(HGFr) pathway. In particular, the invention described in this application is a murine monoclonal antibody, designated D1, which specifically binds to an epitope in the extracellular domain of human HGFr/met. The monoclonal antibody can be used, for example, to visualize HGFr/met expression in paraffin-embedded tumor samples and in drug screening assays (competitive binding assays) for antagonists/agonists of HGFr/met.

Determination of AM Binding Proteins and the Association of Adrenomedullin (AM) Therewith

F Cuttitta (NCI), A Martinez (NCI), R Pio (NCI), TH Elasser (USDA–ARS), Serial No. 60/153,397 filed 10 Sep 99

This application relates to isolation and identification of a polypeptide which binds to the hormone adrenomedullin designated adrenomedullin binding protein 1 (AMBP1). Adrenomedullin (AM), a peptide hormone, has been implicated in a variety of physiological functions including the regulation of insulin production, anti-microbial activity, mitogenesis and angiogenesis. The activities of AM are believed to be mediated by a variety of binding proteins in a manner similar to the way in which Insulin-like Growth Factor (IGF) is regulated. AMBP1 has been purified to homogeneity and its amino acid sequence determined.

The application is directed to methods of measuring AM levels in plasma based on the finding that AMBP1 binds in a specific and reversible competitive fashion with AM and methods of treating AM related disease by administering AMBP1. Other aspects of the invention are complexes of AM with AMBP1 and antibodies which specifically bind to an epitope by the complex of AM with AMBP1 as well as assays for detecting the complex of AM with AMBP1.

This work has been published in part in Elsasser TH, et al. Endocrinology 140(10):4908–11 (Oct. 1999).

In addition to being available for licensing the NIH is willing to consider interest from companies who are interested in pursuing commercialization opportunities through a Cooperative Research and Development Agreement (CRADA).

AAV5 Vector and Uses Thereof

JA Chiorini, RM Kotin (NHLBI)
Serial No. PCT/US99/11958 filed 28 May 1999 based on USSN 60/087,029 filed 28 May 1998

The invention described and claimed in this patent application provides for novel vectors and viral particles which comprise adeno-associated virus serotype 5 (AAV5). AAV5 is genetically distinct from others AAVs with respect to its capsid proteins, VP1, VP2, and VP3, which contributes to different tissue tropisms for AAV5. The ITR and Rep proteins of AAV5 are also distinct which results in a biochemically unique mechanism of replication compared to the other AAVs. This difference in replication activity contributes to the fact that AAV5 is only able to replicate and package AAV5 ITR containing DNA in contrast to AAV2 which is able to replicate and package other AAV serotypes. Vectors produced using AAV5 proteins may be useful in gene therapy.

AAV5 offers several advantages which make it attractive for use in gene therapy: (1) increased production (10–50 fold greater than AAV2); (2) its distinct replication mechanism when compared to AAV2; (3) its Rep protein and ITR regions which do not complement other serotypes; (4) it appears to utilize different cell surface attachment molecules than those of AAV type 2; and (5) improved efficiency of transduction of certain cell types including airway epithelial, striated muscle, endothelial, and neuronal cells when compared to AAV type 2.

This work has been published, in part, in J. Virol. 73(5): 4293–98 (May 1999) and J. Virol. 73(2): 1309–19 (Feb. 1999).

Prevention of Fetal Alcohol Syndrome and Neuronal Cell Death with ADNF Polypeptides

DE Brenneman (NICHD), CY Spong (NICHD), I Gozes (TAU), M Bassan (TAU), R Zamostiano (TAU)
Serial No. 09/267,511 filed 12 Mar 1999

This patent application describes an extension of prior work related to peptides derived from proteins known as ADNF and ADNF III/ADNP. These peptides are known as SAL (ADNF-derived) and NAP (ADNP-derived). SAL and NAP (L-isomers) have previously been demonstrated, in in vitro work, to be able to prevent neuronal cell death and to protect against the toxic activities of a cholinotoxin suggesting that they are useful as therapeutics for neurodegenerative diseases. The new work presented in this EIR demonstrates that NAP and SAL (L-isomers), alone or in combination, prevent damage to neurons due to oxidative stress. In particular, the new work shows that NAP and SAL (L-isomers) alone or together are effective in preventing damage due to oxidative stress in a model for fetal alcohol syndrome. Thus, NAP and SAL (L-isomers), alone or together may be useful therapeutically to treat fetal alcohol syndrome.

In addition, a number of other patent applications and patents related to this technology have been filed by PHS and are available for licensing. These include: USP 5,767,240 (PCT/US92/03109); 08/324,297 (PCT/US95/12929); 60/037,404 (PCT/US98/07485); 09/187,330 (PCT/US99/26213) 60/149,956; and 09/364,609.

Dated: April 18, 2000.

Jack Spiegel,

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

[FR Doc. 00–10180 Filed 4–21–00; 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 contacting Richard U. Rodriguez, M.B.A., at the Office of Technology Transfer, National Institutes of Health, 6011 Executive Boulevard, Suite 325, Rockville, Maryland 20852–3804; telephone: 301/496–7056 ext. 287; fax: 301/402–0220; e-mail: rr154z@nih.gov. A signed Confidential Disclosure Agreement will be required to receive copies of the patent applications.

