

participants, and an indication of the approximate time requested to make their presentation on or before October 7, 2011. Time allotted for each presentation may be limited. If the number of registrants requesting to speak is greater than can be reasonably accommodated during the scheduled open public hearing session, FDA may conduct a lottery to determine the speakers for the scheduled open public hearing session. The contact person will notify interested persons regarding their request to speak by October 11, 2011.

Persons attending FDA's advisory committee meetings are advised that the Agency is not responsible for providing access to electrical outlets.

FDA welcomes the attendance of the public at its advisory committee meetings and will make every effort to accommodate persons with physical disabilities or special needs. If you require special accommodations due to a disability, please contact Caleb Briggs at least 7 days in advance of the meeting.

FDA is committed to the orderly conduct of its advisory committee meetings. Please visit our Web site at <http://www.fda.gov/AdvisoryCommittees/AboutAdvisoryCommittees/ucm111462.htm> for procedures on public conduct during advisory committee meetings.

Notice of this meeting is given under the Federal Advisory Committee Act (5 U.S.C. app. 2).

Dated: September 30, 2011.  
**Leslie Kux,**  
*Acting Assistant Commissioner for Policy.*  
 [FR Doc. 2011-25684 Filed 10-4-11; 8:45 am]  
**BILLING CODE 4160-01-P**

**DEPARTMENT OF HEALTH AND HUMAN SERVICES**

**National Institutes of Health**

**Proposed Collection; Comment Request; STAR METRICS (Science and Technology for America's Reinvestment: Measuring the Effects of Research on Innovation, Competitiveness and Science)**

**SUMMARY:** In compliance with the requirement of Section 3506(c)(2)(A) of the Paperwork Reduction Act of 1995, for opportunity for public comment on proposed data collection projects, the Office of the Director of the National Institutes of Health (NIH) will publish periodic summaries of proposed projects to be submitted to the Office of Management and Budget (OMB) for review and approval.

*Proposed Collection: Title:* STAR METRICS (Science and Technology for America's Reinvestment: Measuring the Effects of Research on Innovation,

Competitiveness and Science). *Type of Information Collection Request:* Reinstatement of OMB number 0925-0616, expiration date 01/31/2011. *Need and Use of Information Collection:* The aim of STAR METRICS is twofold. The goal of STAR METRICS is to continue to provide mechanisms that will allow participating universities and Federal agencies with a reliable and consistent means to account for the number of scientists and staff that are on research institution payrolls, supported by Federal funds. In subsequent generations of the program, it is hoped that STAR METRICS will allow for measurement of science impact on economic outcomes (such as job creation), on knowledge generation (such as citations and patents) as well as on social and health outcomes. *Frequency of Response:* Quarterly. *Affected Public:* Universities and other research institutions. *Type of Respondents:* University administrators. The annual reporting burden is as follows: *Estimated Number of Respondents:* 100. *Estimated Number of Responses per Response:* 4. *Average Burden Hours per Response:* 2.5. *Estimated Total Annual Burden Hours Requested:* 1,315. The annualized cost to respondents is estimated to be \$65,750. There are no Capital Costs to report. There are no Operating or Maintenance Costs to report.

**A.12-1 ESTIMATES ANNUAL BURDEN HOURS**

Form	Number of respondents	Frequency of response	Average time per response (in hours)	Annual hour burden
Stage 1: One time data input .....	7	1	45	315
Stage 2: Ongoing quarterly data input .....	100	4	2.5	1000
Total .....				1,315

*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 function of the agency, including whether the information will have practical utility; (2) 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) 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 those who are to respond, including the use of appropriate automated, electronic,

mechanical, or other technological collection techniques or other forms of information technology.

**FOR FURTHER INFORMATION CONTACT:** To request more information on the proposed project or to obtain a copy of the data collection plans and instruments, contact Dr. Julia Lane, e-mail: [julia.lane@nih.gov](mailto:julia.lane@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: September 27, 2011.  
**Stefano Bertuzzi,**  
*Office of the Director, Office of Science Policy Analysis, Office of Science Policy, National Institutes of Health.*  
 [FR Doc. 2011-25732 Filed 10-4-11; 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.

### Platform Technology Using Ubiquitin To Improve the Delivery and Efficacy of Cytosolic Targeted Toxins

*Description of Technology:* Targeted toxins (TT) are hybrid protein drugs consisting of ligands that bind to the surface of cancer cells and deliver polypeptide toxins that kill malignant cells by inactivating cytosolic protein synthesis and inducing cell death. A major challenge in the construction of targeted toxins is reducing the nonspecific binding of the toxin moiety to normal tissues and increasing the cytotoxicity of the treatment.

