(reduced levels of melanin concentrating hypothalmic mRNA) inconsistent with fasted animals. These data point to the existence of a novel cholinergic pathway involving M3 cholinergic receptor mediated stimulation of food intake. This technology strongly suggests that agents which can specifically and selectively act as antagonists of the M3 subtype receptors may be useful in the treatment of obesity.

Methods for Preventing Strokes by Inducing Tolerance to E-selectin

John M. Hallenbeck, et al. (NINDS), Serial No. 60/206,693 filed 24 May 2000, Licensing Contact: Norbert Pontzer; 301/496–7736 ext. 284; e-mail: pontzern@od.nih.gov

This invention provides methods of treating or preventing brain damage in stroke through administration of E-selectin, an inducible adhesion molecule on endothelial cells. The expression of E-selectin is induced on human endothelium in response to activation by cytokines IL–1 and TNF. E-selectin mediates the adhesion of various leukocytes, including neutrophils, monocytes, eosinophils, natural killer cells, and a subset of T cells to activated endothelium. Activation of vascular endothelial cells by proinflammatory cytokines is believed to be involved in conversion of the luminal surface of endothelium from anticoagulant and anti-inflammatory to procoagulant and pro-inflammatory leading to thrombosis. Segmental vascular activation and thrombosis are involved in the development of strokes.

Recently, a new method and pharmaceutical formulation have been found that induce tolerance mucosally, such as by intranasal administration. The potential of mucosally administered antigens to inhibit immune responses in an antigen specific fashion has encouraged attempts to apply these routes to counteract immune dysfunctions such as allergies and in particular, autoimmune disease. Intranasal administration of E-selectin induces tolerance to E-selectin and leads to immune-deviation of a subset of lymphocytes such that they can suppress activation of vessel segments that are beginning to express E-selectin. Thus the ability of intranasal E-selectin treatment to decrease stroke lesions and delay the onset of stroke in stroke-prone spontaneously hypertensive rats suggests that the initial vessel activation and damage in stroke may be immunologically mediated. Production of immunosuppression via antigen-specific modulation of the immune response (mucosal tolerance) should have no systemic immunosuppressive effects.

Dated: March 23, 2001.

Jack Spiegel,

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

[FR Doc. 01–8086 Filed 4–2–01; 8:45 am]

BILLING CODE 4140–01–P

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.

Analogs of Thalidomide as Potential Angiogenesis Inhibitors

William Figg et. al. (NCI)
DHHS Reference No. E–282–00/0; filed 27 Feb 2001
Licensing Contact: Matthew Kiser; 301/496–7735 ext. 224; e-mail: kiserm@od.nih.gov

The present invention relates to anti-angiogenesis compositions and methods of using the same. In particular, thalidomide analogs that actively inhibit angiogenesis in humans and animals are claimed. The present methods provide for the inhibition of unwanted angiogenesis through the administration of a composition comprising an effective amount of an ''active'' thalidomide analog.

Angiogenesis is the formation of new blood vessels from pre-existing vessels, and it is a prominent feature in solid tumor formation and metastasis. For example, angiogenesis seems to play an important role in tumors such as prostate cancer, breast cancer, CNS glioma, and renal cancer, to name a few. Prevention of angiogenesis could halt the growth of these types of tumors and help prevent the resultant damage due to the presence of these tumors.

Recent studies have promoted thalidomide as a potential inhibitor of angiogenesis. The anti-angiogenic activity initially attributed to thalidomide is actually the resulting effects of compounds that are only present following metabolic activation, i.e. ''active'' thalidomide metabolites. Accordingly, there is a need for the isolation, identification and characterization of these thalidomide metabolites that exhibit superior anti-angiogenic properties. Furthermore, there is a need for purified thalidomide analogs that can mimic the effects of these metabolites.

A number of thalidomide metabolites having superior anti-angiogenic properties have now been isolated and identified. In addition, thalidomide analogs that mimic the effects of the ''active'' thalidomide (metabolites and variations of such thalidomide analogs) have been synthesized and evaluated. Such thalidomide analog compounds show enhanced potency in the inhibition of angiogenesis without the undesirable effects of administration of thalidomide.

Detection and Quantification of Cripto-1 in Human Milk Using ELISA

Caterina Bianco, David S. Salomon (NCI)
DHHS Reference No. E–290–00/0 filed 26 Jan 2001
Licensing Contact: Matthew Kiser; 301/496–7735 ext. 224; e-mail: kiserm@od.nih.gov

Cripto-1 (CR1) is a member of the epidermal growth factor (EGF)-related families of peptides and is involved in the development and progression of various human carcinomas. In particular, CR1 overexpression has been detected in 50–90% of carcinomas of the colon, pancreas, stomach, gallbladder, breast, lung, endometrium and cervix. Current methodologies of cancer detection, e.g. immunohistochemistry, can be time consuming, inconvenient and oftentimes, inaccurate, and therefore, a need exists for more efficient, reliable and less time consuming methods of detection. The invention relates to such a method of detection. The inventors

disclose methods for the detection and quantification of CR1 in human milk, using an ELISA-based protocol. Thus, this test could be used to more effectively detect and perhaps stage cancers. Additionally, should particular tumor cells, e.g. breast tumor cells, express a sufficiently high level of CR1, it may be possible to use the disclosed assay to detect and measure CR1 in human serum and/or plasma. Claims to these routes of detection are also present in the patent application. As such, a novel, efficient and useful in vitro diagnostic and prognostic test is now available to suitable commercial partners.

