REPORTING REQUIREMENTS—Continued

Regulatory/section requirements	Number of respondents	Responses per respondent	Total annual responses	Hours per response	Total hour burden
57.215(a)(d), Administrative Hearings	0	0	0	0	0
HPSL Subtotal	4,052		67,398		11,637
NSL Program:					
57.305(a)(2), Excess Cash	Burden included under 0915–0044 and 0915–0046				
57.306(a)(2), Student Financial Aid Transcript	2,250	1	2,250	0.25	563
57.310(b)(1)(i), Entrance Interview	347	24	8,328	0.167	1,391
57.310(b)(1)(ii), Exit Interview	[1] 607	4	2,428	0.5	1,214
57.301(b)(1)(iii), Notification of Repayment	[1] 607	6	3,642	0.167	608
57.310(b)(1)(iv), Notification During Deferment	[1] 607	1	607	0.083	50
57.310(b)(1)(vi), Notification of Delinquent Accounts	[1] 607	5	3,035	0.167	507
57.310(b)(1)(x), Credit Bureau Notification	[1] 607	8	4,856	0.6	2,914
57.310(b)(4)(i), Write-off of Uncollectible Loans	20	1.0	20	0.5	10
57.311(a), Disability Cancellation	7	1.0	7	0.8	6
57.312(a)(3), Evidence of Educational Loans	Inactive Provision				
57.315(a)(1), Reports	Burden included under 0915–044				
57.315(a)(1)(ii), Administrative Hearings	0	0	0	0	0
57.316(a)(d), Administrative Hearings	0	0	0	0	0
NSL Subtotal	2,857		25,173		7,263

[1] Includes active and closing schools.

Written comments and recommendations concerning the proposed information collection should be sent within 30 days of this notice to: John Morrall, Human Resources and Housing Branch, Office of Management and Budget, New Executive Office Building, Room 10235, Washington, DC 20503.

Dated: June 11, 2002.

Jane M. Harrison,

Director, Division of Policy Review and Coordination.

[FR Doc. 02–15161 Filed 6–14–02; 8:45 am]

BILLING CODE 4165–15–P

DEPARTMENT OF HEALTH AND HUMAN SERVICES

National Institutes of Health

Prospective Grant of Exclusive License: Activity Dependent Neurotrophic Factor (ADNF) III

AGENCY: National Institutes of Health, Public Health Services, 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, Department of Health and Human Services, is contemplating the grant of an exclusive license to practice the inventions embodied in U.S. provisional patent application 60/037,404 filed February 7, 1997 and entitled ''Activity Dependent Neurotrophic Factor (ANDF) III,'' PCT application PCT/US98/02485 filed on February 6, 1998 and entitled ''Activity Dependent Neurotrophic Factor (ANDF) III,'' U.S. Continuation-in-Part application 09/187,330 filed on November 6, 1999 and entitled ''Activity Dependent Neurotrophic Factor III (ANDF III),'' PCT application PCT/US99/26213 filed February 4, 1999 and entitled ''Activity Dependent Neurotrophic Factor (ANDF) III,'' U.S. provisional patent application 60/208,944 filed on May 31, 2000 and entitled ''Use of Activity-Dependent Neurotrophic Factor-Derived Polypeptides for Enhancing Learning and Memory,'' U.S. provisional patent application 60/267,805 filed on February 8, 2001 and entitled ''Prenatal Treatment with ADNF Polypeptides to Improve Learning and Memory,'' PCT application PCT/US01/17758 filed on May 31, 2001 and entitled ''Use of Activity Dependent Neurotrophic Factor Derived Polypeptide for Enhancing Learning and Memory: Pre- and Post-Natal Administration.'' U.S. patent application 09/267,511 filed on March 12, 1999, PCT application PCT/US00/06364 filed on March 10, 2000 and entitled ''Prevention of Fetal Alcohol Syndrome and Neuronal Cell Death with ADNF Polypeptides,'' U.S. provisional patent application 60/149,956 filed on August 18, 1999, and PCT application PCT/US00/22861 filed on August 17, 2000, and entitled ''Orally Active Peptides that Prevent Cell Damage and Death,'' to Allon Therapeutics, of San Diego California. The patent rights in these inventions have been assigned to the United States of America.

The prospective exclusive license territory will be worldwide. The field of use will be all neurodegenerative diseases, but may be limited to Alzheimer's disease and stroke.

DATES: Only written comments and/or license applications which are received by the National Institutes of Health on or before August 16, 2002, will be considered.