To address these issues, the NIH inventors have identified that the protein ubiquitin, a small protein in eukaryotic cells that plays a role in protein recycling, can separate the targeting moiety and the catalytic moiety of a TT in the cytosol of cells. By decoupling the two moieties, the cytotoxicity of the TT treatment can be greatly increased since the catalytic domain remains longer in the cytosol. This technology would be highly useful for all TT and immunotoxins that access the cytosol to either affect cytosolic targets or traffic to further sites of action. To validate this approach, the inventors have tested ubiquitin variants within a TT consisting of anthrax toxin lethal factor N-terminus (LF<sub>N</sub>) and Pseudomonas exotoxin A catalytic domain (PEIII). Here, they show that the intracellular release of the PEIII (catalytic moiety) is achievable and that ubiquitination of the TT controls the persistence of the TTs in the cytosol and thus controls the observed cytotoxicity.

*Potential Commercial Applications:*

- Chimeric or fusion molecules for increasing the efficacy and cytotoxicity of targeted toxins and immunotoxins.

- Methods for cytosol delivery of targeted toxins to target cells.

#### *Competitive Advantages:*

- Broadly applicable to all cytotoxic immunoconjugates.
- Increased stability and cytotoxicity of the TT without affecting the delivery or specificity of the treatment.

- Therapeutic access to the cytosol and/or trafficking to further sites of action such as the nucleus.

- Rapid cytosolic release of the catalytic moiety and degradation of the targeting moiety.

#### *Development Stage:*

- Pre-clinical

- In vitro data available

*Inventors:* Christopher Bachran (NIAID), Stephen Leppla (NIAID), Shihui Liu (NIAID), Thomas Morley

#### *Publications:*

1. Tcherniuk S, *et al.* Construction of tumor-specific toxins using ubiquitin fusion technique. *Mol Ther.* 2005 Feb;11(2):196-204. [PMID 15668131]

2. Wang F. Selective cytotoxicity to HER2-positive tumor cells by a recombinant e23sFv-TD-tBID protein containing a furin cleavage sequence. *Clin Cancer Res.* 2010 Apr 15;16(8):2284-2294. [PMID 20371697]

3. Heisler I. A cleavable adapter to reduce nonspecific cytotoxicity of recombinant immunotoxins. *Int J Cancer.* 2003 Jan 10;103(2):277-282. [PMID 12455044]

*Intellectual Property:* HHS Reference No. E-150-2011/0—U.S. Provisional Application No. 61/473,450 filed 08 April 2011

#### *Related Technologies:*

- HHS Reference No. E-293-1999—Mutated Anthrax Toxin Protective Antigen Proteins That Specifically Target Cells Containing High Amounts of Cell-Surface Metalloproteinases or Plasminogen Activator Receptors (Leppla/NIAID)

- HHS Reference No. E-070-2007—Human Cancer Therapy Using Engineered Metalloproteinase-Activated Anthrax Lethal Toxin That Target Tumor Vasculature (Leppla/NIAID)

- HHS Reference No. E-059-2004—Multimeric Protein Toxins to Target Cells Having Multiple Identifying Characteristics (Leppla/NIAID)

*Licensing Contact:* Whitney Hastings; 301-451-7337; *hastingw@mail.nih.gov*

### NOX5 Immunogenic Peptides and Monoclonal Antibodies for the Detection of Cancer and Inflammatory Responses

*Description of Technology:* The membrane-associated NADPH oxidase 5

(NOX5) protein is expressed in various fetal tissues, uterus, testis, spleen, lymph nodes and endothelial cells. In addition, the reactive oxygen species (ROS) generated by NOX5 have been shown to participate in signaling cascades regulating proliferation in several cancers and pre-cancerous conditions, such as hairy cell leukemia, melanoma, prostate cancer, and Barrett's esophagus. Further, excess ROS produced by NOX5 has been associated with coronary artery disease, inflammation, and atherosclerosis.

The present invention discloses the identification and characterization of a purified monoclonal antibody against NOX5 protein. This NOX5 antibody can detect endogenous levels of NOX5 in human cells and could aid in studies and diagnostic tests of NOX5-based redox signaling involved in cancer, cell growth and differentiation, as well as angiogenic and inflammatory responses. In addition, the NOX5 antibody may have therapeutic applications (e.g. anti-inflammatory, antiangiogenic, or antiproliferative activity) by interfering with NOX5 activation at the cell surface.

