HRSA form	Number of respondents	Responses per respondent	Total responses	Hours per responses	Total burden hours
Total	54	16	864	0.75	648

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: August 31, 2000.

James J. Corrigan,

Associate Administrator for Management and Program Support.

[FR Doc. 00–22947 Filed 9–6–00; 8:45 am]

BILLING CODE 4160–15–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.

Use of Cumulative Distribution Functions To Determine Protein Purity and Homogeneity

Alfred L. Yergey, Paul S. Blank, Christin M. Sjomeling (NICHD)

DHHS Reference No. E–163–00/0 filed 28 Apr 2000

Licensing Contact: Vasant Gandhi; 301/496–7056 ext. 224; e-mail:gandhiv@od.nih.gov

Successful solutions to numerous problems in the biochemical sciences depend on the ability to produce ''pure'' proteins and recognize the degree to which proteins might be modified. Current methods used for assessing purity are relatively nonspecific and insensitive to small differences in molecular weight. The inventors have developed a computer-implemented method and system for nonparametric statistical analysis of matrix-assisted laser desorption ionization (MALDI) protein spectra but is equally applicable to deconvoluted electrospray ionization (ESI) spectra. The invention facilitates assessing protein heterogeneity and detection of otherwise indistinguishable differences in the distribution of molecular weight. A principal advantage is that no additional instrumentation is required beyond that typically included in a mass spectrometry analysis system.

Hsp70-Like ATPase Peptide Binds Chap1/Dsk2

Frederic J. Kaye (NCI)

DHHS Reference No. E–282–99/0 filed 15 Sep 1999

Licensing Contact: Elaine White; 301/496–7056 ext. 282; e-mail: gesee@od.nih.gov

The current invention embodies the identification of a novel gene and protein, Chap1/Dsk2, a ubiquitin-linked protein which appears to play a vital role in regulating mitosis. Identified also is the conserved 20 amino acid region within the ATPase domain of the protein chaperone STCH, an Hsp70-like protein, which is the binding site for Chap1/Dsk2 and other ubiquitin-linked proteins.

Protein chaperones are essential for cell viability, regulating various cell cycle events including the biosynthesis, folding and unfolding, transport, multiunit assembly, and degradation of cell proteins. Overexpression of protein chaperones, such as STCH, can serve to suppress tumorigenesis and apoptosis. It therefore is believed that the peptide identified as the binding domain of STCH may have potential for use as a therapeutic agent against cancer or various infectious diseases, via modulation of tumorigenesis, apoptosis, or the multiunit assembly of viral particles such as HIV.

Polypeptides Comprising IL–6 Ligand Binding Receptor Domains and Related Nucleic Acids, Antibodies, Compositions and Methods

W. Carl Saxinger (NCI)

DHHS Reference No. E–061–99/0 filed 27 Aug 1999

Licensing Contact: Richard Rodriguez; 301/496–7056 ext. 287; e-mail: rodrigur@od.nih.gov

The biological activities of IL–6 include the stimulation of B and T cell growth and differentiation, production of acute-phase proteins by hepatocytes, multilineage hematopoiesis, osteoblast formation, maturation of megakaryocytes and platelet production. An abnormal expression of IL–6 may be involved in the pathogenesis of a variety of diseases, among which are multiple myeloma, rheumatoid arthritis, postmenopausal osteoporosis, chronic autoimmune diseases, Castleman's disease and AIDS. Methods of abrogating the effects of abnormal expression of IL–6 can be made at its site of production or at its target. The inventors of this technology have focused on the latter technique. Using a unique, newly patented, automated peptide array system, the inventors have studied specific sequences potentially involved in protein-protein interactions at the molecular level. This system was used to identify and isolate potential target peptide sequences within the IL–6 receptor molecule. Candidate peptide sequences were identified by direct binding to the IL–6 ligand by optimally displayed IL–6 receptor peptide segments in solid phase form. The specific binding properties of the peptide sequences were verified by using IL–6 heteroantisera, and the peptides have been shown to mitigate or reverse the effects of the above referenced properties of IL–6 in tissue culture.

Receptor-Mediated Uptake of an Extracellular Bcl-XL Fusion Protein Inhibits Apoptosis

Richard J. Youle, Xiuhuai Liu, JoAnn Castelli (NINDS)

DHHS Reference No. E–073–99/0 filed 16 Aug 1999

Licensing Contact: Richard Rodriguez; 301/496–7056 ext. 287; e-mail: rodrigur@od.nih.gov

The present invention relates to the field of apoptosis, in particular, it relates to apoptosis-modifying fusion proteins with at least two domains, one of which targets the fusion proteins to a target cell, and another of which modifies an apoptotic response of the target cell. For example, fusing various cell-binding domains to Bcl-XL and Bad allows targeting to specific subsets of cells in vivo, permitting treatment and/or prevention of cell-death related consequences of various diseases and injuries. This technology could be used to minimize or prevent apoptotic damage that can be caused by neurodegenerative disorders, *e.g.,* Alzheimer's disease, Huntington's disease or spinal-muscular atrophy, stroke episodes or transient ischemic neuronal injury, *e.g.,* spinal cord injuries. Additionally, apoptotic-enhancing fusion proteins of the current invention could be used to inhibit cell growth, *e.g.,* uncontrolled cellular proliferation.