Identification of a Novel Amplified Gene, MB1, at 17q23

Anne H Kallioniemi, Maarit T Barlund, Outi M Monni, Juha T Kononen, Olli P Kallioniemi (NHGRI)
DHHS Reference No. E–038–00/0 filed 13 Dec 1999

DNA amplification at 17q23 is one of the most common genetic alterations in breast cancer. Genes affected by this amplification may have a critical role in

breast cancer development and progression and may provide targets for anti-cancer therapy. The inventors have identified a novel gene from the amplified region, named MB1, which has no homology to any known genes. MB1 is amplified in about 9% of primary breast tumors and is overexpressed in breast cancer cell lines with amplification. MB1 may define a critically important breast cancer gene which could have significance for development of improved diagnostics against breast cancer.

The Use of Recombinant Cholera Toxin-B for the Treatment of Inflammatory Bowel Disease

Warren Strober, Monica Boirivant, Ivan J Fuss, Brian L Kelsall (NIAID)
Serial No. 60/165,111 filed 12 Nov 1999

The present invention provides methods of treating or preventing inflammation in a subject, comprising administering to the subject an effective amount of cholera toxin subunit B (CT–B). In particular, the present invention provides methods of decreasing the activity of interferon-gamma in a subject, decreasing the activity of IL–12 in a subject, and treating or preventing a Th1 T-cell mediated autoimmune disorder.

Dated: April 18, 2000.

Jack Spiegel,

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

[FR Doc. 00–10181 Filed 4–21–00; 8:45 am]

BILLING CODE 4140–01–P

DEPARTMENT OF HEALTH AND HUMAN SERVICES

National Institutes of Health

Government-Owned Invention Availability for Licensing: ''Therapeutic and Diagnostic Antibodies and Immunotoxins to a Mutant Form of Epidermal Growth Factor Receptor, Designated ''EGFRVIII'', Which is Highly Expressed in Glioblastomas, Carcinomas of the Breast and Ovary, and Non-Small Cell Lung Carcinomas''

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

ACTION: Notice.

SUMMARY: The invention listed below is owned by an agency of the U.S. Government and is 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.

ADDRESSES: Licensing information and a copy of the U.S. patent application referenced below may be obtained by contacting J. R. Dixon, Ph.D., at the 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: jd212g@NIH.GOV). A signed Confidential Disclosure Agreement is required to receive a copy of any patent application.

SUPPLEMENTARY INFORMATION:

Invention Title: ''Anti-EGFRvIII ScFvs with Improved Cytotoxicity and Yield, Immunotoxins Based Thereon, and Methods of Use Thereof''.

Inventors: Drs. Ira H. Pastan (NCI), Richard Beers (NCI), Partha S. Chowdury (NCI) and Darell Bigner (EM).

USPA SN: [= DHHS Ref. No. E–009–00/0]—Filed with the U.S.P.T.O. on January 25, 2000.

Abstract

A mutant form of the epidermal growth factor receptor, designated ''EGFRvIII,'' is highly expressed in some 50–60% of glioblastomas and has also been shown to be present in some 70–80% of carcinomas of the breast and ovary, and about 16% of non-small cell lung carcinomas. The mutation consists of an in-frame deletion of exons 2–7 near the amino-terminus of the extracellular domain which results in the expression of an EGFR mRNA with an 801 base deletion. The mutant protein contains a new glycine codon at the splice junction. The receptor has constitutive tyrosine activity that enhances the tumorigenicity of glioblastomas in vivo. Because of the tumor-specific extracellular sequence, the mutant receptor is an attractive potential target for cancer therapy, particular via the use of immunotoxins (e.g., MR1(Fv)–PE38).

Technology

The technology claimed in the patent application is directed to antibodies to an epidermal growth factor receptor known as EGFRvIII. In particular, the invention provides an antibody, designated MR1–1, which mutates MR1 in the CDR3 of the (V_H) and (V_L) chains to provide an antibody with especially good cytotoxicity. The described polypeptides can be coupled, attached or otherwise linked to an effector molecule, therapeutic moiety, or detectable label. The patent application provides nucleic acid molecules encoding the polypeptides with a mutated antibody variable heavy (V_H) chain regions or a mutated light chain (V_L) region, or both. The invention also provides methods of killing a cell bearing an antigen comprising contacting the cell with an immunotoxin comprising a toxic moiety and a targeting moiety. The Antibodies and Immunotoxins of claimed in this patent application could be used to develop cancer therapeutics and diagnostics.

The above mentioned Invention is available, including any available foreign intellectual property rights, for licensing.

Dated: April 17, 2000.

Jack Spiegel,

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

[FR Doc. 00–10182 Filed 4–21–00; 8:45 am]

BILLING CODE 4140–01–P

DEPARTMENT OF HEALTH AND HUMAN SERVICES

National Institutes of Health

National Center for Complementary & Alternative Medicine; Notice of Meeting

Pursuant to Section 10(a) of the Federal Advisory Committee Act, as amended (5 U.S.C. Appendix 2), notice is hereby given of the National Advisory Council for Complementary and Alternative Medicine (NACCAM).

The meeting will be open to the public as indicated below, with attendance limited to space available. Individuals who plan to attend and need special assistance, such as sign language interpretation or other reasonable accommodations, should notify the Contact Person listed below in advance of the 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/or contract proposals 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 and/or contract proposals, the disclosure of which would constitute a clearly unwarranted invasion of personal privacy.

Name of Committee: National Advisory Council for Complementary and Alternative Medicine.

Date: May 8–9, 2000.

Open: May 8, 2000, 8:30 am to adjournment.

Agenda: The agenda includes the Director's Report and presentation of NCCAM's Draft Strategic Plan, Development of Trans-NIH Health