy of FDA'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 respondents, including through the use of automated collection techniques, when appropriate, and other forms of information technology.

How to Use E-Mail to Submit a Request for a Meeting or Teleconference to the Office Of New Animal Drug Evaluation—21 CFR 10.65 (OMB Control Number—(0910-0452)—Extension

CVM holds meetings and/or teleconferences when a sponsor requests a presubmission conference under 21 CFR 514.5, or requests a meeting to discuss general questions. Generally, meeting requests are submitted to CVM on paper. However, CVM now allows registered sponsors to submit information electronically, and to request meetings electronically, if they determine this is more efficient and time saving for them. CVM's guidance entitled "How to Use E-Mail to Submit a Request for a Meeting or Teleconference to the Office of New Animal Drug Evaluation" provides sponsors with the option to submit a request for a meeting or teleconference as an e-mail attachment by the Internet.

The likely respondents are sponsors for new animal drug applications.

CVM estimates the burden for this information collection activity as follows:

TABLE 1.—ESTIMATED ANNUAL REPORTING BURDEN¹

21 CFR Section/FDA Form #	No. of Respondents	Annual Frequency per Response	Total Annual Responses ²	Hours per Respondent	Total Hours
10.65/FDA Form 3489	25	6.24	156	.08	12.5

¹ There are no capital costs or operating and maintenance costs associated with this collection of information.

² Electronic submissions received between July 1, 2005, and June 30, 2006.

The number of respondents in table 1 of this document is the number of sponsors registered to make electronic

submissions (25). The number of total annual responses is based on a review of the actual number of such

submissions made between July 1, 2005, and June 30, 2006. (156 x hours per response (.08) = 12.5 total hours).

Dated: November 2, 2006.

Jeffrey Shuren,

Assistant Commissioner for Policy.

[FR Doc. E6-18911 Filed 11-7-06; 8:45 am]

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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.

Mice Lacking Expression of Chemokine Receptor CCR9 Generated by Gene Targeting (CCR9 KO Mice)

Description of Technology: Chemokines and their receptors are key regulators of thymocytes migration and maturation in normal and inflammation conditions. The chemokine CCL25 is highly expressed in the thymus and small intestine. CCR9, the receptor for CCL25, is expressed on the majority of thymocytes, indicating that CCR9 and its ligand may play an important role in thymocyte development. To investigate the role of CCR9 during lymphocyte development, CCR9 knockout mice were developed. Knockout mice had increased numbers of peripheral $\gamma\delta$ -T cells but reduced numbers of $\alpha\beta$ -T cells. In competitive transplantation experiments bone marrow from CCR9 knockout mice was much less efficient at repopulating the thymus than control (wild type) bone marrow. Thus, CCR9 KO mice are a model for studying

thymocyte development and trafficking in the body. Additionally, as the ligand for CCR9 is highly expressed in the small intestine, CCR9 potentially plays a role in the specialization of immune responses in the gastrointestinal tract.

Applications: (1) Evaluate drugs aimed at blocking or augmenting lymphocyte trafficking; (2) A model for studying T cell development; (3) A model for studying immunological based gastrointestinal disorders.

Inventors: Paul E. Love (NICHD), Joshua M. Farber (NIAID), Shoji Uehara (NICHD).

Publications:

1. S Uehara *et al.* A role for CCR9 in T lymphocyte development and migration. *J Immunol.* 2002 Mar 15;168(6):2812-2819.

2. S Uehara *et al.* Characterization of CCR9 expression and CCL25/thymus-expressed chemokine responsiveness during T cell development: CD3^{high}CD69⁺ thymocytes and $\gamma\delta$ TCR⁺ thymocytes preferentially respond to CCL25. *J Immunol.* 2002 Jan 1;168(1):134-142.

Patent Status: HHS Reference No. E-328-2006/0—Research Tool.

Licensing Status: This technology is available as a research tool under a Biological Materials License.

Licensing Contact: Jennifer Wong; 301/435-4633; wongje@mail.nih.gov.

mFPR2 Transgenic and Knockout Mouse Models for Alzheimer's and Other Inflammatory Diseases

Description of Technology: Human Formyl Peptide-Like Receptor 1 (hFPLR1) has been implicated in host defense for disease processes including Alzheimer's disease, infection, and other inflammatory diseases. hFPLR1 and its mouse homologue Formyl Peptide Receptor 2 (mFPR2) are G-protein coupled receptors that are expressed at high levels on phagocytic leukocytes, mediating leukocyte chemotaxis and activation in response to a number of pathogen- and host-derived peptides. Activation of hFPLR1/mFPR2 by lipoxin A4 may play a role in preventing and resolving inflammation. Also, hFPLR1/mFPR2 has been shown to mediate the chemotactic activity of amyloid β 1-42, a key pathogenic peptide in Alzheimer's disease.

Available for licensing are mice expressing the mFPR2 transgene on either the FVB or C58BL background, as well as mFPR2 knockout mice on the C57BL background. These mice are anticipated to be highly useful in the study of a wide variety of inflammatory, infectious, immunologic and neurodegenerative diseases.

Applications: (1) Drug development model for Alzheimer's disease and other inflammatory diseases; (2) Tool to probe the role of hFPLR1/mFPR2 in host responses in a variety of disease processes, including inflammatory, infectious, immunologic, and neurodegenerative disease.

Inventors: Ji Ming Wang *et al.* (NCI).

Related Publications:

1. K Chen, P Iribarren, J Hu, J Chen, W Gong, EH Cho, S Lockett, NM Dunlop, and JM Wang. Activation of Toll-like receptor 2 on microglia promotes cell uptake of Alzheimer disease-associated amyloid beta peptide. *J Biol Chem.* 2006 Feb 10;281(6):3651-3659.

2. H Yazawa, ZX Yu, Takeda, Y Le, W Gong, VJ Ferrans, JJ Oppenheim, CC Li, and JM Wang. Beta amyloid peptide (A β 42) is internalized via the G-protein-coupled receptor FPRL1 and forms fibrillar aggregates in macrophages. *FASEB J.* 2001 Nov;15(13):2454-2462.

3. YH Cui, Y Le, W Gong, P Proost, J Van Damme, WJ Murphy, and JM Wang. Bacterial lipopolysaccharide selectively up-regulates the function of the chemotactic peptide receptor formyl peptide receptor 2 in murine microglial cells. *J Immunol.* 2002 Jan 1;168(1):434-442.

Patent Status: HHS Reference No. E-303-2006/0—Research Tool.

Licensing Status: This technology is available as a research tool under a Biological Materials License.

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

Collaborative Research Opportunity: The National Cancer Institute—Frederick, Laboratory of Molecular Immunoregulation, is seeking statements of capability or interest from parties interested in collaborative research to further develop, evaluate, or commercialize mFPR2 Transgenic and Knockout Mouse Models for Alzheimer's and Other Inflammatory Diseases. Please contact Betty Tong, Ph.D. at 301-594-4263 or tongb@mail.nih.gov for more information.

Vaccine Production Strain for Acellular Pertussis Vaccine

Description of Technology: Available for licensing from the NIH is a vaccine production strain of *Bordetella bronchiseptica* that produces *Bordetella pertussis* toxin in high yield. The *Bordetella bronchiseptica* strain has been modified to eliminate expression of filamentous hemagglutinin, which typically has to be removed in purification of the toxin, thereby