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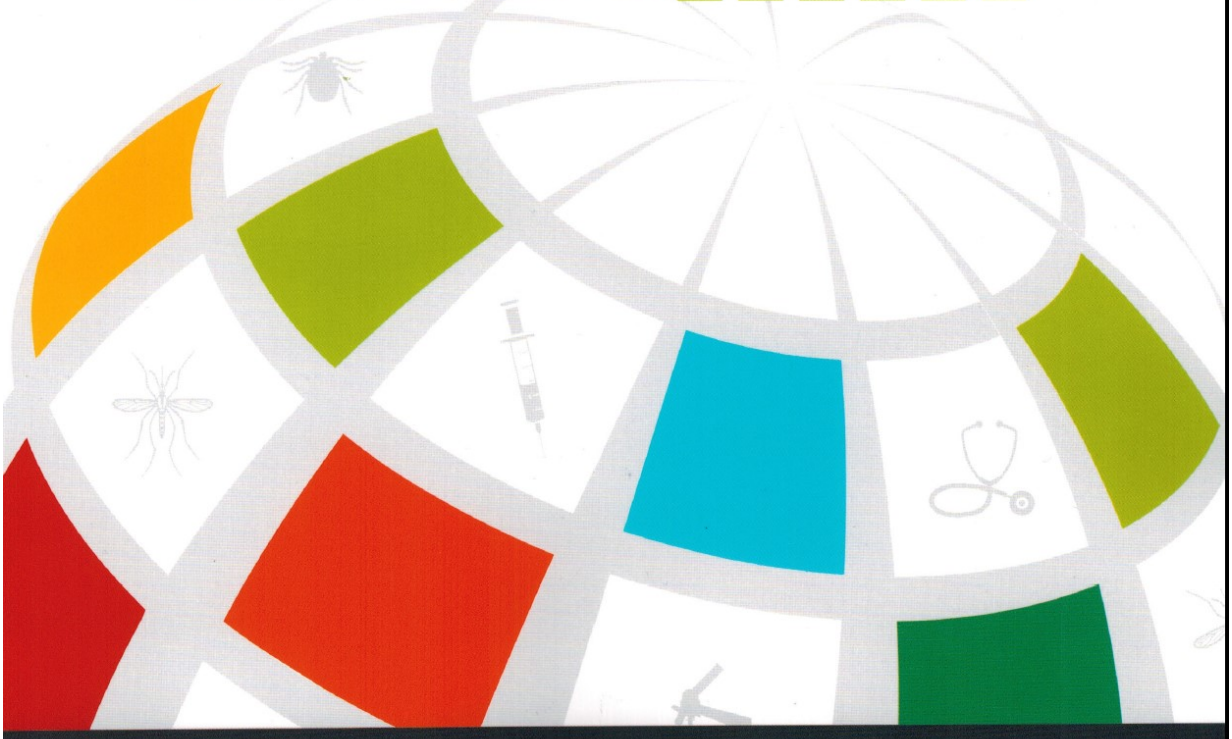

Supplement to
The American Journal of Tropical Medicine and Hygiene

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HE MALARIA ATTACK RATE IN A TANZANIAN P. A COMPARISON BETWEEN SUBJECTS E AND NON-IMMUNE POPULATIONS ALARIA PROPHYLAXIS, VACCINE AND TUDIES

ncis Filbert[1], Charles Mwanziva[2], Lucky Temu[3], Sarah nabel[4], George Amoo[5], Lalaine Anova[6], Colin Ohrt[4], Eyako esase[1], Yadon M. Kohi[2], Deus Ishengoma[1], Dennis Janga[2]
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THONOUS MALARIA IN THE ATLANTIC FOREST E JANEIRO STATE, BRAZIL

olis, Brazil

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VE COHORT STUDY REVEALS NO N BETWEEN ABO BLOOD GROUPS AND THE OMPLICATED MALARIA

bdramane Traore[2], Lala B. Sissoko[2], Moussa Niagaly[2], thily[2], Tuan M. Tran[3], Shanping Li[3], Ogobara K. Doumbo[2], [2], Kassoum Kayentao[2], Aissata Ongoiba[2], Peter D. Crompton[3],

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GPS DATA LOGGERS TO DESCRIBE SPATIO-MOVEMENT PATTERNS AND CORRELATIONS RIA RISK IN AN AREA OF HYPERENDEMIC NORTHERN ZAMBIA

Mike Chaponda[2], Kelly M. Searle[1], James Lupiya[2], Jailos ki Kobayashi[1], Timothy M. Shields[1], Modest Mulenga[2], Frank C. m J. Moss[1]
Bloomberg School of Public Health, Baltimore, MD, United States, Research Centre, Ndola, Zambia, [3]Macha Research Trust, Macha,