#### *Potential Commercial Applications:*

- Diagnostic for the detection of NOX5 in human cells and NOX5-based redox signaling

- Antibody can be used in ELISA, Western Blot, Immunofluorescence, Immunoprecipitation and Immunohistochemistry

- Tool to aid in the understanding of NOX5's functional significance in human physiology and pathophysiology

- Possible therapeutic for the treatment of various human diseases associated with NOX5 and/or ROS

#### *Competitive Advantages:*

- Antibody is the only mouse monoclonal commercially available to the best of our knowledge

- Antibody is highly specific in recognizing the NOX5 protein with greater efficiency and the accurate detection compared to other Nox5 antibodies

#### *Development Stage:* Pre-clinical

*Inventors:* James H. Doroshow, Krishnendu K. Roy, Smitha Antony (NCI)

#### *Publications:*

1. Kamiguti AS, *et al.* Expression and activity of NOX5 in the circulating malignant B cells of hairy cell leukemia. *J Immunol.* 2005 Dec 15;175(12):8424-8430. [PMID: 16339585]

2. Brar SS, *et al.* NOX5 NAD(P)H oxidase regulates growth and apoptosis in DU 145 prostate cancer cells. *Am J Physiol Cell Physiol.* 2003 Aug;285(2):C353-C369. [PMID: 12686516]

3. Hong J, *et al.* Bile acid reflux contributes to development of esophageal adenocarcinoma via activation of phosphatidylinositol-specific phospholipase C $\gamma$ 2 and NADPH oxidase NOX5—S. *Cancer Res.* 2010 Feb 1;70(3):1247–1255. [PMID: 20086178]

*Intellectual Property:* HHS Reference No. E–149–2011/0—U.S. Provisional Application No. 61/471,596 filed 04 April 2011

*Licensing Contact:* Whitney Hastings; 301–451–7337; [hastingsw@mail.nih.gov](mailto:hastingsw@mail.nih.gov)

#### **mGluR5 Tumor Mouse Model**

*Description of Technology:* Glutamate receptor mGluR5 has been reported to function in the brain. There were no prior reports of it being involved in melanoma. The NIH investigators have discovered that when over expressed in transgenic animals, mGluR5 induces melanoma. The establishment of an mGluR5 tumor mouse model will provide a unique opportunity to help elucidate the mechanisms underlying tumor formation, and allow the study of aggressive melanoma in animals and a screen of potential therapeutics. Such an mGluR5 tumor mouse model is established at the National Institutes of Health and is available for licensing.

*Potential Commercial Applications:*

- Drug screening for melanoma therapeutics

- Research Tool

*Competitive Advantage:* Tumor mouse model only available from the NIH lab.

*Development Stage:*

- Prototype
- Pre-clinical
- In vivo data available (animal)

*Inventors:* Katherine W. Roche and Kyu Yeong Choi (NINDS)

*Publication:* Choi KY, *et al.*

Expression of the metabotropic glutamate receptor 5 (mGluR5) induces melanoma in transgenic mice. *Proc Natl Acad Sci USA* 2011; published ahead of print September 6, 2011, doi:10.1073/pnas.1107304108.

*Intellectual Property:* HHS Reference No. E–123–2010/0—Research Tool. Patent protection is not being pursued for this technology.

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

#### **Monoclonal Antibodies to FCRL5 (CD307e/IRTA2/FcRH5) as Therapeutics and Diagnostics for B-cell Cancers**

*Description of Technology:* The Fc receptor-like (FCRL) genes (also known as CD307, IRTA, FcRH, IFGP or SPAP) encode cell membrane proteins that are believed to play roles in immunity and

B cell differentiation. Some FCRL genes have been implicated in B cell lymphomas and multiple myelomas. Data suggest that the FCRL1–5 proteins are expressed differently on malignant B cells as well as subpopulations of normal B cells. Due to this differential expression, FCRL proteins represent potential targets for the treatment of cancers of a B cell origin.

This technology relates to the development of novel monoclonal antibodies for a specific member of the FCRL protein family: FCRL5. FCRL5 is normally induced on mature B cells upon activation, but its expression is deregulated in multiple myeloma and Burkitt's lymphoma. Due to the correlation of FCRL5 overexpression and B cell malignancies, antibodies to FCRL5 may have value as a therapeutic or diagnostic tool. Specifically, the antibodies can be used as therapeutic agents by themselves or they can be attached to a cytotoxic agent such as *Pseudomonas* exotoxin A. Alternatively, the antibodies can be used to detect the deregulation of FCRL5 as a means of diagnosing B cell malignancies.

