sense of alarm among patients and practitioners.

Response: The purpose of the November 17, 2003, **Federal Register** notice was to describe the proposed study and offer an opportunity for comment by the sponsors of marketed triptans. The responses to the comments in this notice also provide additional explanation and another opportunity for all interested parties to participate through additional comments. In addition, FDA has responded in this document to those comments expressing concern with the study methods.

References

The following references have been placed on display in the Division of Dockets Management (HFA–305), Food and Drug Administration, 5630 Fishers Lane, rm. 1061, Rockville, MD 20857, and may be seen by interested persons between 9 a.m. and 4 p.m., Monday through Friday. (FDA has verified the Web site address, but FDA is not responsible for any subsequent changes to the Web site after this document publishes in the **Federal Register**.)

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11. McAlindon, T., M. Formica, M. LaValley, M. Lehmer, K. Kabbara, ''Effectiveness of glucosamine for symptoms of knee osteoarthritis: results from an internet-based randomized double-blind controlled trial,'' *American Journal of Medicine*, 117(9):643–9, 2004.

12. McAlindon, T., M. Formica, K. Kabbara, M. LaValley, M. Lehmer, ''Conducting clinical trials over the internet: feasibility study,'' *The British Medical Journal*, 327(7413):484–7, 2003.

13. McAlindon, T.E., M.K. Formica, C.E. Chaisson, R. Woods, J. Fletcher, ''Feasibility Of An Internet-Based Case-Control Study Of Recent-Onset SLE,'' *Arthritis Rheum*, 50(9 (Suppl)):682, 2004.

FDA estimates that approximately 500 persons will voluntarily complete the questionnaire. The estimated time for completing each questionnaire is approximately 2 hours, resulting in a total burden of 1,000 hours per year. The burden of this collection of information is estimated as follows:

TABLE 1.—ESTIMATED ONE-TIME REPORTING BURDEN[1]

No. of Respondents	Annual Frequency per Response	Total Annual Responses	Hours per Response	Total Hours
500	1	500	2	1,000

[1] There are no capital costs or operating and maintenance costs associated with this collection of information.

Dated: October 26, 2005.

Jeffrey Shuren,

Assistant Commissioner for Policy.

[FR Doc. 05–21807 Filed 11–1–05; 8:45 am]

BILLING CODE 4160–01–S

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 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.

Antibodies Against Insulin-Like Growth Factor II and Uses Thereof

Dimiter S. Dimitrov et al. (NCI).

U.S. Provisional Application No. 60/709,226 filed 17 Aug 2005 (HHS Reference No. E–217–2005/0–US–01).

Licensing Contact: Michelle A. Booden; 301/451–7337; *boodenm@mail.nih.gov.*

The type 1 insulin-like growth factor (IGF) receptor (IGF1R) is over-expressed by many tumors and mediates proliferation, motility, and protection from apoptosis. Agents that inhibit IGF1R expression or function can potentially block tumor growth and metastasis. Its major ligand, IGF–II, is over-expressed by multiple tumor types. Previous studies indicate that inhibition of IGF–II binding to its cognizant receptor negatively modulates signal transduction through the IGF pathway and concomitant cell growth.

The present invention relates to the identification of multiple, novel fully human monoclonal antibodies that are specific for IGF–II and do not cross-react with IGF–1 or insulin. The present invention also describes methods employing these novel antibodies to inhibit IGF–1R phosphorylation and

concomitant cell growth and motility. The invention also encompasses other IGF–II antibodies or derivatives of the original antibodies and methods of using said antibodies to block binding of ligands. Additional embodiments describe methods for treating various human diseases associated with aberrant cell growth and motility including breast, prostate, and leukemia carcinomas. Thus, these novel IGF–II antibodies may provide a therapeutic intervention for multiple carcinomas without the negative side effects associated with IGF I and insulin inhibition.

This technology is available for licensing under an exclusive or non-exclusive patent license.

In addition to licensing, the technology is available for further development through collaborative research opportunities with the inventors.

