

development through collaborative research opportunities with the inventors.

### A Knockout Mouse for Transcription Factor Nurr1

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HHS Reference No. E-024-1999/0—  
Research Tool  
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Transcriptional factor Nurr1 is an obligatory factor for neurotransmitter dopamine biosynthesis only in ventral midbrain as demonstrated by the Nurr1 genomic locus inactivation using homologous recombination.

From a neurological and clinical perspective, it suggests an entirely new mechanism for dopamine depletion in a region where dopamine is known to be involved in Parkinson's disease. Clinically, our findings indicate that activation of Nurr1 may be therapeutically useful for Parkinson's disease patients; therefore, the mice would be useful in Parkinson's disease research.

Dated: January 3, 2006.

#### 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

#### Prospective Grant of Exclusive License: Anthrax Lethal Factor Is a MAPK Kinase Protease

**AGENCY:** National Institutes of Health, Public Health Service, HHS.

**ACTION:** Notice.

**SUMMARY:** This is notice, in accordance with 35 U.S.C. 209(c)(1) and 37 CFR 404.7(a)(1)(i), that the National Institutes of Health (NIH), Department of Health and Human Services, is contemplating the grant of an exclusive license to practice the inventions embodied in U.S. Patent Nos. 6,485,925 B1, issued November 26, 2002, 6,893,835 B2, issued May 17, 2005, and 6,911,203 B1, issued June 28, 2005, and U.S. Patent App. No. 11/112,137, filed April 22, 2005 and published on September 8, 2005 as U.S. Pat. Pub. No. 2005/019682 A1, all titled "Lethal Factor is a MAPK Kinase Protease" (HHS Ref. Nos. E-066-1998/0-US-06, -07, -08, and -10) to Van Andel

Research Institute, of Grand Rapids, Michigan. The patent rights in these inventions have been assigned to the Government of the United States.

The prospective exclusive license territory will be worldwide. The field of use may be limited to the development and sale of Anthrax lethal factor, a MAPK kinase protease, as a therapeutic agent for the treatment of cancer.

**DATES:** Only license applications which are received by the National Institutes of Health on or before March 13, 2006 will be considered.

**ADDRESSES:** Requests for information, inquiries, comments, and other materials relating to the contemplated co-exclusive license should be directed to: Thomas P. Clouse, Office of Technology Transfer, National Institutes of Health, 6011 Executive Boulevard, Suite 325, Rockville, MD 20852-3804; Telephone: 301-435-4076; Facsimile: 301-402-0220; E-mail: [clouset@mail.nih.gov](mailto:clouset@mail.nih.gov). Copies of the U.S. patent publications can be obtained from <http://www.uspto.gov>.

**SUPPLEMENTARY INFORMATION:** The above-identified patents relates to the discovery that Mitogen Activated Protein Kinase (MAPK) signal transduction pathway is an evolutionarily conserved pathway for effecting gene regulation that controls cell proliferation and differentiation in response to extracellular signals and also plays a crucial role in regulating oocyte meiotic maturation. The above-identified patent discloses in vitro and in vivo methods of screening for modulators, homologues, and mimetics of LF mitogen activated protein kinase (MAPKK) protease activity. Mos (i.e., an oncogene first identified as the transforming determinant of Moloney Murine Sarcoma Virus) is a serine/threonine kinase which phosphorylates and activates MAPK1 kinase which in turn phosphorylates and activates MAPK. The patent also discloses that LF prevents activation of MAPK in oocytes of *Xenopus laevis* and tumor derived NIH3T3 (490) cells expressing an effector domain mutant form of the human V12HaRas oncogene. The tumor derived NIH3T3 cells reverted to a more normal morphology after LF treatment. Therefore, LF directly inhibits the Mos/MAPK pathway. Tumor cells utilize MAPK kinases in a different way than normal cells as in tumor cells there is a constitutive MAPK kinase activity. Additionally, MAPKK1 was found to be a proteolytic substrate for the metalloprotease LF. By analysis of MAPKK2, a consensus sequence for LF activity was found. The disclosure is claimed in the above-identified patent

and other patents in the same patent family.

The prospective exclusive license will be royalty-bearing and will comply with the terms and conditions of 35 U.S.C. 209 and 37 CFR 404.7. The prospective exclusive license may be granted unless within sixty (60) days from the date of this published notice, the NIH receives written evidence and argument that establish that the grant of the license would not be consistent with the requirements of 35 U.S.C. 209 and 37 CFR 404.7.

Applications for a license in the field of use filed in response to this notice will be treated as objections to the grant of the contemplated exclusive license. Comments and objections submitted to this notice will not be made available for public inspection and, to the extent permitted by law, will not be released under the Freedom of Information Act, 5 U.S.C. 552.

Dated: January 3, 2006.

#### Steven M. Ferguson,

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

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## DEPARTMENT OF HEALTH AND HUMAN SERVICES

### Substance Abuse and Mental Health Services Administration

#### Agency Information Collection Activities: Proposed Collection; Comment Request

In compliance with Section 3506(c)(2)(A) of the Paperwork Reduction Act of 1995 concerning opportunity for public comment on proposed collections of information, the Substance Abuse and Mental Health Services Administration will publish periodic summaries of proposed projects. To request more information on the proposed projects or to obtain a copy of the information collection plans, call the SAMHSA Reports Clearance Officer on (240) 276-1243.

Comments are invited on: (a) Whether the proposed collections of information are necessary for the proper performance of the functions of the agency, including whether the information shall have practical utility; (b) the accuracy of the agency's estimate of the burden of the proposed collection of information; (c) ways to enhance the quality, utility, and clarity of the information to be collected; and (d) ways to minimize the burden of the collection of information on