To obtain copies of the supporting statement and any related forms for the proposed paperwork collections referenced above, access HCFA's Web Site address at http://www.hcfa.gov/regs/prdact95.htm, or E-mail your request, including your address, phone number, OMB number, and HCFA document identifier, to Paperwork@hcfa.gov, or call the Reports Clearance Office on (410) 786–1326.

Interested persons are invited to send comments regarding the burden or any other aspect of these collections of information requirements.

However, as noted above, comments on these information collection and recordkeeping requirements must be mailed and/or faxed to the designees referenced below, by January 8, 2001:

Health Care Financing Administration, Office of Information Services, Security and Standards Group, Division of HCFA Enterprise Standards, Room N2–14–26, 7500 Security Boulevard, Baltimore, MD 21244–1850. Fax Number: (410) 786–0207 Attn: Julie Brown HCFA–10026

and,

Office of Information and Regulatory Affairs, Office of Management and Budget, Room 10235, New Executive Office Building, Washington, DC 20503, Fax Number: (202) 395–6974 or (202) 395–5167 Attn: Wendy Taylor HCFA Desk Officer.

Dated: December 5, 2000.

John P. Burke III,

HCFA Reports Clearance Officer, HCFA, Office of Information Services, Security and Standards Group, Division of HCFA Enterprise Standards.

[FR Doc. 00–32335 Filed 12–19–00; 8:45 am]

BILLING CODE 4120–03–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 Vasant Gandhi, J.D., 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. 224; fax: 301/402–0220; e-mail: gandhiv@od.nih.gov. A signed Confidential Disclosure Agreement will be required to receive copies of the patent applications.

Human Erythropoietin Receptor Transgenic Mice

Constance T. Noguchi (NIDDK)
DHHS Reference No. E–272–00/0

The inventors have developed a transgenic mouse which expresses the human erythropoietin receptor. Erythropoietin is a cytokine or hormone required for the production of red blood cells and acts by binding on early, undifferentiated blood progenitor cells to stimulate red blood cell formation. The model is particularly useful as human infectious agents or gene therapy vectors that selectively target human cells expressing the erythropoietin receptor can be studied.

Background scientific detail may be found in Liu, C., Liu, Z.Y., Shen, K. and Noguchi, C. T. (1997), ''Regulated human erythropoietin receptor expression in mouse brain'', J. Biol. Chem. 272:32395–32400.

Antitumor Immunity Elicited by Defensin-Tumor Antigen Fusions

Arya Biragyn, Larry W. Kwak (NCI)
DHHS Reference No. E–196–00/0 filed 15 Sep 2000

Tumor antigens are known to be poorly immunogenic and attempts to elicit immune responses against the epitopes of antigens specific to tumor cells have been largely unsuccessful. The inventors have developed a cancer vaccine comprising a defensin fused to a tumor antigen or viral antigen to enhance the immunogenicity of the tumor antigen or viral antigen. The inventors have demonstrated, with animal data, that chimeric proteins, comprising a defensin fused to a model tumor antigen (lymphoma-derived single-chain Fv), when administered to a subject, generate a measurable humoral and anti-tumor cellular immune response.

Methods and Compositions of Viral Chemokine-Antigen Fusion Proteins as Vaccines for Tumors and AIDS

Arya Biragyn, Larry W. Kwak (NCI)
DHHS Reference No. E–194–00/0 filed 15 Sep 2000

Tumor antigens are known to be poorly immunogenic and attempts to elicit immune responses against the epitopes of antigens specific to tumor cells have been largely unsuccessful. The inventors have developed a cancer vaccine comprising a tumor antigen fused with a human chemokine or viral antigen to enhance the immunogenicity of the tumor antigen or viral antigen. The inventors have demonstrated, with animal data, that chimeric proteins, comprising a viral chemokine fused to a model tumor antigen (lymphoma-derived single-chain Fv), when administered to a subject, generate a measurable humoral and anti-tumor cellular immune response.

HCDS1 Kinase Activates Breast Tumor Suppressor BRCA1 and Promotes DNA Damage Repair

Jay H. Chung (NHLBI)
DHHS Reference No. E–192–00/0 filed 06 Jul 2000

BRCA1 plays an important role in the cellular response to DNA damage. The technology relates to the development of BRCA1 serine 988 mutants and a method to modulate BRCA1 activity. For example, one mutant interferes with normal BRCA1 function and may thereby increase sensitivity of tumor cells to chemotherapeutic agents. Another mutant shows constitutive activity in the absence of cell cycle checkpoint enzyme hCds1 activation and may thereby increase the resistance of normal tissue to genotoxic agents such as ionizing radiation.

Specific Binding Agents for KSHV vIL–6 that Neutralize a Biological Activity

Yoshiyasu Aoki, Giovanna Tosato (NCI)
DHHS Reference No. E–180–00/0 filed 31 Jul 2000

This invention relates to the field of herpesviruses, more specifically to human herpesvirus 8 (HHV–8), also known as Kaposi's sarcoma associated herpesvirus (KSHV), and to agents that bind the viral IL–6 encoded by this virus. KSHV encodes various proteins that have features suggesting their role in promoting cellular growth and transformation, including viral homologues of cyclin D, G-protein coupled receptor, interferon regulatory factor, macrophage inflammatory proteins and IL–6. All these viral proteins display structural similarities to their cellular counterparts. The inventors have developed a specific binding agent for KSHV interleukin-6 (vIL–6), which neutralizes vIL–6 activity.

