whether the information will have practical utility; (2) evaluate the accuracy of the agency's estimate of the burden of the proposed collection of information, including the validity of the methodology and assumptions used; (3) enhance the quality, utility, and clarity of the information to be collected; and (4) minimize the burden of the collection of information on those who are to respond, including the use of appropriate automated, electronic, mechanical, or other technological collection techniques or other forms of information technology.

Direct Comments to OMB: Written comments and/or suggestions regarding the item(s) contained in this notice, especially regarding the estimated public burden and associated response time, should be directed to the: Office of Management and Budget, Office of Regulatory Affairs, *OIRA_submission@omb.eop.gov* or by fax to 202–395–6974, Attention: Desk Officer for NIH. To request more information on the proposed project or to obtain a copy of the data collection plans and instruments, contact: Ms. Kimberly Allen, NIGMS, NIH, Natcher Building, Room 2AN–18H, 45 Center Drive, MSC 6200, Bethesda, MD 20892–6200, or call non-toll-free number 301–594–2755 or e-mail your request, including your address to *allenki@nigms.nih.gov*.

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.

Dated: July 19, 2010.

Sally Lee,

Executive Officer, NIGMS, National Institute of General Medical Sciences, National Institutes of Health.

[FR Doc. 2010–18509 Filed 7–27–10; 8:45 am]

BILLING CODE 4140–01–P

DEPARTMENT OF HEALTH AND HUMAN SERVICES

Food and Drug Administration

[Docket No. FDA–2009–N–0495]

Draft Guidance for Industry and Food and Drug Administration Staff; Medical Devices; Neurological and Physical Medicine Device Guidance Document; Reopening of Comment Period

AGENCY: Food and Drug Administration, HHS.

ACTION: Notice; reopening of comment period.

SUMMARY: The Food and Drug Administration (FDA) is reopening until September 7, 2010, the comment period for the notice that appeared in the **Federal Register** of April 5, 2010 (75 FR 17143). In the notice, FDA requested comments on draft guidance documents for 11 neurological and physical medicine devices. FDA is reopening the comment period to allow further comment and to receive any new information.

DATES: Submit either electronic or written comments by September 7, 2010.

ADDRESSES: Submit electronic comments to *http://www.regulations.gov*. Submit written comments to the Division of Dockets Management (HFA–305), Food and Drug Administration, 5630 Fishers Lane, rm. 1061, Rockville, MD 20852.

FOR FURTHER INFORMATION CONTACT: Robert J. DeLuca, Center for Devices and Radiological Health, Food and Drug Administration, 10903 New Hampshire Ave., Bldg. 66, rm. G214, Silver Spring, MD 20993–0002, e-mail: *Robert.DeLuca@fda.hhs.gov*, 301–796–6630.

SUPPLEMENTARY INFORMATION:

I. Background

In the **Federal Register** of April 5, 2010 (75 FR 17093), FDA published a notice announcing the availability of draft special controls guidance documents for 11 neurological and physical medicine devices. Interested persons were originally given until July 6, 2010, to comment on the draft guidance documents. The agency expressed specific interest in comments on the types of claims appropriate for devices included within the 11 classifications and, for devices that remain subject to premarket review, the data sponsors should submit to support those claims.

II. Request for Comments

Following publication of the April 5, 2010, notice, FDA received requests to allow interested persons additional time to comment. The requests asserted that the 90-day time period was insufficient to respond fully to FDA's specific requests for comments and to allow potential respondents to thoroughly evaluate and address pertinent issues. The agency has considered the requests and is reopening the comment period until September 7, 2010. The agency believes the additional comment period allows adequate time for interested persons to submit comments without significantly delaying rulemaking on these important issues.

III. How to Submit Comments

Interested persons may submit to the Division of Dockets Management (see **ADDRESSES**) either electronic or written comments regarding this document. It is only necessary to send one set of comments. It is no longer necessary to send two copies of mailed comments. Identify comments with the docket number found in brackets in the heading of this document. Received comments may be seen in the Division of Dockets Management between 9 a.m. and 4 p.m., Monday through Friday.

Dated: July 22, 2010.

David Dorsey,

Acting Deputy Commissioner for Policy, Planning and Budget.

[FR Doc. 2010–18406 Filed 7–27–10; 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, 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.

