attitudes towards substance abuse. In order to test the effectiveness of the interactive multimedia module, data will be collected in the form of pre and post test surveys from 10th and 11th grade high school students utilizing the developed module. The findings will provide valuable information regarding information pertaining to the use of interactive multimedia educational modules in high school science classrooms and their ability to increase knowledge and change attitudes and perceptions.

Frequency of Response: 4. *Affected Public:* High school students engaged with the *ArchieMD: The Science of Drugs* program. *Type of Respondent:* Participants will include high school students enrolled in the tenth and eleventh grade. *Estimated Total Annual Number of Respondents:* 360. *Estimated Number of Responses per Respondent:* 4. *Average Burden Hours per Response:* One high school period lasting 50 minutes. *Estimated Total Annual Burden Hours Requested:* 1199.95. There are no Capital Costs to report. There are no Operating or Maintenance Costs to report. The estimated annualized burden is summarized below.

Type of respondents	Number of respondents	Frequency of response	Average burden hours per response	Estimated total burden hours requested
Participants—High School Students	360	4	.8333	1199.95
Total	360	4	.8333	1199.95

Request for Comments: Written comments and/or suggestions from the public and affected agencies are invited on one or more of the following points: (1) Whether the proposed collection of information is necessary for the proper performance of the functions of the agency, including whether the information shall have practical utility; (2) the accuracy of the agency's estimate of the burden of the proposed collection of information; (3) ways to enhance the quality, utility, and clarity of the information to be collected; and (4) ways to minimize the burden of the collection of information on respondents, including through the use of automated collection techniques or other forms of information technology.

Comments Due Date: Comments regarding this information collection are best assured of having their full effect if received within 30-days of the date of this publication.

FOR FURTHER INFORMATION CONTACT: To request more information on the proposed project or to obtain a copy of the information collection plans, please contact Dr. Cathrine Sasek, Coordinator, Science Education Program, Office of Science Policy and Communications, National Institute on Drug Abuse, 6001 Executive Blvd, Room 5237, Bethesda, MD 20892, or call non-toll-free number (301) 443–6071; fax (301) 443–6277; or by e-mail to *csasek@nida.nih.gov.*

Comments Due Date: Comments regarding this information collection are best assured of having their full effect if received within 60-days of the date of this publication.

Dated: August 25, 2008.

Mary Affeldt,

Associate Director for Management, National Institute on Drug Abuse, National Institutes of Health.

[FR Doc. E8–20778 Filed 9–5–08; 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.

Over-Expression and Mutation of a Tyrosine Kinase Receptor FGFR4 in Tumors

Description of Technology: Rhabdomyosarcoma (RMS) is the most common type of pediatric soft tissue sarcoma. Most children (>70%) with the disease die at higher stage (metastatic disease).

Researchers at NIH have identified mutations in fibroblast growth factor receptor 4 (FGFR4) that are associated with RMS tumors. It is proposed that individuals with FGFR4 mutations may have an increased risk for tumor metastasis. The identified FGFR4 variants can be used to identify individuals who may benefit most from treatment with an FGFR4 inhibitor as an adjuvant to standard anticancer therapeutics to decrease the risk of tumor metastasis.

Available for licensing are methods for identifying candidates for treatment with an inhibitor of FGFR4 by determining the presence of at least one FGFR4 variant, kits for identifying said candidates, and methods for identifying compounds that induce tumor cell death or that inhibit tumor growth or metastasis.

Applications:

- Potential new method for treatment of Rhabdomyosarcomas (RMS).
- Potential new method to prepare kits to diagnose activating mutations in FGFR4.
- These mutations can be used in laboratory settings to screen thousands of compounds for more specific FGFR4 gene inhibitors.
- FGFR4 is also a potential target for lung and breast cancer.
- FGFR4 monoclonal can be developed to target RMS tumors.

Market:

- In the United States, approximately 12,000 new cases of cancer are diagnosed in children each year. Childhood cancer remains the leading disease-related cause of death in children and adolescents in North America, with about 2,300 deaths each year.
- Rhabdomyosarcoma accounts for about 3 percent of childhood cancers. In the U.S., about 350 children are diagnosed with Rhabdomyosarcoma each year.

Development Status: Early-stage of development.

Inventors: Javed Khan *et al.* (NCI).

Patent Status: U.S. Provisional Application No. 61/044,875 filed 14 Apr 2008 (HHS Reference No. E–175–2008/0–US–01).

Licensing Status: Available for exclusive and non-exclusive licensing.

Licensing Contact: Betty B. Tong, Ph.D.; 301–594–6565; *tongb@mail.nih.gov*.

