EDITORIAL

Preparing the United States for Zika Virus: Pre-emptive Vector Control and Personal Protection

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Discovered in 1947 in a monkey in the Zika forest of Uganda, Zika virus was dismissed as a cause of a mild illness that was confined to Africa and Southeast Asia and transmitted by *Aedes* mosquitoes. In 2007, Zika virus appeared outside of its endemic borders in an outbreak on the South Pacific Island of Yap. In 2013, Zika virus was associated with a major neurological complication, Guillain-Barré syndrome, in a larger outbreak in the French Polynesian Islands. From the South Pacific, Zika invaded Brazil in 2015 and caused another severe neurological complication, fetal microcephaly. The mosquito-borne transmission of Zika virus can be propagated by sexual transmission and, possibly, by blood transfusions, close personal contacts, and organ transplants, like other flaviviruses. Since these combined mechanisms of infectious disease transmission could result in catastrophic incidences of severe neurological diseases in adults and children, the public should know what to expect from Zika virus, how to prevent infection, and what the most likely failures in preventive measures will be. With federal research funding stalled, a Zika vaccine is far away. The only national strategies to prepare the United States for Zika virus invasion now are effective vector control measures and personal protection from mosquito bites. In addition to a basic knowledge of *Aedes* mosquito vectors and their biting behaviors, an understanding of simple household vector control measures, and the selection of the best chemical and physical mosquito repellents will be required to repel the Zika threat.

Key words: Zika virus, arboviruses, mosquito-borne infectious diseases, *Aedes aegypti*, *Aedes albopictus*

Four unexpected mosquito-borne arboviruses immigrated to the Americas from the tropics within 3 decades.¹ First, dengue slipped into the Americas from Southeast Asia (1980s) and is now established on the Mexican border. Then West Nile virus arrived from Africa and the Middle East (1999) and quickly crossed the continental United States. Recently, chikungunya arrived from the Caribbean via East Africa and India (2013), and Zika virus invaded the Americas from the South Pacific (2015).¹ Although all of the mechanisms responsible for these viral migrations cannot

be explained, the global movements of these tropical viruses have been facilitated by international air travel capable of transporting virus-infected humans from endemic regions to anywhere in a warming world within 24 hours.

THE EPIDEMIOLOGY OF ZIKAVIRUS DISEASE

First discovered in 1947 in a rhesus monkey in the Zika forest of Uganda, Zika virus was dismissed as a cause of a periodic, mild febrile illness with rash and conjunctivitis confined to Africa and Southeast Asia.² Decades later, Zika erupted outside of its endemic borders on the South Pacific island of Yap in 2007.³ The Yap outbreak was once again characterized by uncomplicated, short-term febrile illnesses.³ However, by 2013, Zika virus was first associated with a major neurological complication, Guillain-Barré syndrome (GBS), an ascending flaccid paralysis, with over 40 cases reported in a larger outbreak

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in the French Polynesian Islands.⁴ From the French Polynesian Islands, Zika moved to Easter Island and then on to the Americas, invading Brazil in 2015.⁵

Clinical and neuroimaging studies have demonstrated that Zika's neurological complications represent a spectrum of central nervous system disorders with GBS linked to myelitis and meningoencephalitis, and microcephaly associated with severe brain, optic nerve, and chorioretinal damage. The Zika virus has now caused over 4000 cases of congenital microcephaly, a tragic birth defect characterized by a small, misshapen head with severe brain and ocular malformations, in Brazil.⁵ Zika virus has been detected in the amniotic fluid and placentas of infected mothers and in the brains of microcephalic stillbirths and neonates.⁵ In just a short period of time (2007–2013), the Zika virus has gained the capability to cause not only asymptomatic (80%) or mild (20%) illnesses in most people, but also severe neurological complications in adults and infants.

In an unprecedented method of arthropod-borne disease transmission, the sexual transmission of the Zika virus from males and females to their sex partners was confirmed serologically by rising immunoglobulin M Zika antibody titers and molecularly by viral RNA detection by reverse transcriptase-polymerase chain reaction.^{6,7} In addition to local mosquito-transmitted infections, the spread of Zika virus disease will be accelerated by sexual transmission, blood and body fluid contact, and organ transplantation. Such combined mechanisms of infectious disease transmission could result in catastrophic incidences of severe neurological diseases in adults and children.

Since 2015, over 3000 Americans have contracted Zika virus disease while travelling in Zika-endemic regions. Today, the mosquito-borne, local transmission of Zika virus disease occurs as close to the US mainland as its Caribbean territory, Puerto Rico, which is experiencing a \$70 billion debt crisis and a weakened public health infrastructure. Puerto Rico has reported over 17,000 mosquito-transmitted cases of Zika virus disease, with over 800 in pregnant women, 1 fatal case of GBS, and 1 neonatal fatality with microcephaly.