Software System With Applications in Clinical Prognosis, Personalized Medicine and Clinical Research

Description of Invention: Available for licensing is software that can provide prognostic information for different diseases and in particular for cancer. The software can determine whether a particular genotype has a significant association with survival time for an

individual receiving treatment. For example, it can determine if a specific genetic pattern is associated with an increased or decreased time to recurrence of a particular type of cancer for patients on a given treatment regimen.

Applications:

• Applications in clinical research:

—Studying relationship between genotypes and survival times.

—Evaluation of treatment regimens.

• Applications in drug discovery programs.

• Clinical prognosis.

• Personalized medicine.

Development Status:

• The invention has been fully developed.

• The software will be readily available in executable form if so requested.

Inventor: Brian T. Luke (SAIC/NCI).

Patent Status: HHS Reference No. E–182–2010/0—Software. Patent protection is not being pursued for this technology.

Licensing Status: Available for licensing.

Licensing Contacts:

• Uri Reichman, PhD, MBA; 301–435–4616; *UR7a@nih.gov.*

• Michael Shmilovich, Esq.; 301–435–5019; *shmilovm@mail.nih.gov.*

Collaborative Research Opportunity: The NCI is seeking statements of capability or interest from parties interested in collaborative research to further develop, evaluate, or commercialize this technology. Please contact John Hewes, PhD, at 301–435–3121 or *hewesj@mail.nih.gov* for more information.

Software for Accurate Segmentation of Cell Nuclei in Breast Tissue

Description of Invention: Automatic segmentation of cell nuclei is critical in several high-throughput cytometry and pathology applications (1), such as spatial analysis of genetic loci by fluorescence *in situ* hybridization ("FISH"), whereas manual segmentation is laborious (2). Current automated segmentation methods have varying performance in the presence of distortions introduced during sample preparation, non-uniform illumination, clustering of the individual objects of interest (cells or cell nuclei), and seldom assess boundary accuracy.

Researchers at the National Cancer Institute-Frederick, NIH, have developed an automatic algorithm to segment cell nuclei (3) and FISH signals from two-dimensional images of breast tissue. This automated system integrates a series of advanced image processing methods to overcome the delays inherent to current manual methods for segmenting (delineating) individual cell nuclei in tissue samples. The system automatically selects a subset of nuclei that with high likelihood are accurately segmented. This system has been validated using both simulated and actual datasets that have been accurately analyzed by manual methods. The system generalizes to independent analysis of many spatial parameters useful for studying spatial gene positioning in interphase nuclei, and potentially has a wide range of diagnostic pathology, cytological and high throughput screening applications.

Applications:

• Investigations on genomic organization (nuclear architecture and non-random gene positioning) in the individual nuclei in tissues.

• Other pathology and cytological and high throughput screening applications requiring precise, quantitative analysis of a subset of cell nuclei in the sample.

Advantages:

• Automatic.

• Efficient, robust and effective in extracting individual nuclei with FISH labels.

• Facilitates reproducible and unbiased spatial analysis of DNA sequences in interphase nuclei.

Development Status:

• Early stage.

• Negotiations are underway with several companies to scale up development of the system and to undertake pre-clinical validation for gene positioning in the nuclei of breast sections as a possible early-stage diagnostic or prognostic test for cancer.

Inventors: Kaustav Nandy *et al.* (NCI).

Related Publications:

1. Gudla PR, Nandy K, Collins J, Meaburn KJ, Misteli T, Lockett SJ. A high-throughput system for segmenting nuclei using multiscale techniques. Cytometry A. 2008 May;73(5):451–466. [PubMed: 18338778].

2. Meaburn KJ, Gulda PR, Khan S, Lockett SJ, Misteli T. Disease-specific gene repositioning in breast cancer. J Cell Biol. 2009 Dec 14;187(6):801–812. [PubMed: 19995938].

3. Nandy K, Gudla PR, Meaburn KJ, Misteli T, Lockett SJ. Automatic nuclei segmentation and spatial FISH analysis for cancer detection. Conf Proc IEEE Eng Med Biol Soc 2009;2009:6718–6721. [PubMed: 19963931].

Patent Status: HHS Reference No. E–106–2010/0—Research Tool. Patent protection is not being pursued for this technology.

Licensing Status: Available for licensing.

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

Collaborative Research Opportunity: The inventers, working for the Office of the Director, National Cancer Institute, are seeking statements of capability or interest from parties interested in collaborative research (using the Cooperative Research and Development Agreement (CRADA) or Material Transfer Agreement (MTA)) to further develop, evaluate, or commercialize the software for accurate segmentation of cell nuclei and FISH signals in tissue sections. Collaborators working in the field of quantitative and automated pathology may be interested. Please contact John Hewes, PhD, at 301–435–3121 or *hewesj@mail.nih.gov* for more information.