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IVE AND SUSTAINED DECLINE OF MALARIA OR NINE YEARS AT THE COMMUNITY LEVEL IN TERN TANZANIA

a, Filbert Fransis, Bruno P. Mmbando, Deus S. Ishengoma, cela, Johari Y. Sadi, Mathias L. Kamugisha, Martha M. Lemnge
nited Republic of Tanzania

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OST OF INFECTION: MALARIA ACCELERATES DEGRADATION

sghar[1], Victor Yman[1], Klara Sonden[1], Manijeh Vafa Homann[1], quist[2], Staffan Bensch[2], Anna Färnert[1]
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MALARIA EPIDEMIOLOGICAL STRATIFICATION IN VIETNAM, 2014

Thang D. Ngo[1], Hung X. Le[1], Hung M. Nguyen[1], Thieu Q. Nguyen Q. Nguyen[1], Trung D. Ho[1], Xa X. Nguyen[1], Anh Q. Nguyen[1], Ha S. Dinh[1], Nicholas Martin[2], Colin Ohrt[3], Duong T. Tran[1], The NIMPE Advisory Group Members[1]
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IMPACT OF ONE VS. TWO ROUNDS OF ANNUAL IRS ON MALARIA PARASITAEMIA IN CHILDREN IN NORTHERN GHANA

Benjamin Abuaku[1], Paul Psychas[2], Philip Ricks[3], Collins Ahorlu[1], Peter Mumba[4], David Mensah[1], Sedzro Mensah[1], William Sackey[1], Samuel Oppong[5], Kwadwo Koram[1]
[1]Noguchi Memorial Institute for Medical Research, Accra, Ghana, [2]University of Florida, Gainesville, FL, United States, [3]U.S Agency for International Development, Accra, Ghana, [4]Ghana Africa IRS Project, Abt Associates, Accra, Ghana, [5]National Malaria Control Programme, Public Health Division, Ghana Health Service, Accra, Ghana

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HIGH PREVALENCE OF MALARIA DESPITE PREVENTION AMONG PREGNANT WOMEN ATTENDING A DISTRICT HOSPITAL IN DOUALA, CAMEROON

Leopold G. Lehman[1], Gaëlle Mbiakop Kemajou[1], Celestin Zam-Ngono[2], Loick P. Kojom Foko[1], Larissa Kouodjip Nono[3], Herve Nyabeyeu Nyabeyeu[1], Arlette L. Ngapmen Yamadji[3], Lafortune Kangam[3], Nicolas Nolla[1], Calvin Tonga[1]
[1]University of Douala, Douala, Cameroon, [2]District Hospital of Cité des Palmiers, Douala, Cameroon, [3]University of Yaounde I, Yaounde, Cameroon

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MALARIA CHARACTERISTICS AND TRENDS, UNIVERSITY HOSPITAL, TEGUCIGALPA, HONDURAS, 2000-2014

Jackeline Alger, Jorge A. Garcia
University Hospital, Tegucigalpa, Honduras

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THE IMPACT OF FIONET™ TECHNOLOGY ON PASSIVE MALARIA SURVEILLANCE IN TANZANIAN MILITARY HEALTH SYSTEM

Christopher Mswanya[1], Akili Kalinga[2], Lucky Temu[3], Lalaine Anova[4], Eyako Wurapa[5], Colin Ohrt[5], Sarah Chiduo[3], George Amoo[6], Charles Mwanziva[1], Ian Fine[7], Geeta Bhat[7], Dennis Janga[1]
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IMPACT OF INTERMITTENT MASS SCREENING AND TREATMENT (IMSAT) ON COMMUNITY MALARIA PARASITEMIA PREVALENCE IN AN AREA OF HIGH TRANSMISSION - KENYA 2013-2014

Aaron M. Samuels[1], Norbert Awino Odero[2], Wycliffe O. Odongo[2], George Okoth[2], John Williamson[1], Kephas Otieno[2], Peter Otieno[2], Ya P. Shi[1], Mary J. Hamel[1], Kim Lindblade[1], S. Patrick Kachur[1], Simon Kariuki[2], Meghna Desai[1]
[1]Centers for Disease Control and Prevention, Atlanta, GA, United States, [2]Kenya Medical Research Institute, Kisumu, Kenya

Scientific Program Committee Meeting

Marriott - Liberty Ballroom Salon AB
Wednesday, October 28, 7 a.m. - 8 a.m.

