Type of activity—45 CFR 60.0	Number of respondents	Responses per respondent	Hours per response	Total burden hours
Requests for Information Disclosure (Query):				
Queries by Hospitals for Practitioner Applications—60.10(a)(1)	6,000	40	.083 5 Minutes	20,000
Queries by Hospitals—Two Year Cycle—60.10(a)(2)	6,000	160	.083	80,000
Queries by Hospitals—Peer Review—60.11(a)(1)	(**)			
Queries by Practitioners (Self-Query)—60.11(a)(2)	60,000	1	.50	30,000
Queries by Licensure Boards—60.11(a)(3)	125	120	.083	1,245
Queries by Non-Hospital Health Care Entities—60.11(a)(4)	3,250	690	.083	186,874
Queries by Plaintiff's Attorneys—60.11(a)(5)	***1	1	.30	.5
Queries by Non-Hospital Health Care Entities—Peer Review—60.11(a)(6)	***			
Requests by Researchers for Aggregate Information—60.11(a)(7)	100	1	.50	50
Disputes:				
Practitioner Places a Dispute in His/Her Data Bank Report—60.14(b)	1,200	1	.5	600
Practitioner Places a Statement in His/Her Data Bank Report—60.14(b)	1,350	1	1.0	1,350
Practitioner Requests Review of the Disputed Report by The Secretary DHHS—60.14(b).	135	1	8.0	1,080
Administrative forms used in operating the National Practitioner Data Bank;				
Entity Registration Form	150	1	1.0	150
Entity Registration Update Form	100	1	.25	25
Authorized Agent Designation Form	25	1	.25	6.25
Authorized Agent Designation Update	5	1	.083	.42
Account Discrepancy Report	200	1	.25	50
Electronic Transfer of Funds Authorization	25	1	.25	6.25
Entity Reactivation	50	1	.25	12.5
Total				336,757

*There have been no hearing requests from reporting entities since the opening of the Data Bank.
**We are unable to distinguish between these and other types of queries made by hospitals and other health care entities.
***There have been approximately 12 attorney requests since the opening of the Data Bank; of these, one has been granted.

Written comments and recommendations concerning the proposed information collection should be sent within 30 days of this notice to: Wendy A. Taylor, Human Resources and Housing Branch, Office of Management and Budget, New Executive Office Building, Room 10235, Washington, DC 20503.

Dated: January 26, 1999.

Jane Harrison,

Director, Division of Policy Review and Coordination.

[FR Doc. 99–2233 Filed 1–29–99; 8:45 am]

BILLING CODE 4160–15–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.

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.

Novel Human Cancer Antigen, NY ESO–1/CAG–3, and Gene Encoding Same

R Wang, SA Rosenberg (NCI)

DHHS Reference No. E–265–97/1 filed 21 Sep 98

Licensing Contact: Elaine Gese; 301/496–7056 ext. 282; e-mail: eg46t@nih.gov

The current invention embodies the identification, isolation and cloning of a gene encoding a novel tumor antigen, NY ESO–1/CAG–3, as well as cancer peptides thereof an antigenic cancer epitopes contained within the cancer peptides. This novel antigen is recognized by cytotoxic T lymphocyte clones derived from the TIL586 (tumor infiltrating lymphocyte) cell line in an HLA restricted manner.

The inventors believe that cancer peptides which are encoded by the NY ESO–1/CAG–3 gene represent potential cancer vaccines, protecting an individual from development of cancer by inhibiting the growth of cells or tumors which express the NY ESO–1/CAG–3 antigen. Also embodied in the invention are pharmaceutical compositions comprising the NY ESO–1/CAG–3 antigen, peptide, or an antigenic cancer epitope thereof in combination with one or more immunostimulatory molecules. These compositions represent potential anticancer therapeutics, stimulating NY ESO–1/CAG–3–specific T cells to elicit an anti-cancer immunogenic response and thereby eliminating or reducing the cancer. While these vaccines and pharmaceutical compositions may be developed for use against a variety of cancers, data obtained to date indicate that they may be of particular value for use against melanoma.

Methods for diagnosing cancer via the detection of NY ESO–1/CAG–3 are also embodied in the invention.

Mouse Models for Huntington's Disease

D. Tagle (NHGRI)

DHHS Reference No. E–101–98/0

Licensing Contact: Marlene Shinn; 301/496–7056 ext. 285; e-mail: ms482m@nih.gov

Huntington's Disease (HD) is one of a number of neurological diseases in which excessive repetition of the CAG nucleotide sequence, which codes for glutamines, causes an abnormally shaped HD protein. This protein then interacts with other proteins produced by the cell thus preventing their normal functions. HD afflicts 1 in every 10,000 individuals in the United States, however HD's pathogenesis and mechanistic action is relevant to at least 13 other neurodegenerative diseases.

The mouse lines which are available for licensing show progressive neurobehavioral and neuropathological changes that resemble clinical findings found in HD patients. These include behavior such as running in circles, performing backflips and other abnormal movements which correlate with the loss of neurons in the striatum, cortex, and other brain regions. The transgenic mice have been genetically engineered to show widespread expression of full length human HD cDNA with either 16, 48, or 89 CAG repeats. It is the mice containing the 48 or 89 CAG repeats which manifest the HD symptoms, the other modified mice are useful as controls. The mouse lines are able to model the early events that occur in Huntington's Disease and how these events ultimately result in neurological cell death. The utility of these mouse lines can be found in screening potential pharmaceutical treatments for HD and other neurodegenerative diseases, as well as testing therapies, including those used to assist neuronal survival.

