Collaborative Research Opportunity: The NIAID, OTD, is seeking statements of capability or interest from parties interested in collaborative research to further develop, evaluate, or commercialize this ''Discovery of Novel Pharmacophores Inhibiting the Growth of Mycobacterium Tuberculosis''. Please contact Anna Amar at 301–451–3525 for more information.

A Varicella-Zoster Virus Mutant That Is Markedly Impaired for Latent Infection Available for the Development of Shingles Vaccines and Diagnostics

Description of Technology: Reactivation of latent Varicella-Zoster virus (VZV) infection is the cause of shingles, which is prominent in adults over the age of 60 and individuals who have compromised immune systems, due to HIV infection, cancer treatment and/or transplant. Shingles is a worldwide health concern that affects approximately 600,000 Americans each year. The incidence of shingles is also high in Europe, South America, and India; the latter having an estimated two million individuals affected, yearly. Recent research studies show that VZV vaccines have a significant effect on decreasing the incidence of shingles in elderly.

The current technology describes compositions, cells and methods related to the production and use of a mutant VZV and the development of vaccines against the infectious agent. Latent VZV expresses a limited repertoire of viral genes including the following six open reading frames (ORFs): 4, 21, 29, 62, 63, and 66. The present invention describes an ORF29 mutant VZV that demonstrates a weakened ability to establish latency in animal studies. The current technology provides methods for using the mutant in the development of live vaccines and diagnostic tools. A related invention is described in PCT/US05/021788 (publication number WO2006012092).

Applications: Development of vaccines and diagnostics for prevention of shingles.

Development Status: Pre-clinical studies have been performed to demonstrate the reduced latency of the ORF29 mutant VZV in animals.

Inventors: Jeffrey Cohen (NIAID) and Lesley Pesnicak (NIAID).

Patent Status: U.S. Provisional Application No. 60/857,766 filed 09 Nov 2006 (HHS Reference No. E–029–2007/0–US–01); PCT Application No. PCT/US2007/084331 filed 09 Nov 2007, which published as WO 2008/079539 on 03 Jul 2008 (HHS Reference No. E–029–2007/0–PCT–02).

Licensing Status: Available for licensing and commercial development.

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

Collaborative Research Opportunity: The NIAID Laboratory of Clinical Infectious Diseases is seeking statements of capability or interest from parties interested in collaborative research to further develop, evaluate, or commercialize vaccine strains of VZV vaccine with impaired latency. Please contact Kelly Murphy, J.D., M.S., at 301/451–3523 or *murphykt@niaid.nih.gov* for more information.

Anti-Plasmodium Compositions and Methods of Use

Description of Technology: The present invention comprises peptides/antibodies specific for the binding proteins of *Plasmodium*, a parasite responsible for malaria, hence in effect blocking the parasite's binding to the erythrocytes. Also included are methods for their use in preventing, diagnosing or treating the related infections.

Although malaria is virtually eradicated in the United States, it continues to be one of the most serious infectious diseases in the world, killing millions of people each year in the countries throughout Africa, Asia and Latin America. In fact, over 41% of the world population lives in the regions affected by malaria. *In vitro* studies using the antibodies described in the current technology showed ~80% reduction in the number of blood cells infected with *Plasmodium* parasite. Infectivity studies using peptides demonstrated that they are also specifically able to prevent binding of parasites to blood cells. The claimed antibodies and peptides can also be used for immunization of humans and animals, or for development of diagnostic kits capable of detecting the presence, localization and quantity of the *Plasmodium* parasites in tissues and cells.

Applications: Diagnostics development; Vaccines development.

Inventors: David L. Narum and Kim Lee Sim (NIAID).

Relevant Publications:

1. Sim BK, Narum DL, Liang H, Fuhrmann SR, Obaldia N 3rd, Gramzinski R, Aguiar J, Haynes JD, Moch JK, Hoffman SL. Induction of biologically active antibodies in mice, rabbits, and monkeys by Plasmodium falciparum EBA–175 region II DNA vaccine. Mol Med. 2001 Apr;7(4):247–254.

2. Narum DL, Haynes JD, Fuhrmann S, Moch K, Liang H, Hoffman SL, Sim BK. Antibodies against the *Plasmodium falciparum* receptor binding domain of EBA–175 block invasion pathways that do not involve sialic acids. Infect Immun. 2000 Apr;68(4):1964–1966.

3. Liang H, Narum DL, Fuhrmann SR, Luu T, Sim BK. A recombinant baculovirus-expressed *Plasmodium falciparum* receptor-binding domain of erythrocyte binding protein EBA–175 biologically mimics native protein. Infect Immun. 2000 Jun;68(6):3564–3568.

Patent Status: HHS Reference No. E–004–2004/2—

- U.S. Patent No. 7,025,961 issued 11 Apr 2006
- Australian Patent No. 20042011615 issued 11 May 2007
- Canadian Application No. CA236247
- Japanese Application No. JP2000–602280 (published as JP,2002–540770,A)

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

Licensing Contact: RC Tang, JD, LLM; 301–435–5031; *tangr@mail.nih.gov*

Dated: *December 1, 2008.*

Richard U. Rodriguez,

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

[FR Doc. E8–29146 Filed 12–9–08; 8:45 am]

BILLING CODE 4140–01–P

DEPARTMENT OF HEALTH AND HUMAN SERVICES

Substance Abuse and Mental Health Services Administration

Mandatory Guidelines for Federal Workplace Drug Testing Programs

Correction

In notice document E8–26726 beginning on page 71858 in the issue ofTuesday, November 25, 2008, make the following correction:

On page 71858, in the first column, under the **DATES** heading, in the first line, ''*Effective Date:* March 25, 2008'' shouldread ''*Effective Date:* May 1, 2010''.

[FR Doc. Z8–26726 Filed 12–9–08; 8:45 am]

BILLING CODE 1505–01–D