Although travel-related or imported Zika virus disease is still the predominant mode of disease transmission in the United States, local mosquito transmission is the predominant mode of disease transmission in the Caribbean and throughout Latin America. The local mosquito transmission of Zika virus disease has now been reported in Florida and is anticipated to occur in additional states shortly as more infected people arrive in the United States from hyperendemic nations in the Caribbean and Latin America.

As the numbers of imported and mosquito-borne cases of Zika virus disease are increasing over time in the

Figure 1. A female *Aedes aegypti* or yellow fever mosquito is acquiring a blood meal from a human host. This daytime-biting mosquito prefers to blood-feed on humans more than on animals and is a competent vector of Zika virus and other flaviviruses, including yellow fever virus, dengue virus, and chikungunya virus. Note the white bands on its legs and the silvery white lyre-shaped markings on its dorsal thorax. Source: The United States Centers for Disease Control and Prevention (CDC) Public Health Image Library (PHIL), PHIL ID 9253, Professor Frank Hadley Collins, Director, Center for Global Health and Infectious Diseases, University of Notre Dame. Photographer: James Gathany, Biomedical Photographer, CDC.

Figure 2. A female *Aedes albopictus* or Asian tiger mosquito is blood-feeding on a human host. This daytime-biting mosquito feeds on both animals and man and is a competent transmitting vector for chikungunya and dengue viruses. *A. albopictus* is suspected to be a competent vector for the Zika virus, which is transmitted by several other *Aedes* species in addition to *Aedes aegypti*, the predominant vector in Latin America and the Caribbean. *A. albopictus* has white bands on its legs like *A. aegypti* and a single, longitudinal silvery white line on its dorsal thorax, which helps to distinguish it from *A. aegypti*. Source: The United States Centers for Disease Control and Prevention (CDC) Public Health Image Library (PHIL), PHIL ID 1864, Professor Frank Hadley Collins, Director, Center for Global Health and Infectious Diseases, University of Notre Dame. Photographer: James Gathany, Biomedical Photographer, CDC.

Figure 3. The regional geographic distribution ranges of *Aedes aegypti* (left) and *Aedes albopictus* (right) in the United States and some of its territories. Note the significantly broader distribution range of *A. albopictus* (right) compared with *A. aegypti* (left), which extends from the New England states throughout the Midwest to California. *A. albopictus* is also distributed throughout the Hawaiian Islands. *A. aegypti* is distributed primarily throughout the southern tier of the United States from coast to coast. *A. aegypti* is the predominant vector of the Zika virus in Latin America and the Caribbean, including Puerto Rico, a US territory. Source: The United States Centers for Disease Control and Prevention (CDC), Surveillance and Control of *Aedes aegypti* and *Aedes albopictus* in the United States.

United States and its territories, interested readers are encouraged to check the updates on Zika cases and complications at publication time and periodically on the US Centers for Disease Control and Prevention website available at <http://www.cdc.gov/zika/geo/united-states.html>.

THE MOSQUITO VECTORS OF ZIKA VIRUS AND THEIR BITING BEHAVIORS

Three flaviviruses, yellow fever, dengue, and Zika, and 1 alphavirus, chikungunya, are all transmitted to humans by female *Aedes* species mosquitoes, predominantly *Aedes aegypti*, the yellow fever mosquito. *A. aegypti* is widely distributed throughout Latin America and the Caribbean to the southern United States (Figure 1). Recently, *Aedes albopictus*, the Asian tiger mosquito, formerly considered a secondary vector for dengue and chikungunya, surpassed *A. aegypti* as a preferred vector for both flaviviruses and facilitated epidemic spread of these diseases in many endemic areas (Figure 2). A gene-directed change in a single amino acid sequence on the chikungunya's surface glycoprotein envelope enabled the virus to utilize *A. albopictus* as a vector.⁸ A similar gene-directed change in Zika's structure may adapt the virus to additional transmission by *A. albopictus*, which

has a much broader distribution range in the United States (36 states) than *A. aegypti* (26 states) (Figure 3).

Since *Aedes* species mosquitoes are container breeders, any site that can collect and hold freshwater, from an upturned bottle cap to an abandoned tire, can serve as a breeding ground for *Aedes* females, which can lay up to 200 eggs at a time, 4 to 5 times per year depending on the temperature. Even during winter seasons, wide diurnal variations in temperatures can stimulate egg-laying and shorten extrinsic incubation times, or the time it takes for the human blood meal–obtained virus to migrate from the mosquito's hemocoel to its salivary glands and become infective. *Aedes* eggs are environmentally stable and remain viable for up to a year during dry seasons in the crusted biofilms of empty containers. Table 1 contrasts the morphological features, geographic ranges, ecosystem preferences, biting behaviors, and resting and egg-laying preferences of *A. aegypti* and *A. albopictus*.

Outdoor enthusiasts are cautioned that *Aedes* mosquitoes are daytime biters and sip feeders, preferring multiple small-sip human blood meals when they can sense, see, and repeatedly attack their hosts best. Although mosquito-proof hammocks, sleeping bags, and nets will protect outdoor campers at night from twilight and nighttime feeding *Anopheles* and *Culex* mosquitoes, they are not as effective against day-biting *Aedes* species.

