Neuroblastomas are malignant cancers that start in nerve tissue and primarily affect infants and children. Although frontline treatments for neuroblastomas are often effective, relapse frequently occurs in high risk cases. The most common form of relapse in neuroblastoma patients is caused by Neuroblastoma tumor initiating cells (NB–TIC). Therefore, if NB–TIC could be eliminated, high risk neuroblastoma patients could have a therapeutic option for preventing a relapse. This invention concerns the discovery that NB–TIC expresses CD22. As a result, NB–TIC are susceptible to treatment with an anti-CD22 immunotoxin. By combining frontline...
neuroblastoma treatments with anti-CD22 immunotoxins, both the primary neuroblastoma and cells capable of initiating a relapse can be eliminated. As a result, even high risk neuroblastoma patients should have an increased chance of surviving neuroblastoma.

**Application:** Treatment and prevention of neuroblastoma relapse.

**Advantages:**
- Increased therapeutic effectiveness with decreased non-specific killing of essential, healthy cells.
- Neuroblastoma relapse commonly begins in the bone marrow, an environment which is accessible to immunotoxins.
- Combined treatment addresses both the tumor and the cause of relapse, leading to more efficient treatments than frontline therapeutics alone.

**Development Status:** Preclinical stage of development for treatment of neuroblastoma relapse; immunotoxins have clinical data associated with neuroblastoma relapse; immunotoxins with decreased non-specific killing of essential, healthy cells.

**Advantages:**
- Identification of potential molecular targets for cancer diagnosis, prevention, and treatment.
- Tools to understand the genetic changes during cancer development.

**Applications:**
- Tools to understand the genetic changes during cancer development.
- Tools to understand the genetic changes during cancer development.

**Collaborative Research Opportunity:**
- Available for collaborative research to further develop, evaluate, or commercialize recombinant anti-CD22 immunotoxins for the treatment of neuroblastoma. Please contact John Hewes, Ph.D. at 301–435–3121 or hewesj@mail.nih.gov for more information.

**Mouse Model of Thyroid Cancer**

**Description of Technology:** This technology describes a mouse model of thyroid cancer where the phosphatidylinositol 3-kinase (PI3K)–AKT/protein kinase B-signaling pathway is overactivated. These mice have a knock-in dominantly negative mutant thyroid hormone receptor β gene (TRβPV mutant) that spontaneously develops thyroid cancer and distant metastasis similar to human follicular thyroid cancer. The thyroids of TRβPV mice exhibit extensive hyperplasia, which progresses to capsular invasion, vascular invasion, anaplasia, and ultimately, metastasis to distant organs. Consequently, this mouse model could be used as a preclinical model to understand genetic changes during cancer development and to identify potential molecular targets for the diagnosis, prevention, and treatment of cancer. For example, the inventors have used the TRβPV mice to show that the peroxisome proliferator-activated receptor γ (PPARγ) could function as a tumor suppressor in vivo and that the activation of the PI3K–AKT signaling contributes to thyroid carcinogenesis and could be a potential therapeutic target in follicular thyroid carcinoma.

**Applications:**
- Identification of potential molecular targets for cancer diagnosis, prevention, and treatment.
- Testing kinase inhibitors and other novel drugs being discovered for the treatment of thyroid cancer.
- Tools to understand the genetic changes during cancer development.

**Advantages:**
- This model provides the opportunity to study the alterations in gene regulation that occur during the progression and metastasis of thyroid carcinogenesis, not just the genes that control initial carcinogenesis.

**Development Status:** The technology is currently in the pre-clinical stage of development.

**Inventors:** Thiele (NCI) et al.


For more information, see:
- U.S. Patent 7,355,012—“Mutated Anti-CD22 Antibodies with Increased Affinity to CD22—Expressing Leukemia Cells”.

**Licensing Status:** Available for licensing.

**Licensing Contact:** David A. Lambertson, PhD; 301–435–4632; lambertsond@mail.nih.gov.

**Related Publications:**


Chemokine-Tumor Antigen Fusion Proteins as Cancer Vaccines

**Description of Technology:** Available for licensing is a tumor vaccine construct comprising a chemotractant (such as human chemokines CCL7 and CCL20) fused to a tumor antigen (including human mucin-1, a transmembrane protein that is aberrantly expressed in cancer; or single chain antibody expressed by B cell malignancy, or melanoma antigen gp100 expressed in human melanomas). The majority of tumor antigens are believed to be poorly immunogenic because they represent oncogene gene products or other cellular genes which are normally present in the host. As a result, poor immunogenicity has been a major obstacle to successful immunotherapy with tumor vaccines. Administration of this fusion chemokine and tumor antigen protein, or a nucleic acid encoding this fusion protein, elicits a tumor specific cellular and humoral immune response thereby providing a potent cancer vaccine.

