

training programs, identify resources that will address the gaps and further strengthen the outcomes from these efforts, examine testimony from the experts in the field, and offer recommendations for improvement of these training programs to the Secretary and the Congress.

*Agenda:* The ACICBL agenda includes an overview of the Committee's general business activities, an orientation for the eight newly appointed members, presentations by and dialogue with experts, and discussion sessions specific to the development of recommendations to be addressed in the 11th Annual ACICBL Report. Agenda items are subject to change as dictated by the priorities of the Committee.

**SUPPLEMENTARY INFORMATION:** Requests to make oral comments or to provide written comments to the ACICBL should be sent to Dr. Joan Weiss, Designated Federal Official at the contact information below. Individuals who plan to attend the meeting and need special assistance should notify Dr. Weiss at least 10 days prior to the meeting, using the address and phone number below. Members of the public will have the opportunity to provide comments at the meeting.

**FOR FURTHER INFORMATION CONTACT:** Anyone requesting additional details should contact Dr. Joan Weiss, Designated Federal Official within the Bureau of Health Professions, Health Resources and Services Administration. Dr. Weiss may be reached by one of the three following methods: (1) Via written request to: Dr. Joan Weiss, Designated Federal Official, Bureau of Health Professions, Health Resources and Services Administration, Parklawn Building, Room 9-36, 5600 Fishers Lane, Rockville, Maryland 20857; (2) via telephone at (301) 443-6950; or (3) via e-mail at [jweiss@hrsa.gov](mailto:jweiss@hrsa.gov). In the absence of Dr. Weiss, CAPT Norma J. Hatot, Senior Nurse Consultant, may be contacted via telephone at (301) 443-2681 or by e-mail at [nhatot@hrsa.gov](mailto:nhatot@hrsa.gov).

Dated: December 21, 2010.

**Robert Hendricks,**

*Director, Division of Policy and Information Coordination.*

[FR Doc. 2010-32560 Filed 12-27-10; 8:45 am]

**BILLING CODE 4165-15-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.

**Hesperetin Therapy for Metabolic Syndrome and Insulin Resistance**

*Description of Technology:* Hesperidin is a flavonoid compound found in citrus fruits. Large epidemiological studies have linked increased consumption of flavonoid-rich foods, such as citrus, with reduced cardiovascular morbidity and mortality. Investigators from the National Center for Complementary and Alternative Medicine have demonstrated that administration of oral hesperidin to patients with metabolic syndrome attenuates biomarkers of inflammation and improves blood vessel relaxation, lipid cholesterol profiles, and insulin sensitivity when compared to controls. Thus, hesperidin and its active aglycone form, hesperetin, may be effective agents for the treatment of diabetes, obesity, metabolic syndrome, dyslipidemias, and their cardiovascular complications including hypertension, atherosclerosis, coronary heart disease, and stroke. This technology discloses methods for using a hesperetin composition to treat metabolic syndrome and insulin resistance.

*Applications:* Therapeutics for metabolic syndrome and insulin resistance.

*Development Status:* Clinical trial data available.

*Inventors:* Michael J. Quon and Ranganath Muniyappa (NCCAM).

*Publications:* Manuscript in preparation.

*Patent Status:* U.S. Provisional Application No. 61/369,229 filed 30 July 2010 (HHS Reference No. E-148-2010/0-US-01).

*Licensing Status:* Available for licensing.

*Licensing Contact:* Tara L. Kirby, Ph.D.; 301-435-4426; [kirbyt@mail.nih.gov](mailto:kirbyt@mail.nih.gov).

**Substituted Triazine and Purine Compounds for the Treatment of Chagas Disease and African Trypanosomiasis**

*Description of Technology:* Parasitic protozoa are responsible for a wide variety of infections in both humans and animals. Trypanosomiasis poses health risks to millions of people across multiple countries in Africa and North and South America. Visitors to these regions, such as business travelers and tourists, are also at risk for contracting parasitic diseases. There are two types of African trypanosomiasis, also known as sleeping sickness. One type is caused by the parasite *Trypanosoma brucei gambiense*, and the other is caused by the parasite *Trypanosoma brucei rhodesiensi*. If left untreated, African sleeping sickness results in death. Chagas disease, caused by *Trypanosoma cruzi* (*T. cruzi*), affects millions of people in Mexico and South and Central America. Untreated, Chagas disease causes decreased life expectancy and can also result in death.

The subject invention covers novel triazine and purine compounds that are inhibitors of key proteases (cruzain and Rhodesian) of the parasites *Trypanosoma brucei rhodesiensi* and *Trypanosoma cruzi*, respectively.

*Applications:* Prophylactic and therapeutic treatment of African trypanosomiasis and Chagas disease.

*Advantages*

- Novel compounds against the cysteine proteases, cruzain and rhodesain.
- Compounds possess low nanomolar inhibitory potential against cruzain and rhodesain.

*Development Status:* *In vitro* and *in vivo* data are available upon request and upon execution of an appropriate confidentiality agreement.

*Inventors:* Craig J. Thomas *et al.* (NHGRI).

*Related Publication:* BT Mott *et al.* Identification and optimization of inhibitors of Trypanosomal cysteine

proteases: cruzain, rhodesain, and TbCatB. *J Med Chem.* 2010 Jan 14;53(1):52–60. [PubMed: 19908842]

*Patent Status:* PCT Application No. PCT/US2009/063078 filed 03 Nov 2009, which published as WO 2010/059418 on 27 May 2010 (HHS Reference No. E-267–2008/0–PCT–02)

*Licensing Status:* Available for licensing.