Use of Cucurbitacins and Withanolides for the Treatment of Cancer

Description of Invention: Certain members of the cucurbitacin and Withanolide family have been identified that can sensitize some tumor cell lines to cell death (apoptosis) on subsequent exposure of the cells to pro-apoptotic receptor agonists (PARAS) of the TRAIL "death receptors". These PARAS include TRAIL itself, and agonist antibodies to two of its receptors death receptor-4 (DR4 or TRAIL–R1) and death receptor 5 (DR5, TRAIL–R2).

The protein TRAIL has a very interesting characteristic that it can preferentially cause death of cancer cells whereas normal non-transformed cells are unaffected. Thus use of TRAIL or agonist antibodies to its so-called "death receptors" has been a current focus in cancer therapy.

Applications:

• Use of the compounds with known TRAIL or agonist antibodies such as Mapatumumab (currently being developed by Human Genome Sciences).

• Use of the compounds in combination with immunotherapeutic approaches for the treatment of cancer.

Advantages: CUCURBITACINS AND WITHANOLIDES can be successfully developed in combination with known TRAIL agonist have the potential of new cancer combination therapies without major toxicities.

Development Status: In vivo studies are ongoing.

Inventors: Thomas J. Sayers *et al.* (NCI).

Publication: NL Booth *et al.* A cell-based high-throughput screen to identify synergistic TRAIL sensitizers. Cancer Immunol Immunother. 2009 Aug;58(8):1229–1244. [PubMed: 19089423].

Patent Status: U.S. Provisional Application No. 61/287,139 filed 16 Dec 2009 (HHS Reference No. E–050–2010/0–US–01).

Licensing Status: Available for licensing.

Licensing Contact: Sabarni Chatterjee, PhD; 301–435–5587; *chatterjeesa@mail.nih.gov.*

Collaborative Research Opportunity: The Center for Cancer Research, Laboratory of Experimental Immunology, Cancer Inflammation Program, is seeking statements of capability or interest from parties interested in collaborative research to further develop, evaluate, or commercialize the use of certain cucurbatacins or withanolides in combination with pro-apoptotic agonists of TRAIL death receptors for cancer therapy. Please contact John Hewes, PhD, at 301–435–3121 or *hewesj@mail.nih.gov* for more information.

Nitroxyl (HNO) Releasing Compounds and Uses Thereof in Treating Diseases

Description of Invention: This technology discloses HNO releasing compounds and methods of treating various diseases with such compounds. HNO has recently emerged as a prospective pharmacological agent. Studies of the chemistry of HNO have led to an understanding that HNO is vastly different from nitric oxide (NO), the one-electron oxidation product of HNO. HNO displays unique cardiovascular properties and has been shown to have positive effects in failing hearts without changing heart rate. HNO has also been shown to have beneficial effects in ischemia reperfusion injury. In addition to the cardiovascular effects observed, HNO has shown initial promise in the realm of cancer therapy. HNO has been demonstrated to inhibit a key glycolytic enzyme. Due to the Warburg effect, inhibiting glycolysis is an attractive target for inhibiting tumor proliferation. HNO has recently been shown to inhibit tumor proliferation in mouse xenografts. Additionally, HNO inhibits tumor angiogenesis and induces cancer cell apoptosis.

Applications:

• Potential treatment for cardiovascular disease, ischemia, and cancer.

• Tool to probe the role of HNO in normal physiology and disease states.

Inventors: Larry K. Keefer (NCI).

Related Publications:

1. CH Switzer, *et al.* The emergence of nitroxyl (HNO) as a pharmacological agent. Biochim Biophys Acta 2009 Jul;1787(7):835–840. [PubMed: 19426703].

2. LK Keefer. Nitric oxide (NO)- and nitroxyl (HNO)-generating diazeniumdiolates (NONOates): emerging commercial opportunities. Curr Top Med Chem. 2005;5(7):625–636. [PubMed: 16101424].

Patent Status: U.S. Provisional Application No. 61/315,604 filed 19 Mar 2010 (HHS Reference No. E–019–2010/0–US–01).

Licensing Status: Available for licensing.