Collaborative Research Opportunity: The National Cancer Institute, Pediatric Oncology Branch, is seeking statements of capability or interest from parties interested in collaborative research to further develop, evaluate, or commercialize Over-expression and Mutation of a Tyrosine Kinase Receptor FGFR4 in Tumors. Please contact John D. Hewes, Ph.D. at 301–435–3121 or *hewesj@mail.nih.gov* for more information.

Small Molecule Inhibitors of c-Met

Description of Technology: Aberrant c Met signaling is documented in a wide variety of malignancies and occurs via several mechanisms including amplification of c-Met (increased gene copy number), point mutations in the gene encoding c-Met, receptor over-expression, and ligand dependent autocrine/paracrine receptor activation. This application describes novel small molecule inhibitors of c-Met signaling. The small molecules selectively bind to c-Met and have an IC50 in the micromolar range. The small molecules belong to two different families. One family of small molecules reduces the level of c Met expression via receptor down-regulation and blocks ATP binding. The other family of small molecules block ATP binding without inducing receptor down-regulation. Evidence suggests that the second family of compounds bind to both active and inactive conformations of c-Met.

Applications: Therapy for cancers associated with aberrant c-Met signaling, for example bladder, breast, cervical, colorectal, endometrial, esophageal, gastric, head and neck, kidney, liver, lung, nasopharyngeal, ovarian, pancreatic, prostate and thyroid cancers, as well as cholangiocarconoma, osteosarcoma, rhabdomyosarcoma, synovial sarcoma, Kaposi's sarcoma, leiomyosarcomas and MFH/fibrosarcoma. In addition to these malignancies, aberrant c Met signaling is associated with hematological malignancies such as acute myelogenous leukemia, adult T cell leukemia, chronic myeloid leukemia, lymphomas and multiple myeloma as well as other tumors like melanoma, mesothelioma, Wilms' tumor, glioblastomata and astrocytomas.

Market: Although the percentage of cancers associated with aberrant c Met signaling is not yet well established, the wide variety of cancers associated with aberrant c Met signaling are indicative of a potentially large market for these compounds. For example, worldwide over 1 million persons per year are diagnosed with colorectal cancer and it is the most common gastrointestinal cancer in industrialized countries. In one study of colorectal cancer 69% of the patients had at least a two-fold elevation of cMet mRNA and 48% of the patients had at least a ten fold elevation of c Met mRNA. In a study of breast cancer, 22% of patients with invasive ductal breast tumor specimens exhibited strong expression of c Met and patients exhibiting c Met expression had only a 52% 5 year survival rate compared with an 89% 5 year survival rate in patients with normal c Met levels.

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

Inventors: Donald P. Bottaro, Terrence Burke, Jr., *et al.* (NCI).

Patent Status: U.S. Provisional Application No. 61/041,523 filed 01 Apr 2008 (HHS Reference No. E–332–2007/0–US–01).

Publications: The patent application has not been published. There are no journal articles available related to this work.

Licensing Status: Available for licensing on an exclusive or non-exclusive basis.

Licensing Contact: Susan S. Rucker; 301–435–4478; *Susan.Rucker@nih.hhs.gov*.

Collaborative Research Opportunity: The National Cancer Institute, Urologic Oncology Branch, is seeking statements of capability or interest from parties interested in collaborative research to further develop, evaluate, or commercialize small molecule inhibitors of the HGF/c-Met signaling pathway. Please contact John D. Hewes, Ph.D. at 301–435–3121 or *hewesj@mail.nih.gov* for more information.

Quantitative Immunoassays for Measurement of Topoisomerase I as a Pharmacodynamic Marker for the Effect of Anti-Cancer Drugs

Description of Technology: Topoisomerase I (TopoI) is an enzyme that catalyses DNA unwinding which is necessary for many cellular functions. Recent data from the Fluorouracil, Oxaliplatin, CPT–11: Use and Sequencing (FOCUS) trial demonstrates that nuclear staining of TopoI correlates with chemotherapy efficacy [J Clin Oncol (2008) 26, 2690–8]. This enzyme covalently binds with the DNA substrate and introduces a single strand break. Some anti-cancer drugs, including those in clinical trials target this cleavage site and prevent re-ligation of the unwound DNA, trapping the TopoI/DNA covalent complex. TopoI trapped by Topo I inhibitor compounds such as Topotecan is degraded by the ubiquitin/proteosome pathway. This change in intracellular TopoI levels makes total TopoI and the TopoI/DNA covalent complex potential pharmacodynamic biomarkers for monitoring TopoI inhibiting agents, used in cancer therapy.