*Licensing Contact:* Kevin W. Chang, Ph.D.; 301–435–5018; [changke@mail.nih.gov](mailto:changke@mail.nih.gov).

*Collaborative Research Opportunity:* The NIH Chemical Genomics Center (NCGC) is seeking statements of capability or interest from parties interested in collaborative research to further develop, evaluate, or commercialize appropriate lead compounds described in the patent application. Please contact Dr. Craig J. Thomas ([craigj@nhgri.nih.gov](mailto:craigj@nhgri.nih.gov)) or Claire Driscoll ([cdriscoll@mail.nih.gov](mailto:cdriscoll@mail.nih.gov)), Director of the NHGRI Technology Transfer Office, for more information.

**A Novel, Inhibitory Platelet Surface Protein (TREM Like Transcript, TLT–1): New Target for the Treatment of Cancer, Infectious Diseases, Cardiac Diseases, and Platelet-Associated Disorders**

*Description of Technology:* Triggering Receptors in Myeloid Cells (TREM) recently were discovered to modulate innate and adaptive immunity. Specifically, TREM1 amplifies the response to sepsis in innate immunity by activating neutrophils and other leukocytes; and TREM2 potentiates dendritic cell maturation in adaptive immunity.

This invention describes a novel, inhibitory platelet surface protein known as TREM like Transcript (TLT–1). TLT–1 is the first inhibitory receptor discovered to reside within the TREM gene locus. Structurally, TLT–1 also possesses inhibitory domains that indicate this regulatory function. TLT–1 is highly expressed in peripheral blood platelets and may modulate many other types of myeloid cells. Additionally, the invention describes specific, human, single chain antibodies (scFvs) that recognize TLT–1.

*Applications*

- This discovery implies the receptor has an important regulatory role in both innate and adaptive immunity.
- TLT–1 is a potential therapeutic target for thrombosis and other platelet-associated disorders, as well as immune disorders, cancer, septic shock, infectious disease, stroke, heart disease, myocardial infarction, vascular disorders.

- Detection of soluble TLT–1 in patient plasma suggests the protein is a marker of ongoing coagulopathies.

- Defective platelet aggregation in TLT–1 null mice confirms a role for the protein in regulation of thrombosis associated with inflammation.

*Advantages*

- *In vitro* proof of concept data available—Three of the anti-TLT–1 scFvs inhibit thrombin-induced aggregation of human platelets in a dose-dependent manner.

- Complete human origin of these antibodies suggests negligible immunogenicity and minimizes the problem of adverse immune responses in human therapy.

- Target validation is complete. TLT–1 null mice demonstrate defects in platelet aggregation with no gross bleeding defect.

*Development Status:* *In vitro* experiments completed. Target validation with null mice completed. *In vivo* animal studies with scFv are currently ongoing.

*Inventors:* Toshiyuki Mori *et al.* (NCI)

*Related Publication:* Giomarelli B, Washington VA, Chisholm MM, Quigley L, McMahon JB, Mori T, McVicar DW. Inhibition of thrombin-induced platelet aggregation using human single-chain Fv antibodies specific for TREM-like transcript-1. *Thromb Haemost.* 2007 Jun;97(6):955–963. [PubMed: 17549298]

*Patent Status:* U.S. Patent No. 7,553,936 issued on 30 Jun 2009 (HHS Reference No. E-177–2006/0–US–01)

*Licensing Status:* Available for licensing.

*Licensing Contact:* Betty B. Tong, PhD; 301–594–6565; [tongb@mail.nih.gov](mailto:tongb@mail.nih.gov).

*Collaborative Research Opportunity:* The National Cancer Institute's Molecular Targets Development Program is seeking statements of capability or interest from parties interested in collaborative research to further develop, evaluate, or commercialize antibodies that react specifically with TLT–1. Please contact John D. Hewes, PhD at 301–435–3121 or [hewesj@mail.nih.gov](mailto:hewesj@mail.nih.gov) for more information.

Dated: December 21, 2010.

**Richard U. Rodriguez,**

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

[FR Doc. 2010–32629 Filed 12–27–10; 8:45 am]

**BILLING CODE 4140–01–P**

**DEPARTMENT OF HEALTH AND HUMAN SERVICES**

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**Engineered Biological Pacemakers**

*Description of Technology:* A common symptom of many heart diseases is an abnormal heart rhythm or arrhythmia. While effectively improving the lives of many patients, implantable pacemakers have significant limitations such as limited power sources, risk of infections, potential for interference from other devices, and absence of autonomic rate modulation.

The technology consists of biological pacemakers engineered to generate normal heart rhythm. The biological pacemakers include cardiac cells or cardiac-like cells derived from embryonic stem cells or mesenchymal stem cells. The biological pacemakers naturally integrate into the heart. Their generation of rhythmic electric impulses involves coupling factors, such as cAMP-dependent PKA and Ca<sup>2+</sup>-dependent CaMK II, which are regulatory proteins capable of modulating/enhancing interactions (*i.e.* coupling) of the sarcoplasmic reticulum-based, intracellular Ca<sup>2+</sup> clock and the surface membrane voltage clock, thereby converting irregularly or rarely spontaneously active cells into pacemakers generating rhythmic excitations.