Licensing Contact: Steve Standley, PhD; 301–435–4074; *sstand@od.nih.gov.*

Collaborative Research Opportunity: The Center for Cancer Research, Laboratory of Comparative Carcinogenesis, is seeking statements of capability or interest from parties interested in collaborative research to further develop, evaluate, or commercialize agents that generate HNO in physiological media for therapeutic benefit. Please contact John Hewes, PhD, at 301–435–3121 or hewesj@mail.nih.gov for more information.

Prolonging Survival in Melanoma Patients: Early Stage Diagnosis and Treatment by Detecting and Inhibiting NUAK2 Overexpression

Description of Invention: Melanoma accounts for only 4% of skin cancers, but is responsible for over 75% of skin cancer deaths worldwide. There are few treatment options available for melanoma and all current options show limited effectiveness. Melanoma is most treatable in its early stages, but most cases are not identified until the disease has progressed to the point where treatment is less effective. As normal melanocytes transform into melanoma tumor cells and metastasize, many changes occur in their gene expression patterns. Identifying genes whose expression levels impact melanoma patient survival is a key factor in developing better early detection tests and more effective treatment modalities for the disease.

NUAK2 is a stress-activated kinase and a member of the SNF–1/AMPK kinase family, a conserved family of serine/threonine kinases ubiquitous to all eukaryotes. This enzyme is normally involved in helping cells cope with glucose starvation, promoting cell-cell detachment for motility, and protecting cells from CD95-mediated apoptosis. SNF–1/AMPK kinases, such as NUAK2, also regulate cell cycle machinery by influencing the function of cyclin-dependent kinases (CDKs), such as CDK2. When deregulated, SNF–1/AMPK family members are known to contribute to cancer development and tumor progression in various cancers.

Scientists at the National Institutes of Health (NIH) have identified the *NUAK2* gene (also known as *SNARK*) as a factor to predict the clinical outcome for melanoma patients. *NUAK2* was selected as a gene of interest through extensive analysis of over 120 primary melanomas using a microarray-based comparative genomic hybridization approach which showed that genetic aberrations in *NUAK2* correlated with disease. The most prominent discovery was that gain at the *NUAK2* locus and deletion at the *PTEN* locus strongly correlated with more severe acral melanoma. Overexpression of phospho-Akt (p-Akt), caused by the *PTEN* deletion, combined with the overexpression of *NUAK2* were found to be associated with rapid disease progression, poor patient survival, and increased tumor thickness, especially in acral melanoma models. The scientists are developing diagnostic tests for *NUAK2* to better detect melanomas at an early stage when the disease is most treatable. They are also developing therapeutic small hairpin RNAs (shRNAs) to inhibit *NUAK2* gene expression and thereby reduce melanoma tumor thickness and prevent aggressive disease progression. The shRNAs utilized to silence these target genes are incorporated into lentiviral vectors, which have the potential to be delivered into humans. These scientists also observed that *NUAK2* overexpression correlated with increased expression of various CDKs. So, they are testing the effectiveness of CDK inhibitors in targeting melanomas that specifically exhibit genetic aberrations in *NUAK2* and *PTEN* leading to *NUAK2* and p-Akt overexpression. These new potential diagnostics and therapeutics centered on *NUAK2* could provide important pharmaceutical tools to detect and treat melanoma at various stages of disease.

Applications:

• Diagnostic tools and kits to identify melanoma at an early stage of disease where treatments are more effective and the mortality rate is reduced. Diagnostic tests for NUAK2 expression may be most useful in detecting acral melanoma, which is one of the most prominent forms of melanoma in Hispanic, Asian, and African-American populations.

• Therapeutic nucleic acids to inhibit melanoma disease progression by targeting specific genes important in poor clinical outcomes, such as *NUAK2* and *PTEN.*

Advantages:

• Genetic aberrations in the *NUAK2* and *PTEN* genes show a high correlation with poor clinical outcomes in

melanoma patients. Diagnostic tests specifically directed at *NUAK2* are anticipated to be highly predictive of the aggression level and course of disease in individual patients. Gaining information about melanoma before late-stage symptoms are observed should give clinicians more opportunity to treat patients before the cancer metastasizes out of control.

• Few therapies exist for melanoma and the treatments utilized by clinicians are prone to toxic side effects. Targeted therapies, such as shRNAs directed against *NUAK2* could combine more effective inhibition of melanoma with fewer harsh side effects.

Development Status: This technology is in a preclinical stage of development.