DNA Binding Protein and Sequence as Insulators Having Specific Enhancer Blocking Activity for Regulation of Gene Expression

Adam C. Bell, Adam G. West, Gary Felsenfeld (NIDDK)

DHHS Reference Nos. E–220–98/0 filed 30 Jun 1999 and E–220-98/1 filed 19 Apr 2000

Licensing Contact: Girish Barua; 301/496–7735 ext. 263; e-mail: gb18t@nih.gov

This patent application has two components. The first is the identification of a functional 50bp fragment of a previously known chicken chromatin insulator protein. The second component is the identification of the REBL (Required for Enhancer Blocking) CTCF protein (CCCTC-binding factor) which binds to the 50bp fragment. [The relationship between these two can be analogized as a receptor (50 bp fragment) and its ligand (the REBL CTCF protein).] These two elements can be used separately or together to regulate gene expression.

An insulator is a DNA sequence which is capable of acting as a barrier to neighboring cis-acting elements, preventing gene activation when juxtaposed between an enhancer and a promoter (*i.e.,* when the insulator is placed between the enhancer and the promoter gene activation is blocked). An insulator will also act to protect a stably integrated reporter gene from position effects. This 50 bp fragment represents a functionally active domain of the chicken insulator protein which is both necessary and sufficient for enhancer blocking activity in human cells. The previously described chicken chromatin insulator is a 1.2 kb fragment which, where overall size of the vector to be delivered is a concern, for example, in gene therapy, may be too large for some applications. The identification of this active 50 bp fragment may therefore be a preferred alternative.

The identification of the REBL CTCF protein as an agent which binds to the 50 bp insulator fragment and whose binding activity is necessary for blocking of enhancer activity provides an additional element which may be used to more specifically control gene regulation. As most gene expression is dependent on the activity of multiple components the identification of a specific binding factor which functions as a blocking enhancer activity may permit more precise control of gene expression. The human REBL protein has regions which share homology with previously disclosed partial human cDNAs. It has a molecular weight of 135 kDa. A chicken homolog has also been identified. CTCF was originally identified as a repressor of the chicken c-myc gene.

Dated: August 29, 2000.

Jack Spiegel,

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

[FR Doc. 00–22880 Filed 9–6–00; 8:45 am]

BILLING CODE 4140–01–U

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.

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ADDRESSES: Licensing information and copies of the U.S. patent applications listed below may be obtained by contacting Marlene Shinn, J.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. 285; fax: 301/402–0220; e-mail: shinnm@od.nih.gov. A signed Confidential Disclosure Agreement will be required to receive copies of the patent applications.

Inhibition of Smad3 To Prevent Fibrosis and Improve Wound Healing

Anita B. Roberts *et al.* (NCI)

DHHS Reference No. E–070–00/0 filed 19 May 2000; PCT/US00/13725

Millions of dollars are spent each year to heal chronic non-healing wounds and in the treatment of severe burn patients. The NIH announces a new technology that may lead to improved approaches to treatment of burn patients and the reduction of scarring and more rapid closure of both acute (surgical) and chronic wounds (e.g., diabetic, decubitus, and venus statis ulcers).

Smad2 and Smad3 are highly homologous cytoplasmic proteins which function to transduce signals from Transforming Growth Factor-beta (TGF–β) and activin receptors to promoters of target genes found in the nucleus. This new technology indicates that interference with specific signaling pathways downstream of TGF–β may be more selective and have a better outcome than approaches aimed at blocking all effects of this pleiotropic cytokine. Specifically, it is proposed that elimination or inhibition of Smad3 may interfere with fibrogenic mechanisms and reduce the accumulation of scar tissue associated with high dose radiation and wound healing, while increasing the rate of re-epithelialization of wounds.

Although this technology is still in an early stage, our researchers have obtained solid evidence of the involvement of Smad3 in these processes by use of a Smad3 null mouse model which they have developed. Based on these results, it is believed that antisense Smad3 or small molecule inhibitors of Smad3 will have clinical applications in wound healing, in improving growth and reducing unwanted fibrosis of autologous skin grafts for treatment of burn patients, and in treatment of radiation fibrosis and other fibrotic diseases associated with chronic inflammation. In addition, the discovery of inhibitors to Smad3 signaling may lead to radiation dose escalation and accelerated tumor cell death while reducing the side effects associated with radiation therapy.