Utilization of Non-Viral Sequences for Minus-Strand DNA Transfer and Gene Reconstitution

Wei-Shau Hu, Vinay K. Pathak (NCI)
DHHS Reference No. E–134–00/0 filed 19 May 2000

This technology relates to novel retroviral vectors for the introduction of heterologous nucleic acid into a host cell. Integration of these vectors into the nucleic acid of a host cell results in reconstitution and duplication of the heterologous nucleic acid in the cellular genome. The invention describes a method to efficiently reconstitute genes during virus replication. Vectors have been developed that enable gene reconstitution, by including two halves of a gene, each half having a small region of homology. The 3′ half of the gene is inserted into the 5′ terminal repeat, before the ''R'' region, and the 5′ half of the gene is inserted into the 3′ terminal repeat, between the ''U3'' region and the ''R'' region. Upon transfer into a cell and viral integration into the genome, two complete copies of the gene are reconstituted (gene duplication), one in the 5′ long terminal repeat (LTR) and one in the 3′ LTR. The virus can be used to transfer two copies of genes, such as toxic genes, into a desired cell population, or can be used to detect the presence of competent retroviruses (as a detection system). This technique can be utilized for delivery of toxic genes for cancer gene therapy or for high-sensitivity detection of replication-competent retroviruses during propagation of viral stocks.

Gadd45a-Null Mice (45C Clone) and Cells Derived from Them

MC Hollander, MS Sheikh, D Bulavin, LA Henmueller (NCI)
DHHS Reference No. E–129–00/0

This technology relates to the creation of a mouse cell line that harbor homozygous deletions of the Gadd45 gene. Gadd45 was the first gene discovered to be controlled by another gene, p53, the most highly mutated gene in human cancer. Cells lacking Gadd45 are less able to deal with DNA damage and are prone to alternations in genomic integrity. Both of these attributes are critical for the prevention of cancer. Gadd45 null mice have a high frequency of parturition failure.

The mice can be used to investigate the effect that the aforementioned attributes have a cell growth and integrity and carcinogenesis. As the Gadd45a-null nice show defects in cell cycle control and DNA repair, they will be useful in toxicology and drug screening. For pharmaceutical studies using chemical libraries, these mice and their derived cells may be useful in identifying inhibitors of specific molecular pathways. Also, the mice will be a useful model for studying delivery failure and cervical dilation.

Usage of Two Yeast Strains in the Identification of Specific Inhibitors of Polo Kinases

Kyung S. Lee, Sukgil Song (NCI)
DHHS reference No. E–100–00/0 filed 23 May 2000

This technology relates to the usage of two yeast strains in the identification of specific inhibitors of polo kinases. Polo kinases are characterized by the presence of a distinct region of homology in the non-catalytic C-terminal domain termed the ''polo-box''. The polo subfamily of protein kinases appears to play a critical role in cell proliferation and cell division. The polo-box domain of mammalian polo kinase, Plk, and the budding yeast functional homolog, Cdc5, are essential for their subcellular localization and functions. The two yeast mutants can be used to screen for inhibitors of polo-box function.

A Transgenic Mouse Model for Tetracycline Regulated Gene Expression in the Mouse Epidermis

Adam B. Glick (NCI)
DHHS Reference No. E–226–99/0

This technology related to the creation of several transgenic mouse lines that will produce conditional overexpression of foreign genes in the mouse epidermis. Foreign genes are frequently expressed in mice to create models of human disease by using a promoter or regulatory region that is tissue specific. In previous models expression of the target gene is always on. In these new models expression is conditional such that timing and level of expression can be completely controlled by the investigator. The inventor has taken advantage of the bigenic tetracycline regulatory system first described by Grossen and Bujard to create the present transgenic mouse lines. The system utilizes two transgenic lines that are then bred together to create a double transgenic mouse. One transgenic line expresses the tetracycline regulated transcriptional transactivator tTA or rTA linked to keratin 5 (K5) promoter. These transgenic lines have been designated K5/tTA and K5/rTA. The K5 promoter is expressed in the epidermis hair follicles and several other squamous epithelia such as tongue trachea and forestomach. The second transgenic line carries the target gene linked to the tetO binding sites for the tTA or rTA proteins. In double transgenic mice, the tTA binds to the tetO sequence and causes high levels of expression of the target gene. However, the ability of the tTA to bind to DNA is prevented by the antibiotic tetracycline. If animals are maintained on tetracycline in the drinking water or fed, the expression of the target gene is suppressed; upon removal of the antibiotic, gene expression is induced. In contrast tetracyclines are required to induce expression of the target by the rTA. The ability of this bigenic system to suppress expression of the target gene is crucial for a functional analysis of genes which produce an embryonic or neonatal lethal phenotype when expressed at high levels during gestation. In addition, different levels of gene expression can be achieved through titration of the tetracycline dose. Studies in the inventor's laboratory has confirmed that the K5/tTA and rTA can transactivate expression of target genes in the epidermis at high levels, uniformly throughout the tissue, and that transactivation is tightly controlled by tetracycline analogues. The mouse epidermis is a useful system for modeling for human fibrotic and blistering skin diseases, dissecting the critical factors in would healing and multistage carcinogenesis in lining epithelia. This conditional expression system should greatly enhance the ability to assess function of specific target genes in these processes, and to create useful *in vivo* models for the development of novel therapeutics.

Dated: December 12, 2000.

Jack Spiegel,

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

[FR Doc. 00–32366 Filed 12–19–00; 8:45 am]

BILLING CODE 4140–01–P

DEPARTMENT OF HEALTH AND HUMAN SERVICES

National Institutes of Health

National Cancer Institute; Notice of Closed Meeting

Pursuant to section 10(d) of the Federal Advisory Committee Act, as amended (5 U.S.C. Appendix 2), notice is hereby given of the following 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 contract proposals and the discussions could disclose confidential trade secrets or commercial