applications of the technology outlined in the agreement.

7. Incorporating the immunotoxin into formulations in order to increase the therapeutic efficacy and decrease immunogenicity.

8. Providing immunotoxin for laboratory and animal studies.

9. Publishing research results.

Selection criteria for choosing the CRADA Collaborator may include, but not be limited to:

1. The ability to collaborate with NCI on further research and development of this technology. This ability can be demonstrated through experience and expertise in this or related areas of technology indicating the ability to contribute intellectually to ongoing research and development.

2. The demonstration of adequate resources to perform the research and development of this technology (e.g. facilities, personnel and expertise) and accomplish objectives according to an appropriate timetable to be outlined in the CRADA Collaborator's proposal.

3. The willingness to commit best effort and demonstrated resources to the research and development of this technology, as outlined in the CRADA Collaborator's proposal.

4. The demonstration of expertise in the commercial development and production of products related to this area of technology.

5. The level of financial support the CRADA Collaborator will provide for CRADA-related Government activities.

6. The demonstration of expertise pertinent to the development of models to evaluate and improve the efficacy of the RFB4 (dsFv)–PE38 immunotoxin for the treatment of lymphomas and leukemias.

7. The demonstration of expertise in the formulation of drugs.

8. The willingness to cooperate with the NCI in the timely publication of research results.

9. The agreement to be bound by the appropriate DHHS regulations relating to human subjects, and all PHS policies relating to the use and care of laboratory animals.

10. The willingness to accept the legal provisions and language of the CRADA with only minor modifications, if any. These provisions govern the distribution of patent rights to CRADA inventions. Generally, the rights of ownership are retained by the organization that is the employer of the inventor, with (1) the grant of a license for research and other Government purposes to the Government when the CRADA Collaborator's employee is the sole inventor, or (2) the grant of an option to elect an exclusive or nonexclusive license to the CRADA Collaborator when the Government employee is the sole inventor.

Dated: November 5, 1998.

Jack Spiegel,

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

Dated: November 16, 1998.

Kathleen Sybert,

Acting Director, Technology Development and Commercialization Branch, National Cancer Institute, National Institutes of Health.

[FR Doc. 98–31733 Filed 11–27–98; 8:45 am]

BILLING CODE 4140–01–M

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 contacting Dennis H. Penn, Pharm.D., Technology Licensing Specialist, Office of Technology Transfer, National Institutes of Health, 6011 Executive Boulevard, Suite 325, Rockville, Maryland 20852–3804; telephone: 301/496–7056 ext. 211; fax: 301/402–0220. A signed Confidential Disclosure Agreement will be required to receive copies of the patent applications.

A Mitochondrial-Specific ATP-Binding Transporter Gene (ABC7) Is An Iron Transporter In An Interhited Ataxia-Anemia Syndrome

MC Dean, R Allikmets, AA Hutchinson (NCI)

DHHS Reference No. E–181–98/0 filed Oct 23, 1998

The gene responsible for the rare genetic disease, X-linked siderblastic anemia and ataxia (XLSA/A) has been identified and linked to a mutation of the ATP-Binding transporter gene (ABC7). Two sequence changes which correspond to amino acid changes at positions 50 and 396 were detected. This gene may prove useful as a diagnostic for XLSA/A carriers or as a means to rule out XLSA/A from other siderblastic anemias. ABC7, an iron transporter, may prove to be a valuable tool for studying the function and regulation of muscle cells and the loss of motor function associated with many diseases with faculty iron metabolism, i.e. neuromuscular disease, cardiac disorders and neurological disorders.

Compsitions And Uses of FIG-alpha Gene

J Dean, L Liang, S soyal (NIDDK)

Serial No. 60/069,037 filed Dec. 12, 1997

This application related to an isolated and purified polynucleotide encoding an isolated and purified polypeptide associated with the expression of zona pellucida genes. The mouse zona pellucida is composed of three glycoproteins, ZP1, ZP2 and ZP3, encoded by single-copy genes whose expression is temporarlly and spatially restricted to oocytes. All three proteins are required for the formation of the extracellular zona matrix and female mice with a single disrupted zona gene lack a zona and are infertile. An E-box (CANNTG), located approximately 200 bp upstream of the transcription start site of the ZP1, ZP2 and ZP3, forms a protein-DNA complex present in oocytes and, to a much lesser extent, in testes. The integrity of this E-box in ZP2 and ZP3 promoters is required for expression of luciferase reporter genes microinjected into growing oocytes. The presence of the ubiquitous transcription factor E12 in the complex was used to identify a novel basic helix-loop-helix protein FIGα (Factor In the Germline alpha) whose expression was limited to oocytes within the ovary.)

This invention relates to the molecular characterization of FIGα, a novel germ cell specific bHLB transcription factor that binds as a heterodimer with E12 to the E-box in the promoter region of all three mouse zona pellucida genes and has the ability to transactivate reporter gene constructs in vitro. FIGα is critical for folliculogenesis and has a role in the coordinate, oocyte-specific expression of the three zona pellucida genes, the products of which for an extracellular matrix required for fertilization and early development. This invention also relates to monoclonal and polyclonal antibodies, which recognize the FIGα polypeptide.

Ureido Derivatives Of Poly-4-Amino-2-Carboxy-1-Methyl Pyrrole Compounds For Treatment Of inflammation

OM Zac Howard (SAIC), JJ Oppenheim (NCI), WJ Murphy (SAIC), EA Sausville (NCI)

Serial No. 60/067,526 filed Dec 4, 1997

Inflammatory reactions arising from a variety of medical conditions may have serious medical consequences when poorly controlled. Such inflammatory reactions contribute to a variety of disease states such as arthritis, asthma, non-bacterial medicated respiratory distress syndrome, reperfusion injury, and blunt force trauma. Accordingly, there is a need for new methods of diminished inflammation, especially acute inflammation.