**Applications:** Cancer immunotherapy.

**Development Status:** Proof of the concept and pre-clinical development have been successfully completed.

**Market:** The global cancer market is forecasted to reach US$40 billion by 2012. Cancer vaccine research is coming to fruition, with a number of products now in Phase III trials and 15 therapeutic cancer vaccines realistically expected to launch by 2013. The therapeutic vaccine market has the potential to mirror the growth seen in the monoclonal antibody market, and reach sales in excess of US$5 billion by 2012.

**Inventors:** Larry Kwak (NCI) and Aarya Biragyn (NIA) (both NCI at time of invention).


Licensing Contact: Patrick McCue, PhD; 301–435–5560; mccuepat@mail.nih.gov.

**Collaborative Research Opportunity: The National Institute on Aging, Laboratory of Immunology, is seeking statements of capability or interest from parties interested in collaborative research to further develop, evaluate, or commercialize cancer vaccines that target skin antigen-resenting cells.**

Please contact Nicole Guyton at 301–435–3101 or guytonn@mail.nih.gov for more information.


**Richard U. Rodriguez, Director, Division of Technology Development and Transfer, Office of Technology Transfer, National Institutes of Health.**

[FR Doc. 2010–27179 Filed 10–26–10; 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, HHS.

**ACTION:** Notice.

**SUMMARY:** The inventions listed below are owned by an agency 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.

**IL–10 and IFNγ Peptide Inhibitors**

**Description of Invention:** Available for licensing are several potent and selective inhibitors of IL–10 and IFNγ signaling. Although cytokines play important roles in cancer and inflammation, there are no specific inhibitors of any cytokines to date. IL–10 and IFNγ cytokine signaling play crucial roles in inflammation, cancer growth, and autoimmune diseases. The investigators have developed short peptides that potently and selectively interfere with dimerization of the cytokines and their binding to the corresponding receptor. Included in the patent application are also metabolically stable lipopeptides mimicking conserved regions of IL–10 and IFNγ receptors that interfere with STAT3 and STAT1 phosphorylation and subsequent signaling. Lipopeptides potently inhibit STAT3 and STAT1-dependent growth of cancer cells. These compounds are promising drug candidates for the treatment of cancer and many infectious and inflammatory diseases.

**Application:** Cancer, viral infections and anti-inflammatory treatments.

**Advantages:**
- Potent, stable peptide inhibitors.
- Selective IL–10 and IFNγ inhibitors.

**Development Status:** The technology is currently in the pre-clinical stage of development.

**Market:** The annual growth rate for the therapeutic peptide market is estimated at about 7.5%.

**Inventors:** Nadya Tarasova et al. (NCI).


**Licensing Status:** Available for licensing.

**Licensing Contact:** Jennifer Wong; 301–435–4633; wongje@mail.nih.gov.

**Collaborative Research Opportunity:** The Center for Cancer Research, Cancer and Inflammation Program, is seeking statements of capability or interest from parties interested in collaborative research to further develop, evaluate, or commercialize inhibitors of IL–10, IFNγ and STAT3 signaling. Please contact John Hewes, Ph.D. at 301–435–3121 or hewesj@mail.nih.gov for more information.

**Diagnostic and Prognostic HCC-Related Metabolites**

**Description of Invention:** Metabolite profiling identifies and measures changes in cellular metabolites as a means to determine a direct correlation between gene expression and changes in biological function. Investigators at the National Cancer Institute have identified a unique set of metabolite biomarkers associated with hepatocellular carcinoma (HCC), early stage HCC, HCC patient outcome and HCC stem-cell subtype. Subsets of this metabolite/gene signature can distinguish HCC tumors from normal tissues with 88–97% accuracy, identify early stage HCC patients with 62–78% accuracy, wherein early stage is defined as TNM stage I, prognose negative patient outcome, and identify a HCC stem cell subtype with 70–77% accuracy. These metabolites and gene surrogates are elements of the P53K and Myc signaling networks which can potentially be targeted for therapeutic purposes.