

2003 (DHHS Reference No. E-314-2002/0-US-01); PCT Application No. PCT/US04/06703 filed 05 Mar 2004, which published as WO 2004/081179 A2 on 10 Feb 2005 (DHHS Reference No. E-314-2002/0-PCT-02); *Licensing Contact*: Jesse S. Kindra; (301) 435-5559; [kindraj@mail.nih.gov](mailto:kindraj@mail.nih.gov).

This invention relates to the discovery that tristetraprolin (TTP) can promote the poly(A)RNase (PARN) mediated deadenylation of polyadenylated substrates containing AU-rich elements (AREs). As one aspect of the invention, the inventors have developed a cell free system that may be used for the purposes of assessing the effects of the various system components or their derivatives (*i.e.* AREs, PARN, or TTP) on the deadenylation process or the effects of various test agents on the deadenylation process. Aspects of this work have been published as follows: Lai *et al.*, 2003, Tristetraprolin and Its Family Members Can Promote the Cell-Free Deadenylation of AU-Rich Element-Containing mRNAs by Poly(A) Ribonuclease, *MCB* 23(11):3798-3812.

This technology is available for licensing on an exclusive or a non-exclusive basis.

In addition to licensing, the technology is available for further development through collaborative research opportunities with the inventors.

#### **Tristetraprolin (TTP) Knockout Mice**

Perry Blackshear *et al* (NIEHS). DHHS Reference No. B-015-1999/0—Research Material.

*Licensing Contact*: Michelle A. Booden; 301/451-7337; [boodenm@mail.nih.gov](mailto:boodenm@mail.nih.gov).

National Institutes of Health researchers have developed knockout mice that do not express Tristetraprolin (TTP). TTP is an AU-rich element (ARE) binding protein and the prototype of a family of CCCH zinc finger proteins. AREs were identified as conserved sequences found in the 3' untranslated region (3' UTR) of a variety of transiently expressed genes including early response genes, proto-oncogenes, and other growth regulatory genes. AREs function as instability sequences that target ARE-containing transcripts for rapid mRNA decay. TTP functions by binding directly to the ARE sequence contained in the TNF-alpha mRNA, which destabilizes and mediates rapid decay of the TNF-alpha mRNA. More recent studies demonstrate TTP's ability to downregulate IL-2 gene expression.

TTP knockout mice appear normal at birth but soon develop inflammatory arthritis, dermatitis, cachexia, autoimmunity, and myeloid

hyperplasia. Almost all aspects of these phenotypes can be prevented with repeated injections of antibodies to TNF. Moreover, macrophages isolated from these mice exhibit increased production of TNF-alpha and increased amounts of TNF-alpha mRNA.

This transgenic mouse model will be valuable in advancing our understanding of the mechanisms controlling mRNA turnover in immune homeostasis as well as autoimmune diseases. This model will also permit the development of screening assays to elucidate the functions and binding partners for other members of the CCCH zinc finger family as well as compounds capable of inhibiting aberrant TNF-alpha and IL-2 biosynthesis. Lastly, this model will advance understanding of the pathogenetic role for IL-2 and/or TNF in various autoimmune and inflammatory diseases. The mice will be made available on a non-exclusive basis under a Biological Materials License Agreement.

Background scientific detail may be found in *Immunol.* 2005 Jan 15; 174(2):953-61; *Arthritis Res Ther.* 2004; 6(6):248-64; and *Science.* 1998 Aug 14; 281(5379):1001-5.

Dated: May 23, 2005.

#### **Steven M. Ferguson,**

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

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**BILLING CODE 4140-01-P**

## **DEPARTMENT OF HEALTH AND HUMAN SERVICES**

### **National Institutes of Health**

#### **National Center on Minority Health and Health Disparities; Notice of Closed Meeting**

Pursuant to section 10(d) of the Federal Advisory Committee Act, as amended (5 U.S.C. Appendix 2), notice is hereby given of the following meeting.

The meeting will be closed to the public in accordance with the provisions set forth in sections 552b(c)(4) and 552b(c)(6), Title 5 U.S.C., as amended. The grant applications and the discussions could disclose confidential trade secrets or commercial property such as patentable material, and personal information concerning individuals associated with the grant applications, the disclosure of which would constitute a clearly unwarranted invasion of personal privacy.

*Name of Committee*: National Center on Minority Health and Health Disparities

Special Emphasis Panel, NCMHD Endowment.

*Date*: June 27-28, 2005.

*Time*: 3 p.m. to 5 p.m.

*Agenda*: To review and evaluate grant applications.

*Place*: Bethesda Marriott, 5151 Pooks Hill Road, Bethesda, MD 20814.

*Contact Person*: Merlyn M. Rodrigues, PhD, MD, Director, Office of Extramural Activities, National Center On Minority Health and Health Disparities, National Institutes of Health, 6707 Democracy Blvd. Suite 800, Bethesda, MD 20894, (301) 402-1366, [rodrigm1@mail.nih.gov](mailto:rodrigm1@mail.nih.gov).

Dated: May 25, 2005.

#### **LaVerne Y. Stringfield,**

*Director, Office of Federal Advisory Committee Policy.*

[FR Doc. 05-11095 Filed 6-2-05; 8:45 am]

**BILLING CODE 4140-01-M**

## **DEPARTMENT OF HEALTH AND HUMAN SERVICES**

### **National Institutes of Health**

#### **National Institute of Diabetes and Digestive and Kidney Disorders; Notice of Closed Meeting**

Pursuant to section 10(d) of the Federal Advisory Committee Act, as amended (5 U.S.C. Appendix 2) notice is hereby given of the following meeting.

The meeting will be closed to the public in accordance with the provisions set forth in sections 552b(c)(4) and 552b(c)(6), Title 5 U.S.C., as amended. The purpose of this meeting is to evaluate requests for preclinical development resources for potential new therapeutics for Type 1 diabetes. The outcome of the evaluation will be a decision whether NIDDK should support the request and make available contract resources for development of the potential therapeutic to improve the treatment or prevent the development of Type 1 diabetes and its complications. The research proposals and the discussions could disclose confidential trade secrets or commercial property such as patentable material, and personal information concerning individuals associated with the proposed research projects, the disclosure of which would constitute a clearly unwarranted invasion of personal privacy.

*Name of Committee*: National Institute of Diabetes and Digestive and Kidney Disorders Special Emphasis Panel; Type 1 Diabetes—Rapid Access to Intervention Development.

*Date*: June 21, 2005.

*Time*: 3 p.m.—4 p.m.

*Agenda*: To evaluate requests for preclinical development resources for