ADDRESSES: Requests for copies of the patent(s)/patent application(s), inquiries, comments and other materials relating to the contemplated exclusive license should be directed to: Jonathan V. Dixon, Technology Licensing Specialist, Office of Technology Transfer, National Institutes of Health, 6011 Executive Boulevard, Suite 325, Rockville, MD 20852–3804; Telephone: 301.496.7056, x270; Facsimile 301.402.0220; email *dixonj@od.nih.gov.*

SUPPLEMENTARY INFORMATION: The above-referenced patent(s)/patent application(s) relate to Activity

Dependent Neurotrophic factor III (ADNF III) and a specific eight amino acid peptide denoted as NAP (NAPVSIQ) derived from the cloned ADNF III. NAP has been discovered to have potent neuroprotective properties in vitro and in vivo. NAP has been shown to significantly reduce the number of apoptotic cells and to protect neurons against numerous toxins and cellular stresses including in vitro exictotoxicity, oxidative stress, and glucose deprivation. NAP also exhibits neuroprotective activity in a variety of animal models including a learning deficient apolipoprotein E knockout mice (a model related to Alzheimer's disease), mouse paradigms of traumatic head injury (associated with an inflammatory response) and fetal alcohol syndrome (oxidative stress), and a rat model of cholinotoxicity.

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: May 28, 2002.

Jack Spiegel,

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

[FR Doc. 02–15147 Filed 6–14–02; 8:45 am]

BILLING CODE 4140–01–P

DEPARTMENT OF HEALTH AND HUMAN SERVICES

National Institutes of Health

Prospective Grant of Exclusive License: Cytotoxic Treatment of Cancer Cells That Overexpress Matrix Metalloproteinases, Plasminogen Activators and/or Plasminogen Activator Receptors

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

ACTION: Notice.

SUMMARY: This notice, in accordance with 35 U.S.C. 209(c)(1) and 37 CFR Part 404.7(a)(1)(i), that the National Institutes of Health, Department of Health and Human Services is contemplating the grant of an exclusive patent license to practice the inventions embodied in U.S. Patent Application, 60/155,961 (refiled): ''Mutated anthrax toxin protective antigen proteins that specifically target cells containing high amounts of cell-surface metalloproteinase or plasminogen activator receptors' (DHHS Ref. E–293–99/0); PCT Patent Application, PCT/US00/26192 [WO01/21656] (refiled): ''Mutated anthrax toxin protective antigen proteins that specifically target cells containing high amounts of cell-surface metalloproteinase or plasminogen activator receptors' (DHHS Ref. E–293–99/1); U.S. Patent Application, S/N 10/088,952: ''Mutated anthrax toxin protective antigen proteins that specifically target cells containing high amounts of cell-surface metalloproteinase or plasminogen activator receptors' (DHHS Ref. E–293–99/2); U.S. Patent 5,591,631, S/N 08/021,601, which issued on January 7, 1997 (DHHS Ref. E–064–93/0), entitled, ''Anthrax toxin fusion proteins, nucleic acid encoding same''; U.S. Patent 5,677,274, S/N 08/082,849, which issued on October 14, 1997 (DHHS Ref. E–064–93/1), entitled, ''Anthrax toxin fusion proteins and related methods''; and any related foreign filed national stage applications claiming priority to such cases to OncoTac Pharmaceuticals which is located in Medicon Valley, Denmark. The patent rights in these inventions have been assigned to the United States of America.

The prospective exclusive license territory will be worldwide and the field of use may be limited to human therapeutics for the treatment of cancer by a mechanism involving cancer-associated enzymes and/or receptors.

DATES: Only written comments and/or license applications that are received by the National Institutes of Health on or before August 16, 2002, will be considered.

ADDRESSES: Requests for copies of the patent, inquiries, comments and other materials relating to the contemplated exclusive license should be directed to: Richard U. Rodriguez, M.B.A., Technology Licensing Specialist, Office of Technology Transfer, National Institutes of Health, 6011 Executive Boulevard, Suite 325, Rockville, MD. 20852–3804. Telephone: (301) 496–7056, X287; Facsimile: (301) 402–0220; and E-mail: *rodrigur@od.nih.gov.*

SUPPLEMENTARY INFORMATION: The primary technology relates to an immunotoxin treatment system that is targeted to cancer cells via an anthrax-based pathway. Native anthrax toxin is a three-component toxin consisting of protective antigen (PrAg), lethal factor (LF), and edema factor (EF). PrAg binds to the recently identified cell surface anthrax receptor and the subsequent steps in toxin action is dependent on cleavage of PrAg at the sequence, ^{164}RKKR^{167}, by a cell-surface, furin-like protease. The carboxyl-terminal 63-kDa fragment (PrAg63) remains bound to receptor, forms a heptamer, and binds and internalizes LF and EF. LF kills animals and lyses mouse macrophages due to proteolytic cleavage of MAP kinase kinases. EF damages cells due to its intracellular adenylate cyclase activity. A potent PrAg dependent cytotoxin, FP59, created by fusing LF amino acids 1–254 to the ADP-ribosylation domain of *Pseudomonas* exotoxin A can kill any cell having receptors for PrAg and the ability to activate PrAg by cleavage at amino acids 164–167.

Activation of the native PrAg is dependent on a cell surface located furin-like proteolytic activity. In the current technology, the furin-site has been manipulated to generate mutant PrAg proteins that are specific for matrix metalloproteinases (MMPs) or the urokinase plasminogen activator (uPA). A combination of the mutated toxins PrAg and FP59 has been shown to be an effective cytotoxic agent that is strictly dependent on cell surface localized MMP and/or uPA-activity.

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 part 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 part 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.