The technology involves a validated, enzyme linked immunosorbent assay (ELISA) with a chemiluminescence readout, using commercially available antibodies to quantitate total TopoI from cell and tumor extracts.

This technology has been used in a high throughput assay for measurement of estrogen and estrogen metabolites in serum. A similar ELISA assay has also been used in NCI Phase 0 and Phase I clinical trials of a PARP inhibitor

Applications:

- Anti-cancer drug testing.
- Patient selection for anti-cancer drug treatment.

Advantages:

- Simple, quantitative, sensitive (LLQ ~40pg/well LLOD= (LOD 220 pg/ml as formulated), range 200 pg/ml to 50ng/ml).
- Uses commercially available antibodies.
- Excludes the use of radioisotopes.
- Validated Assay.
- SOP available.
- In vitro data support use in anti-cancer drug treated melanoma cell lines.
- Mouse model data support use in anti-cancer drug treated melanoma and colon cancer xenografts.

Developmental Status: ELISA was developed in support of Phase I clinical trial on experimental TopoI inhibiting drugs.

Publication: Thomas D. Pfister, Ralph E. Parchment, Joseph Tomaszewski, James Doroshow and Robert J. Kinders. ''Development of a quantitative immunoassay for measurement of topoisomerase I covalent complex as a pharmacodynamic marker for the effect of anti-cancer drugs.'' AACR Annual Meeting, Los Angeles, CA April 14–18, 2007.

Inventors: Thomas D. Pfister and Robert J. Kinders (SAIC/NCI).

Patent Status: HHS Reference No. E–100–2007/0—Research Tool. Patent protection is not being pursued for this technology.

Licensing Status: Available for non-exclusive licensing of biological material.

Licensing Contact: John Stansberry, Ph.D.; 301–435–5236; *stansbej@mail.nih.gov*.

Collaborative Research Opportunity: The National Cancer Institute's Laboratory of Human Toxicology and Pharmacology is seeking statements of capability or interest from parties interested in collaborative research to further develop, evaluate, or commercialize Quantitative Immunoassays for Measurement of Topoisomerase I as a Pharmacodynamic Marker for the Effect of Anti-Cancer Drugs. Please contact John D. Hewes, Ph.D. at 301–435–3121 or *hewesj@mail.nih.gov* for more information.

New Tumor Endothelial Markers: Genes That Distinguish Physiological and Pathological Angiogenesis

Description of Technology: Angiogenesis, the formation of new blood vessels, is associated with normal physiological processes such as wound healing, ovulation or menstruation as well as with many diseases. Presently, it is thought to be required for the progressive growth of solid tumors and age-related macular degeneration. Lack of disease-specific endothelial markers has hindered the development of cancer therapies targeted against angiogenesis.

This invention describes specific markers that can be used to identify tumor angiogenesis, separate from normal physiological angiogenesis. Several markers have been identified which may serve as potential targets for tumor vessels by using comparative gene expression analysis on various normal and tumor endothelial cells. Furthermore, the invention describes several organ-specific endothelial markers that can aid in the selective delivery of molecular medicine to specific sites. For example, brain endothelial markers (BEMs) and liver endothelial markers (LEMs) described herein could potentially be used to direct molecular medicine specifically to these tissues.

The novel tumor endothelial markers (TEMs) described in this invention also have potential diagnostic ability. These markers can be used to distinguish between normal and tumor tissues. Some of the secreted TEMs can serve as surrogate markers in the determination of the optimum biological dose (OBD) for the current anti-angiogenic drugs in clinical trials.

Applications and Modality:

• Novel therapeutic targets associated with tumor vessels.

• New agents can be developed against these novel targets.

• Novel endothelial markers that distinguish pathological angiogenesis from normal physiological angiogenesis.

• Surrogate tumor endothelial markers that can be used to determine optimal biological dose (OBD) of anti-angiogenic drugs.

Market:

• Sales of the first FDA approved anti-angiogenic drug Avastin™ has reached $600 million.

• Another promising anti-angiogenic molecule, Thalidomide™, has been approved as an anti-cancer agent and for other use in Europe and Australia.

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

Inventors: Brad St. Croix and Steven Seaman (NCI).

Relevant Publication: A Nanda and B St. Croix. Tumor endothelial markers: new targets for cancer therapy. Curr Opin Oncol. 2004 Jan;16(1):44–49.

Patent Status:

• U.S. Provisional Application No. 60/858,068 filed 09 Nov 2006 (HHS Reference No. E–285–2006/0–US–01).