Compositions and Methods for Diagnosis and Treatment of Chemotherapy-Resistant Neoplastic Disease

John Park (NINDS).
U.S. Provisional Application No. 60/571,296 filed 15 May 2004 (HHS Reference No. E–192–2004/0–US–01); PCT Application No. PCT/US2005/016924 filed 13 May 2005 (HHS Reference No. E–192–2004/0–PCT–02).
Licensing Contact: Jesse S. Kindra; 301/435–5559; *kindraj@mail.nih.gov.*

The present invention relates to compositions and methods for the treatment of a neoplastic disease state (*i.e.* tumors) using RNA interference-mediated down regulation of stathmin expression. This invention also discloses methods for determining the presence or predisposition to a neoplastic disease state.

Stathmin is a cytoplasmic protein that is highly expressed in many different types of tumors such as leukemias, lung cancers and brain tumors. Stathmin is believed to be involved in the regulation of the cell cycle via its interactions with microtubules. Lowering the expression of stathmin in tumor cells using RNA interference (RNAi) technology causes a decrease in tumor cell growth and also causes such cells to become more sensitive to the effects of standard chemotherapeutic agents.

Accordingly, the delivery of stathmin RNAi oligonucleotides either alone or in combination with standard chemotherapies may be used to treat patients with various tumors. For example, retroviruses or adeno-associated viruses containing stathmin RNAi oligonucleotides could be delivered to brain tumors in order to decrease cell growth and increase sensitivity to standard chemotherapies.

Serine Protease Inhibitors

Peter P. Roller, Peng Li (NCI).
PCT Patent Application No. PCT/US2004/34108 filed 15 Oct 2004 (HHS Reference No. E–272–2002/1–PCT–01).
Licensing Contact: Mojdeh Bahar; 301/435–2950; *baharm@mail.nih.gov.*

This disclosure concerns novel serine protease inhibitors and methods for using the inhibitors to reduce tumor progression and/or metastasis. Embodiments of the inhibitors are highly effective, selective inhibitors of matriptase, which has been implicated in tissue remodeling associated with the growth of cancerous tumors and cancer metastasis.

Angiogenesis and tumor invasion require that the normal tissue surrounding the tumor be broken down in a process referred to as tissue remodeling. Tissue remodeling is accomplished by a host of enzymes that break down the proteins in the normal tissue barriers comprising the extracellular matrix. Among the enzymes associated with degradation of the extracellular matrix and tissue remodeling are a number of proteases. The expression of some of these proteases has been correlated with tumor progression.

The disclosed compounds can be used to inhibit matriptase, MTSP1, or both, in vitro and in vivo and thus can be used in the prevention or treatment of conditions characterized by abnormal or pathological serine protease activity. For example, the compounds are useful for prevention or treatment of conditions characterized by the pathological degradation of the extracellular matrix, such as conditions characterized by neovascularization or angiogenesis, including cancerous conditions, particularly metastatic cancerous conditions where matriptase is implicated. The disclosed compounds can be used to decrease the degradation of the cellular matrix and thereby reduce concomitant tumor progression and metastasis. Conditions characterized by abnormal or pathological serine protease activity that can be treated according to the disclosed method include those characterized by abnormal cell growth and/or differentiation, such as cancers and other neoplastic conditions. Typical examples of cancers that may be treated according to the disclosed inhibitors and method include colon, pancreatic, prostate, head and neck, gastric, renal, and brain cancers.

Dated: October 25, 2005.

Steven M. Ferguson,

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

[FR Doc. 05–21831 Filed 11–1–05; 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.

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Method To Disrupt Protein-Protein Interactions and Its Use To Identify Compounds Able To Inhibit HIV–1 Rev Protein Multimerization

George Pavlakis and Leonid Suvoroz (NCI).
HHS Reference No. E–303–2005/0—Research Tool.
Licensing Contact: Sally Hu; 301/435–5606; *hus@mail.nih.gov.*

The invention provides a FRET-based assay for the study of Rev-Rev interaction in vitro, based on YFP and CFP expression constructs for Rev. Using this assay, Rev-derived small peptides that can inhibit Rev-Rev interactions and disrupt dimerization were discovered. This assay can be used as an *in vitro* assay for studying protein-protein interactions in general, and for the discovery of inhibitors or agonists of such interactions as potential drugs against HIV infections, as well as for the