*Potential Commercial Applications:*

- Detection or diagnosis of B cell cancers using monoclonal antibodies to FCRL5

- Treatment of B cell cancers using monoclonal antibodies to FCRL5 for inducing antibody-dependent cell death

- Treatment of B cell cancers using monoclonal antibodies to FCRL5 for targeting cytotoxic agents specifically to cancer cells (e.g., immunotoxins)

*Competitive Advantages:*

- No cross-reactivity with other FCRL proteins demonstrates strong selectivity as both a therapeutic and diagnostic agent

- Targeted therapeutics such as monoclonal antibodies and immunotoxins decrease non-specific killing of healthy, essential cells, resulting in fewer side-effects and healthier patients

*Development Stage:* Pre-clinical

*Inventors:* Ira H. Pastan *et al.* (NCI)

*Publications:*

1. Ise T, *et al.* Elevation of soluble CD307 (IRTA2/FcRH5) protein in the blood and expression on malignant cells of patients with multiple myeloma, chronic lymphocytic leukemia, and mantle cell lymphoma. *Leukemia.* 2007 Jan; 21(1):169–174. [PMID 17051241]

2. Ise T, *et al.* Immunoglobulin superfamily receptor translocation associated 2 protein on lymphoma cell lines and hairy cell leukemia cells detected by novel monoclonal antibodies. *Clin Cancer Res.* 2005 Jan 1;11(1):87–96. [PMID 15671532]

*Intellectual Property:* HHS Reference No. E–287–2004/1—U.S. Patent 7,999,077 issued 16 Aug 2011

*Licensing Contact:* David A. Lambertson, Ph.D.; 301–435–4632; [lambertsond@mail.nih.gov](mailto:lambertsond@mail.nih.gov)

#### **Potent Inhibitory RNAs for Non-Surgical Treatment of Salivary Gland Cancers**

*Description of Technology:* In the U.S., approximately 40,000 cases of head and neck cancer, including salivary gland tumors, are diagnosed each year. Surgery with post-operative radiotherapy is the most common treatment for salivary gland tumors. However, complete removal is difficult due to the three-dimensional growth pattern of these tumors which impedes a surgeon's ability to determine once the tumor has been fully removed. Both surgeons and patients desire minimal surgical approaches for cosmetic reasons, as well as to preserve nerve function in the facial area. Thus a significant need exists for non-surgical approaches to treating salivary gland tumors.

Researchers at the National Cancer Institute, NIH, have discovered that mucoepidermoid (MEC) salivary gland tumors arise from a chromosomal rearrangement which generates a fusion oncogene, Mect1–Maml2, that functions to alter Notch and CREB signaling pathways. An RNAi vector has been developed that selectively suppresses the oncogene and inhibits growth of certain MEC tumor cell lines containing the oncogene by at least 90%. The RNAi vector has no effect on cells that do not express the oncogene. This ability of the RNAi vectors to block the “gain-of-function” activity of the acquired Mect1–Maml2 oncogene suggests new possibilities for the diagnosis and therapy of these cancers.

*Potential Commercial Applications:*

- Diagnosis of MEC salivary gland tumors

- Treatment of MEC salivary gland tumors

*Competitive Advantages:*

- Non-surgical
- Selective
- Potent
- Can be used in combination with other known treatments, such as radiation and chemotherapy

*Development Stage:*

- Pre-clinical
- In vitro data available

*Inventors:* Frederic Koye (formerly NCI), Takefumi Komiya (NCI)

*Publications:*

1. Tonon G, *et al.* t(11;19)(q21;p13) translocation in mucoepidermoid carcinoma creates a novel fusion

product that disrupts a Notch signaling pathway. *Nat Genet.* 2003

Feb;33(2):208–213. [PMID 12539049]

2. Martins C, *et al.* A study of MECT1–MAML2 in mucoepidermoid carcinoma and Warthin's tumor of salivary glands. *J Mol Diagn.* 2004 Aug;6(3):205–210. [PMID 15269296]

3. Coxon A, *et al.* Mect1–Maml2 fusion oncogene linked to the aberrant activation of cyclic AMP/CREB regulated genes. *Cancer Res.* 2005 Aug 15;65(16):7137–7144. [PMID 16103063]

4. Komiya T, *et al.* Sustained expression of Mect1–Maml2 is essential for tumor cell growth in salivary gland cancers carrying the t(11;19) translocation. *Oncogene.* 2006 Oct 5;25(45):6128–6132. [PMID 16652146]

5. Kaye FJ. Emerging biology of malignant salivary gland tumors offers new insights into the classification and treatment of mucoepidermoid cancer. *Clin Cancer Res.* 2006 Jul 1;12(13):3878–3881. [PMID 16818681]