Dated: March 23, 2001.

Jack Spiegel,

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

[FR Doc. 01–8087 Filed 4–2–01; 8:45 am]

BILLING CODE 4140–01–P

DEPARTMENT OF HEALTH AND HUMAN SERVICES

National Institutes of Health

Prospective Grant of Exclusive License: The use of cyanovirin-N in a Topical Microbicide To Prevent the Transmission of HIV and Other Sexually Transmitted Diseases

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

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 a exclusive license worldwide to practice the invention embodied in the patents and patent applications referenced below to Biosyn, Inc., of Philadelphia, PA. The patent rights in these inventions have been assigned to the United States of America.

(1) U.S. Patent No. 5,821,081, issued Oct. 13, 1998, entitled ''Nucleic Acids Encoding Antiviral Proteins and Peptides, Vectors and Host Cells Comprising Same, and Methods of Producing the Antiviral Proteins and Peptides'' (PHS Reference No. E–117–95/1)

(2) U.S. Patent No. 5,843,882, issued Dec. 01, 1998, entitled ''Antiviral Proteins and Peptides, DNA, DNA-coding Sequences Therefor, and Uses Thereof'' (E–117–95/0)

(3) U.S. Patent No. 5,998,587, issued Dec. 7, 1999, entitled ''Anti-Cyanovirin Antibody'' (E–117–95/6)

(4) U.S. Patent No. 6,015,876, issued Jan. 18, 2000, entitled ''Method of Using Cyanovirins'' (E–117–95/3)

(5) U.S. Patent Application No. 09/267,447, filed Mar. 12, 1999, pending, entitled ''Cyanovirin Conjugates and Matrix-Anchored Cyanovirin and Related Composition and Methods of Use'' (E–074–99/0)

(6) U.S. Patent Application No. 09/416,434, pending, entitled ''Cyanovirin Conjugates and Matrix-Anchored Cyanovirin and Related Composition and Methods of Use'' (E–074–99/1)

(7) U.S. Patent Application No. 09/427,873, filed 10/27/99, pending, entitled ''Methods of Using Cyanovirins to Inhibit Viral Infection'' (E–074–99/3)

(8) U.S. Patent Application No. 09/417,797, filed 10/27/99, pending, entitled ''Methods of Using Cyanovirins Topically to Inhibit Viral Infection'' (E–074–99/4)

(9) PHS Reference Number E–074–99/7, filed 3/22/01, entitled ''Glycosylation-Resistant Cyanovirins and Related Conjugates, Compositions, Nucleic Acids, Vectors, Host Cells, Methods of Production and Methods of Using Nonglycosylated Cyanovirins''

DATES: Only written comments and/or application for a license which are received by the NIH Office of Technology Transfer on or before July 2, 2001 will be considered.

ADDRESSES: Requests for a copy of the patent applications, inquiries, comments and other materials relating to the contemplated license should be directed to: Sally Hu, Ph.D., Office of Technology Transfer, National Institutes of Health, 6011 Executive Boulevard, Suite 325, Rockville, MD 20852–3804; Telephone: (301) 496–7056, ext. 265; Facsimile: (301) 402–0220; e-mail: hus@od.nih.gov.

SUPPLEMENTARY INFORMATION: The patents and patent applications describe a novel protein, cyanovirin-N, discovered by Dr. Michael R. Boyd and colleagues at the National Cancer Institute. Cyanovirin-N was isolated from a blue-green algae and has been demonstrated to bind avidly to and inactivate the human immunodeficiency virus (HIV).

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 90 days from the date of this published Notice, NIH receives written evidence and argument that establishes that the grant of the license would not be consistent with the requirements of 35 U.S.C. 209 and 37 CFR 404.7.

The field of use may be limited to compositions, devices and methods for the prevention of infection by HIV and other sexually transmitted pathogens, topically, but not systemically, utilizing cyanovirin-N, anti-HIV mutants of cyanovirin-N, and anti-HIV fragments of both, but excluding pegylated cyanovirin-N, pegylated anti-HIV mutants of cyanovirin-N and pegylated anti-HIV fragments of both.

Properly filed competing applications for a license filed in response to this notice will be treated as objections to the contemplated license. Comments and objections submitted in response 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: March 26, 2001.

Jack Spiegel,

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

[FR Doc. 01–8091 Filed 4–2–01; 8:45 am]

BILLING CODE 4140–01–P

DEPARTMENT OF HEALTH AND HUMAN SERVICES

National Institutes of Health

Prospective Grant of Exclusive License: Identification of TRP–2 as a New Human Tumor Antigen Recognized by Cytotoxic T Lymphocytes

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

ACTION: Notice.

SUMMARY: This 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. Patent Applications S/N 08/725,736, filed on October 4, 1996, and now U.S. Patent 5,831,016 which issued on November 3, 1998; S/N 09/161,877 (DIV of 08/725,736), filed on September 28, 1998, and now U.S. Patent 6,132,980 which issued on October 17, 2000; S/N 09/162,368 (DIV of 08/725,736), filed on September 28, 1998, and now U.S. Patent 6,083,703 which issued on July 4, 2000; and S/N 09/651,210 (DIV of 08/725,736), filed on August 30, 2000, all entitled ''Identification of TRP–2 as a New