Press Room

Marriott - Room 405
Wednesday, October 28, 8 a.m. - 5 p.m.

Symposium 112

Epidemiology and Treatment of Congenital Chagas Disease

Marriott - Grand Ballroom Salon AB
Wednesday, October 28, 8 a.m. - 9:45 a.m.

Chagas disease, caused by infection with the parasite *Trypanosoma cruzi*, affects 8–11 million persons globally. In the endemic areas of Mexico, Central America and South America, most infections are transmitted by triatomine insect (kissing bug) vectors. However, infection also can be acquired congenitally. Migration from endemic areas has led to an estimated 300,000 persons in the United States with chronic Chagas disease, including women of reproductive age who risk transmitting the infection to their children. Recently, the first documented case of congenital transmission of *T. cruzi* was detected and treated in the U.S. (http://www.cdc.gov/mmwr/preview/mmwrhtml/mm6126a1.htm). Detection of pregnant women infected with *T. cruzi* is a first step in detecting congenital transmission. Strategies to detect congenital infection in endemic countries present challenges. Many women deliver at home, rather than in a health care facility. Obtaining specimens from cord or peripheral blood of the infant in order to detect parasitemia is challenging in a home-delivery situation. Infected newborns often are asymptomatic or have subtle manifestations. The 10%–40% of newborns who are symptomatic might have low birth weight, low Apgar scores, hepatosplenomegaly, respiratory distress, anasarca, cardiac failure or meningoencephalitis. Severe congenital Chagas disease carries a high risk for neonatal death. The diagnosis can be made by detecting *T. cruzi* in cord blood or peripheral blood from the newborn by examination of Giemsa-stained blood smears or buffy coat by light microscopy. A positive PCR should be confirmed with a second specimen, because low levels of DNA occasionally are found at birth in uninfected children born to infected mothers. If all results are initially negative, testing of the child should be repeated at 4–6 weeks to confirm lack of infection, because the level of parasitemia increases in the month after birth. Results of serologic testing of uninfected children should be negative at age 9–12 months, after maternal antibodies have waned (http://www.cdc.gov/mmwr/preview/mmwrhtml/mm6126a1.htm). Treatment of congenital infection is highly effective, with cure rates >90% when instituted in the first few weeks of life. Benznidazole and nifurtimox, the antitrypanosomal drugs used to treat Chagas disease, are not Food and Drug Administration–approved in the United States, but they are available through CDC for use under investigational protocols. Access to these drugs and administration in resource poor areas is problematic.

CHAIR

Joe P. Bryan
Centers for Disease Control and Prevention, Guatemala City, Guatemala

Celia Cordon-Rosales
Centro para Estudios de Salud, Universidad del Valle de Guatemala, Guatemala City, Guatemala

8 a.m.
EPIDEMIOLOGY OF CONGENITAL CHAGAS' DISEASE

Caryn Bern
University of California San Francisco School of Medicine, San Francisco, CA, United States

8:20 a.m.
SCREENING FOR *T. CRUZI* INFECTION IN PREGNANT WOMEN AND INFANTS

Pierre Buekens
Tulane University School of Public Health and Tropical Medicine, New Orleans, LA, United States

Jackeline Alger
Hospital Escuela Universitario, Tegucigalpa, Honduras

8:40 a.m.
TREATMENT OF CONGENITAL CHAGAS' DISEASE

Sergio Sosa-Estani
ANLIS Dr. Carlos G. Malbran, Instituto Nacional de Parasitología, Buenos Aires, Argentina

9 a.m.
POLICY AND PRACTICE FOR DETECTION AND TREATMENT OF CONGENITAL CHAGAS' DISEASE

Luis Gerardo Castellanos
Pan American Health Organization, Washington, DC, United States

Scientific Session 113

Dengue: Pathogenesis

Marriott - Grand Ballroom Salon CD
Wednesday, October 28, 8 a.m. - 9:45 a.m.

CHAIR

Henry Puerta-Guardo
University of California Berkeley, Berkeley, CA, United States

Kelly L. Warfield
Unither Virology LLC, Silver Spring, MD, United States

8 a.m. **1253**

DENGUE VIRUS NON-STRUCTURAL PROTEIN 1 INDUCES ACTIVATION OF HEPARANASE/CATHEPSIN-L AND DEGRADATION OF THE ENDOTHELIAL GLYCOCALYX, LEADING TO ENDOTHELIAL PERMEABILITY

Henry Puerta Guardo, Dustin Glasner, Eva Harris
Division of Infectious Diseases and Vaccinology, School of Public Health, University of California Berkeley, Berkeley, CA, United States