6. Tirado Y, *et al.* CRT1/MAML2 fusion transcript in high grade mucoepidermoid carcinomas of salivary and thyroid glands and Warthin's tumors: implications for histogenesis and biologic behavior. *Genes Chromosomes Cancer.* 2007 Jul;46(7):708–715. [PMID 17437281]

7. Komiya T, *et al.* Enhanced activity of the CREB co-activator Crtc1 in LKB1 null lung cancer. *Oncogene.* 2010 Mar 18;29(11):1672–1680. [PMID 20010869]

*Intellectual Property:* HHS, Reference No. E–086–2003/0 —

- U.S. Patent No. 7,553,822 issued 30 June 2009

- U.S. Patent Application No. 12/493,901 filed 29 June 2009

*Licensing Contact:* Patrick McCue, Ph.D.; 301–435–5560; [mccuepat@mail.nih.gov](mailto:mccuepat@mail.nih.gov)

Dated: September 29, 2011.

**Richard U. Rodriguez,**

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

[FR Doc. 2011–25734 Filed 10–4–11; 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.

#### Humanized Monoclonal Antibodies Efficient for Neutralization of Tick-Borne Encephalitis Virus (TBEV)

*Description of Technology:* TBEV causes serious illnesses from meningitis to meningo-encephalitis, totaling 3,000 cases of hospitalization in Europe and between 5,000–10,000 cases in Russia reported every year. The Far Eastern hemorrhagic TBEV strains are associated with a mortality rate (between 1–2%), higher than other strains isolated in the Siberia or Western Europe. There is a high proportion (up to 46%) of TBEV patients with temporary or permanent neurological sequelae. The number of TBEV infections has increased steadily and TBEV cases have been reported in new areas, probably reflecting an increased spread of vector tick species. Prevention of TBEV infections has been carried out in a few countries in Europe by immunization using an inactivated TBEV vaccine. The vaccine carries a high manufacturing cost and requires a regimen of multiple doses, and for this reason, vaccination is not generally carried out. The materials disclosed are humanized monoclonal antibodies derived from TBEV-neutralizing Fab antibodies isolated from infected chimpanzees by repertoire cloning. One antibody in particular, MAb 2E6, has been demonstrated to bind to and neutralize a TBEV/dengue type 4 virus chimera (via interaction with the TBEV antigenic determinants) as well as the related Langat virus. Protection against TBEV/DEN–4 infection and Langat infection has been demonstrated using animal models of infection. The antibodies disclosed, in particular MAb 2E6, have the potential for use as prophylactic and therapeutic agents

against TBEV and Langat virus. Additionally, these antibodies may be suitable as diagnostic reagents for the detection of TBEV and/or Langat virus.

#### *Potential Commercial Applications:*

- TBEV Prophylaxis.
- TBEV Therapy.
- TBEV Diagnostics.

#### *Competitive Advantages:*

- Cost effective alternative to existing vaccine.

- Fully humanized antibody.
- Strongly neutralizing antibody.
- Efficient production methods.

#### *Development Stage:*

- Pre-clinical.
- In vitro data available.
- In vivo data available (animal).

*Inventors:* C. J. Lai, Robert Purcell, Alexander Pletnev (NIAID).

*Intellectual Property:* HHS Reference No. E–231–2011/0—Research Tool. Patent protection is not being pursued for this technology.

*Licensing Contact:* Peter Soukas; 301–435–4646; [soukasp@mail.nih.gov](mailto:soukasp@mail.nih.gov)

#### *Collaborative Research Opportunity:*

The NIAID is seeking statements of capability or interest from parties interested in collaborative research to further develop, evaluate or commercialize TBEV monoclonal antibodies. For collaboration opportunities, please contact Wade Williams at 301–827–0258.

#### Rapid Molecular Assays for Specific Detection and Quantitation of Loa Loa Microfilaremia

*Description of Technology:* The risk of fatal reactions in some infected individuals administered drug treatments for Loa loa infection, and the lack of accurate, convenient, diagnostics for this infection have thwarted efforts to eradicate the disease. Time consuming, labor intensive and training intensive microscope-based analysis of blood samples is the standard available diagnostic for Loa loa infection. This new assay technology introduces an easy to use, species-specific, highly sensitive, diagnostic that is able to be performed with minimal training. Positive test results may be indicated by an easily visualized color change and this test may be run without the need for expensive equipment such as a thermocycler. Because this test is rapid, cost efficient, labor efficient, accurate, and simple to run and read, it may be readily incorporated into portable point-of-care formats. These attributes make it ideally suited for use in locations where Loa loa infection is endemic. These advantages may lead to this technology becoming the new standard for diagnosis of Loa loa infections and a valuable tool, in control programs, to