Market: There remains a long-felt public health need to develop new therapeutics and diagnostics for treating melanoma. Melanoma is the most serious type of skin cancer, accounting the majority of skin cancer deaths, and the percentage of people who develop melanoma has more than doubled in the past 30 years. With the increase in Hispanic and Asian populations in the United States, the incidence of acral melanoma has risen to become a major public health problem as it accounts for between 30%–70% of melanoma cases in dark-skinned individuals. In the United States alone in 2009, it is estimated that 68,720 new cases of melanoma were diagnosed and 8,650 people were expected to die of the disease. In 2005, the American Academy of Dermatology and the Society for Investigative Dermatology released a comprehensive study that quantified the estimated total direct cost associated with the treatment of melanoma in 2004 at $291 million in the United States. Currently, there are more than 200 therapeutics in active development to target melanoma—from early pre-clinical to marketed drugs. Clearly, a sizable portion of the melanoma diagnostic and therapeutic markets is available, since no one course of treatment is effective for all patients and very few diagnostic tools exist to identify melanoma at early stages.

Inventors: Vincent J. Hearing (NCI) and Takeshi Namiki (formerly NCI).

Publications:

1. T Namiki, *et al.* Genomic alterations in primary cutaneous melanomas detected by metaphase comparative genomic hybridization with laser capture or manual microdissection: 6p gains may predict poor outcome. Cancer Genet Cytogenet. 2005 Feb;157(1):1–11. [PubMed: 15676140].

2. JH Kim, *et al.* SNARK, a novel downstream molecule of EBV latent membrane protein 1, is associated with resistance to cancer cell death. Leuk Lymphoma. 2008 Jul;49(7):1392–1398. [PubMed: 18452098].

Patent Status: U.S. Provisional Application No. 61/321,136 filed 05 April 2010 (HHS Reference No. E–281–2009/0–US–01).

Licensing Status: Available for licensing.

Licensing Contact: Samuel E. Bish, PhD; 301–435–5282; *bishse@mail.nih.gov.*

Collaborative Research Opportunity: The Center for Cancer Research, Laboratory of Cell Biology, is seeking statements of capability or interest from parties interested in collaborative research to further develop, evaluate, or commercialize Prolonging Survival in Melanoma Patients. Please contact John Hewes, PhD, at 301–435–3121 or *hewesj@mail.nih.gov* for more information.

Immortalized Human Bronchial Epithelial Cell Line

Description of Invention: Normal cells can be cultured *in vitro* for a limited period of time before they exhibit a "crisis" or senescence, wherein they display abnormal cell morphology and significant reduction or cessation of cell proliferation. Investigators at the National Cancer Institute developed immortalized cell line by isolating bronchial epithelial cells from non-cancerous individuals and subsequent infection with an adenovirus 12–SV40 virus hybrid. Unlike normal cells, the immortalized cells be cultured continuously *in vitro* in suitable medium and retain features of normal human bronchial epithelial cells, including the absence of invasive behavior *in vitro* or *in vivo.* These cells can also be transfected with oncogenes and used as a model for multistage carcinogenesis, or employed to assay a biological or chemical agent's ability to induce differentiation and carcinogenesis as well as test potential chemotherapeutic agents.

Applications:

• Model to study multistage bronchial carcinogenesis.

• Identification of potential chemotherapeutic drugs.

• Identification of carcinogenic agents.

Advantages: Immortalized cells that retain normal human bronchial characteristics.

Market:

• Global cancer market is worth more than eight percent of total global pharmaceutical sales.

• Cancer industry is predicted to expand to $85.3 billion by 2010.

Inventors: Curtis C. Harris (NCI) *et al.*

Relevant Publication: RR Reddel *et al.* Transformation of human bronchial epithelial cells by infection with SV40 or adenovirus-12 SV40 hybrid virus, or transfection via stronium phosphate coprecipitation with a plasmid containing SV40 early region genes. Cancer Res. 1988 Apr 1;48(7):1904–1909. [PubMed: 2450641].

Patent Status: HHS Reference No. E–287–1987/0—Research Material. Patent protection is not being pursued for this technology.

Licensing Status: Available for licensing.

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

Collaborative Research Opportunity: The Center for Cancer Research, Laboratory of Human Carcinogenesis, is seeking statements of capability or interest from parties interested in collaborative research to further develop, evaluate, or commercialize Immortalized Human Bronchial Epithelial Cell Line. Please contact John Hewes, PhD, at 301–435–3121 or *hewesj@mail.nih.gov* for more information.

Dated: July 22, 2010.

Richard U. Rodriguez,

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

[FR Doc. 2010–18487 Filed 7–27–10; 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