• U.S. Provisional Application No. 60/879,457 filed 08 Jan 2007 (HHS Reference No. E–285–2006/1–US–01).

• PCT Application No. PCT/US2007/072395 filed 28 Jun 2007, which published as WO 2008/057632 on 15 May 2008 (HHS Reference No. E–285–2006/2–PCT–01).

Licensing Status: Available for exclusive and non-exclusive licensing.

Licensing Contact: Adaku Nwachukwu, J.D.; 301–435–5560; *madua@mail.nih.gov*.

Collaborative Research Opportunity: The NIH National Cancer Institute, Tumor Angiogenesis Section, is seeking statements of capability or interest from parties interested in collaborative research to further develop, evaluate, or commercialize specific biomarkers that can be used to identify tumor angiogenesis. Please contact John D. Hewes, PhD at 301/435–3121 or *hewesj@mail.nih.gov* for more information.

Methods of Treating and Preventing Renal Cancer Using a Dimethane Sulfonate Compound

Description of Technology: Currently only a few small molecule inhibitors are effective in patients with renal cell carcinoma. Approximately 30,000 patients per year are diagnosed with this disease but many of them are untreatable because of intrinsic drug resistance, and efficient drug transport and detoxification mechanisms. This invention described and claimed in the patent application describes a series of dimethane sulfonate compounds based on NSC 281612 that are suitable for the treatment of renal cancer. Compositions comprising a pharmaceutically-acceptable carrier and a compound, or a salt suitable for use in the treatment or prevention of renal cancer are also described. The anti-tumor activity of NSC 281612 has been established in vivo against human renal tumor xenografts in mice. Suitable dosing and administration schedules for treatment of renal tumors have also been determined in this study.

Applications: For treatment or prevention of renal cancer.

Development Status: The technology is currently in the pre-clinical stage of development. Phase I clinical trials will begin this fall.

Inventors: Susan D. Mertins, Susan E. Bates, David G. Covell, Geoffrey W. Patton, Melinda G. Hollingshead, B. Rao Vishnuvajjala (NCI).

Patent Status: U.S. Patent Application No. 12/083,583 filed 14 Apr 2008, claiming priority to 14 Oct 2005 (HHS Reference No. E–249–2005/0–US–04).

Licensing Status: Available for exclusive or non-exclusive licensing.

Licensing Contact: Adaku Nwachukwu, J.D.; 301–435–5560; *madua@mail.nih.gov*.

Collaborative Research Opportunity: The National Cancer Institute, Screening Technologies Branch, is seeking statements of capability or interest from parties interested in collaborative research to further develop, evaluate, or commercialize biomarker assays for clinical utility (potential molecular targets have been identified). Please contact John D. Hewes, Ph.D. at 301–435–3121 or *hewesj@mail.nih.gov* for more information.

2-Amino-O^4-Substituted Pteridines: Improved Chemotherapy Adjuvants

Description of Technology: O^6-Benzylguanine derivatives, some O^6-benzylpyrimidines, and related compounds are known to be inactivators of the human DNA repair protein O^6-alkylguanine-DNA alkyltransferase (alkyltransferase). This repair protein is the primary source of resistance many tumor cells develop when exposed to chemotherapeutic agents that modify the O^6-position of DNA guanine residues. Therefore, inactivation of this protein can bring about a significant improvement in the therapeutic effectiveness of these chemotherapy drugs. The prototype inactivator O^6-benzylguanine is currently in clinical trials in the United States as an adjuvant in combination with the chloroethylating agent 1, 3-bis (2-chloroethyl)-1-nitrosourea (BCNU) and the methylating agent temozolomide. A

similar alkyltransferase inactivator, O^6-(4-bromothenyl) guanine is in clinical trials in the UK.

This technology is directed to the discovery of a new class of potent alkyltransferase inactivators, 2-amino-O^4-benzylpteridine derivatives targeted for use in cancer treatment in combination with chemotherapeutic agents such as 1, 3-bis (2-chloroethyl)-1-nitrosurea (BCNU) or temozolomide. The derivatives of the present invention inactivate the O^6-alkylguanine-DNA-alkyltransferase repair protein and thus enhance activity of such chemotherapeutic agents. Some of the derivatives are water soluble and possess tumor cell selectivity in particular by inactivating alkyltransferase in tumor cells that overexpress folic acid receptors. The 2-amino-O^4-benzylpteridine derivatives represent a promising new class of alkyltransferase inactivator with representatives that may be great candidates as chemotherapy adjuvants.