Inhibition of T-Type Voltage-Gated Calcium Channels by a New Scorpion Toxin

K Swartz, H Jaffe (NINDS)
Serial No. 60/101,158 filed 21 Aug 98
Licensing Contact: Marlene Shinn, 301/496–7056 ext. 285; e-mail: ms482m@nih.gov

The T-Type calcium channel is found in neurons, cardiac and vascular smooth muscle and is thought to be important for generative specific patterns of electrical activity. We have identified, isolated, and determined the chemical composition of an inhibitor (named Kurtoxin-1) of the T-type calcium channel. Kurtoxin-1 (or drugs developed using it as a probe) may be useful therapeutic reagents to control heart rate (e.g., antiarrhythmic drugs), vascular smooth muscle tone (e.g., controlling blood pressure) or epileptic discharges in the central nervous system. T-type calcium channels may also be important for transmission of pain stimuli and therefore inhibitors of these channels may have analgestic properties.

Kurtoxin is from the venom of the *Parabuthus transvaalicus* scorpion. It binds to the α_{1G} T-type Ca^{2+} channel with high affinity and inhibits the channel by modifying voltage-dependent gating. The biophysical properties of T-type voltage-gated Ca^{2+} channels make them well suited to serve important pacemaking roles, and to support c flux near the resting membrane potential in both excitable and non-excitable cells. Until now, no selective high affinity ligands were available for T-type Ca^{2+} channels. Kurtoxin distinguishes between the α_{1G} T-type Ca^{2+} channels and other types of voltage-gated Ca^{2+} channels, such as α_{1E}, α_{1C}, α_{1B} and α_{1A}. Its primary amino acid sequence indicates it belongs to a family of ŧ-scorpion toxins that slow inactivation of Na^{+} channels. It is foreseen that kurtoxin will facilitate characterization of the molecular composition of T-type Ca^{2+} channels and will help delineate their involvement in electrical and biochemical signaling.

Composition and Methods for Identifying and Testing Tyrosine Kinase Substrates and Their Agonists and Antagonists

LE Samelson, W Zhang (NICHD)
Serial No. 60/068,690 filed 23 Dec 97
Licensing Contact: Susan S. Rucker; 301/496–7056 ext. 245; e-mail: sr156v@nih.gov

This application relates to T cell receptors (TCRs) and TCR mediated signal transduction. More particularly, the application describes the isolation, purification and cloning of an integral membrane protein, Linker for Activation of T cells (LAT), a tyrosine kinase substrate for ZAP–70/Syk protein tyrosine kinases (PTKs). LAT is phosphorylated by ZAP–70/Syk and this phosphorylation is necessary for the recruitment of multiple signaling molecules, such as Grb2, PLC-γ1, the p85 subunit of PI3K and other critical signaling molecules. Thus, LAT plays a role in linking the TCR to cellular activation. Tissues which express LAT are limited to the thymus, peripheral blood, and at low levels, the spleen. Cells, found in these tissues, which express LAT and T cells, NK cells and mast cells. In addition recent work has also demonstrated that LAT is expressed in megakarocytes. B cells and monocytes do not express LAT. This pattern of expression and its role in cell signaling suggest that LAT may be a specific target for the development of drugs for allergy and other T cell associated diseases. Such drugs may include antibodies which recognize LAT and inhibit its action.

In addition to the isolation, purification and cloning of LAT the application describes antibodies which specifically recognize LAT. Recent work has shown that LAT is palmitoylated and this palmitoylated LAT localizes to glycolipid-enriched microdomains (GEMs). The palmitoylation of LAT is necessary for the tyrosine phosporylation of LAT and for the targeting of LAT to the GEMs. Other recent work includes the generation of LAT knockout mice.

This research has been published in *Cell* 92(1): 83–92 (Jan 9, 1998) and *Immunity* 9(2): 239–46 (Aug 1998).

Probe To Identify Enteroinvasive *E. coli* and *Shigella* Species

KA Lampel, JA Jagow (FDA)
Serial No. 07/266,038 filed 02 Nov 88; U.S. Patent 5,041,372 issued 20 Aug 91
Licensing Contact: Carol Salata, 301/496–7735 ext. 232; e-mail: cs253n@nih.gov

Standard means for detecting pathogenic organisms in food or clinical specimens rely on animals or large DNA fragments, such as the 17 kb *Eco*RI fragment of Boileau. These methods are expensive, time-consuming, difficult to use, and have not been able to distinguish between nonvirulent enteroinvasive *E. coli* and *Shigella*. This invention described DNA probes for enteroinvasive *E. coli* and *Shigella* species, including the sequence of the 2.5 kb fragment (*Sma*II and Falkow's) on which the probe is based.

The probe is more reliable, more sensitive, and less expensive than methods now is use.

Dated: January 25, 1999.

Jack Spiegel,

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

[FR Doc. 99–2245 Filed 1–29–99; 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