This invention describes a method of inhibiting inflammation, particularly non-TNF dependent inflammation, by administering pharmacologically active ureido derivatives of distamycin. Since TNF is only one of many inducers of chemokines, this invention provides a more inclusive method for treatment of many inflammatory conditions, including conditions in which TNF does not play a substantial deleterious role in the pathology of the condition.

Therapeutic Chemokine Antagonists

JJ Oppenheim, JM Wang, OY Chertov, LO Arthur, F Ruscetti (NCI)

DHHS Reference No. E–170–96/0 filed Sep 06, 1996; PVT/US97/15594 filed Sep 05, 1997

This invention relates to a new class of chemoattractant antagonists, which are therapeutic candidates for treating disease conditions involving recruitment of inflammatory cells. These chemoattractant antagonists are comprised of a group consisting of gp120, gp41, domains and variants of gp41 and gp120.

Chemoattractants include the subgroup of chemokines and are known to mediate chemotaxis and other pro-inflammatory phenomena. The chemoattractants are generally short peptides. The family of chemokines is subdivided into distinct subfamilies, C–X–C and C–C, based on the arrangements of the first two cysteines of the primary amino acid sequence.

Members of the chemokine subfamily have remarkable similarities in their structural organization and biochemical properties. These homologies are consistent with the similarities observed in their biological effects, both in vitro and in vivo. These properties have prompted speculation that chemokines are mediators in autoimmune and allergic disorders.

Dated: November 16, 1998.

Jack Spiegel,

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

[FR Doc. 98–31730 Filed 11–27–98; 8:45 am]

BILLING CODE 4140–01–M

DEPARTMENT OF HEALTH AND HUMAN SERVICES

National Institutes of Health

Government-Owned Inventions; Availability for Licensing: Drug and Method for the Therapeutic Treatment of Primary Brain Tumors (Such as Intracranial Human Glioma, Astrocytomas, Medulloblastomas and Metastatic Tumors to the Central Nervous System)

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

ACTION: Notice.

SUMMARY: The National Institutes of Health (NIH) is seeking Licensees to further develop, evaluate, and commercialize a Transforming Growth Factor-alpha-Pseudomonas Exotoxin fusion protein, known a TGF-alpha-PE38, for the therapeutic treatment of refractory brain tumors such as intracranial human glioma, astrocytomas, medulloblastomas and metastatic tumors to the central nervous system (''SNS'').

The invention claimed in USPN 4,892,827, Entitled: ''Recombinant Pseudomonas Exotoxins: Construction of an Active Immunotoxin with Low Side Effects,'' is available for licensing on an exclusive or non-exclusive basis (in accordance with 35 USC 207 and 37 CFR part 404) with the Field of Use limited to the therapy of primary brain tumors, metastatic carcinomas, and leptomeningeal carcinomatosis.

ADDRESSES: Licensing information and copies of the U.S. patent referenced above may be obtained by contacting J.R. Dixon, Ph.D., Technology Licensing Specialist, Office of Technology Transfer, National Institutes of Health, 6011 Executive Boulevard, Suite 325, Rockville, Maryland 20852–3804; telephone: 301/496–7056 ext. 206; fax: 301/402–0220; e-mail: ''DixonJ@od.nih.gov''. Respondees interested in licensing the invention will be required to submit an ''Application for License to Public Health Service Inventions.''

SUPPLEMENTARY INFORMATION: Epidermal growth factor receptor (''EGFR'') is amplified or over expressed in many malignant gliomas, other primary brain tumors, and carcinomas of epithelial origin (e.g., breast, lung, etc.) but is low or undetectable in normal brain tissue. TGF-alpha-PE38 represents a growing class of recombinant toxins designed for use in targeted cancer therapy. These genetically engineered chimeric proteins consist of a targeting moiety and a cytotoxic moiety. While TGF-alpha-PE38 is extremely toxic to tumor cells that have a relatively high expression of EGFR, it is also active against primary human brain tumor cells which are known to have moderate to high EGFR expression. Direct delivery of TGF-alpha-PE38 into brain tumors by intratumoral implanted catheters or controlled-release biodegradable polymers or intrathecal administration into the cerebrospinal fluid of patients with leptomeningeal carcinomatosis, may represent clinically useful applications of recombinant toxin therapy in tumors with high EGFR expression.

Anaplastic astrocytoma and glioblastoma, the most common primary brain tumors in adults, respond poorly to all current therapies: Median survival for patients with these tumors ranges from 19 to 57 weeks. Local tumor recurrence also constitutes a significant problem in medulloblastoma, the most common childhood brain tumor. Despite 5-year survivals for medulloblastoma exceeding 80% in some studies, nearly half of these patients will eventually die from progressive tumor. Treatment failure in patients with brain tumors is a multifactorial process involving the intrinsic resistance of these tumors to radiation therapy and chemotherapy, the development of acquired treatment resistance, and limitations of drug delivery due to blood-brain barrier restrictions. Local recurrence of brain tumors represents the most common pattern of treatment failure. Accordingly, the identification of new therapeutic agents that have high intrinsic activity against brain tumors and are appropriate for local therapy remains a major goal of the NIH.

Dated: November 16, 1998.

Jack Spiegel,

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

[FR Doc. 98–31731 Filed 11–27–98; 8:45 am]

BILLING CODE 4140–01–M