Applications and Modality:

• New small molecules as alkyltransferase inactivators based on 2-amino-O^4-benzylpteridine compounds.

• Promising candidates as chemotherapy adjuvants for the treatment of cancer.

• Therapeutic application for drug resistant tumors where acquired resistance is caused by O^6-alkylguanine-DNA alkyltransferase.

Market:

• 600,000 deaths from cancer related diseases estimated in 2006.

• This technology involving small molecule therapeutics for the treatment of several cancers has a potential market of several billion U.S. dollars.

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

Inventors: Robert C. Moschel (NCI) *et al.*

Publication: ME Nelson, NA Loktionova, AE Pegg, RC Moschel. 2-amino-O^4-benzylpteridine derivatives: Potent inactivators of O^6-alkylguanine-DNA alkyltransferase. J Med Chem. 2004 Jul 15;47(15):3887–3891.

Patent Status:

• U.S. Patent Application No. 10/585,566 filed 29 Aug 2006, claiming priority to 06 Jan 2004 (HHS Reference No. E–274–2003/0–US–03).

• Foreign equivalents

Licensing Status: Available for exclusive or non-exclusive licensing.

Licensing Contact: Adaku Nwachukwu, J.D.; 301–435–5560; *madua@mail.nih.gov*.

Dated: August 26, 2008.

Richard U. Rodriguez,

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

[FR Doc. E8–20651 Filed 9–5–08; 8:45 am]

BILLING CODE 4140–01–P

DEPARTMENT OF HEALTH AND HUMAN SERVICES

National Institutes of Health

Eunice Kennedy Shriver National Institute of Child Health & Human Development; 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 grant applications and the discussions could disclose confidential trade secrets or commercial property such as patentable material, and personal information concerning individuals associated with the grant applications, the disclosure of which would constitute a clearly unwarranted invasion of personal privacy.

Name of Committee: National Institute of Child Health and Human Development Special Emphasis Panel EARDA.

Date: October 3, 2008.

Time: 9 a.m. to 3 p.m.

Agenda: To review and evaluate grant applications.

Place: National Institutes of Health, 6100 Executive Boulevard, 4B01 CRMC, Rockville, MD 20852.

Contact Person: Michele C. Hindi-Alexander, PhD, Division of Scientific Review, National Institutes of Health, Eunice Kennedy Shriver National Institute for Child Health & Development, 1600 Executive Boulevard, 5B01, Bethesda, MD 20812–7510, (301) 435–8382, *hindialm@mail.nih.gov.*

(Catalogue of Federal Domestic Assistance Program Nos. 93.864, Population Research; 93.865, Research for Mothers and Children; 93.929, Center for Medical Rehabilitation Research; 93.209, Contraception and Infertility Loan Repayment Program, National Institutes of Health, HHS)

Dated: August 28, 2008.

Jennifer Spaeth,

Director, Office of Federal Advisory Committee Policy.

[FR Doc. E8–20644 Filed 9–5–08; 8:45 am]

BILLING CODE 4140–01–P

DEPARTMENT OF HEALTH AND HUMAN SERVICES

National Institutes of Health

Eunice Kennedy Shriver National Institute of Child Health & Human Development; 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 grant applications and the discussions could disclose confidential trade secrets or commercial property such as patentable material, and personal information concerning individuals associated with the grant applications, the disclosure of which would constitute a clearly unwarranted invasion of personal privacy.

Name of Committee: National Institute of Child Health and Human Development Special Emphasis Panel; The Role of Human Milk in Infant Nutrition and Health.

Date: October 7, 2008.

Time: 2 a.m. to 5 p.m.

Agenda: To review and evaluate grant applications.

Place: National Institutes of Health, 6100 Executive Boulevard, 5B01, Rockville, MD 20852 (Telephone Conference Call).

Contact Person: Rita Anand, PhD, Scientific Review Administrator, Division of Scientific Review, National Institute of Child Health and Human Development, NIH, 6100 Executive Blvd, Room 5B01, Bethesda, MD 20892, (301) 496–1487, *anandr@mail.nih.gov.*

(Catalogue of Federal Domestic Assistance Program Nos. 93.864, Population Research; 93.865, Research for Mothers and Children; 93.929, Center for Medical Rehabilitation Research; 93.209, Contraception and Infertility Loan Repayment Program, National Institutes of Health, HHS)

Dated: August 28, 2008.

Jennifer Spaeth,

Director, Office of Federal Advisory Committee Policy.

[FR Doc. E8–20646 Filed 9–5–08; 8:45 am]

BILLING CODE 4140–01–P