Table 1. The distribution and distinguishing features of *Aedes aegypti* and *Aedes albopictus*: the anticipated mosquito vectors of Zika virus in the United States

	<i>Aedes aegypti</i>	<i>Aedes albopictus</i>
Common names	Yellow fever mosquito	Asian tiger mosquito
Coloration and morphology		
Legs	White-banded markings on the tarsal segments of jointed legs	White-banded markings on the tarsal segments of jointed legs
Thorax	Silvery-white lyre-shaped markings on the dorsal thorax	Single silvery-white longitudinal stripe on the dorsal thorax
Regional distribution in the United States	26 continental US states Primarily restricted to the Southern tier from the Chesapeake Bay region of the Mid-Atlantic to Southern California. Widely distributed throughout Puerto Rico and the US Virgin Islands.	36 continental US states Broadly distributed from the New England states in the Northeast throughout the Mid-Atlantic and Midwest across the Southern tier to California sparing the Rockies and Pacific Northwest. Distributed throughout the Hawaiian Islands.
Ecosystem preferences	Prefers highly populated urban areas with uncollected refuse and with or without vegetation	Prefers underpopulated parks and garden areas with thickets and significant vegetation
Environmental tolerances	Very sensitive to colder temperatures and inactive below 50°F, but stimulated by wide diurnal variations in temperatures. Attracted to dark objects during the daytime, especially at dawn and dusk.	Active in broader temperature ranges and at cooler temperatures than <i>A. aegypti</i> . Also stimulated by wide diurnal variations in temperatures. Attracted to bright and colorful objects during the daytime.
Biting preferences	Prefers daytime blood feeding on humans rather than animals.	No preferences for biting warm-blooded mammals including domestic and wild vertebrates and humans.
Biting behaviors	Considered a sneaky biter and will attack multiple times from behind for small blood meals (“sip feeder”). Bites, rests, and lays eggs both indoors and outdoors.	Considered an aggressive biter and will launch multiple frontal attacks. Also a sip feeder and will take multiple small blood meals from many hosts. Prefers an outdoor lifestyle for biting, resting, and egg laying.
Infectious diseases vectored (biting females only)	The preferred Zika virus vector worldwide. The main yellow fever and dengue vector worldwide. Becoming a secondary vector to <i>A. albopictus</i> for chikungunya in some areas of Africa and Southeast Asia.	Suspected as a secondary Zika virus vector based on its capabilities to transmit other flaviviruses, especially dengue. Has replaced <i>A. aegypti</i> as the main dengue and chikungunya vector in some areas. Not known to transmit yellow fever.
Progeny development site preferences	Prefers to lay eggs in human-made containers near households that trap and hold stagnant freshwater, such as tires, garbage cans, food cans, bottle caps, and clogged gutters, rather than in natural freshwater traps in tree-holes, tree-forks, bromeliads, and mud puddles. Known to lay from 100–200 eggs in an upturned bottle cap. Unhatched eggs can remain viable for up to a year during dry seasons.	Prefers to lay eggs in natural freshwater traps in tree-holes, tree-forks, bromeliads, and mud puddles away from human activities and households. Will also utilize human-made containers that trap and hold stagnant freshwater nearer households, especially during dry periods. Unhatched eggs can remain viable for up to a year during dry seasons.

Table 2. Vector control strategies and their individual utilities in *Aedes* mosquito control

<i>Vector control methods</i>	<i>Utilities and/or adverse effects in Aedes control</i>
Physical vector control methods	
Interior households: provide air conditioning or pyrethroid-impregnated porch and window screens, curtains, or drapes; remove household debris.	Very effective in developed nations, especially against malaria-transmitting mosquitoes, but difficult to achieve in crowded, impoverished periurban areas.
Exterior households: remove yard debris and any potential containers that can provide mosquito-breeding sites, especially large vessels and tires; trim or remove excessive yard vegetation.	Highly effective against domestic container-breeders, such as <i>Aedes</i> mosquitoes, but difficult to enforce in crowded, impoverished periurban areas.
Wear light-colored, long-sleeved shirts and pants when outdoors.	Effective against <i>Aedes</i> mosquitoes that are attracted to dark colors.
Wear light-colored pyrethroid insecticide-impregnated clothing when outdoors.	Effective against <i>Aedes</i> mosquitoes that are attracted to dark colors and sensitive to pyrethroid insecticides. Pyrethroid resistance is increasing.
Chemical vector control methods	
Widespread organophosphate spraying of mosquito habitats, especially during times of peak mosquito activity, such as dawn and dusk.	Less effective for <i>Aedes</i> mosquitoes that prefer peridomestic habitats.
Interior and exterior household insecticide (synthetic pyrethroids only) spraying of adult mosquito populations.	Effective for <i>Aedes</i> mosquitoes, but the costs of the insecticides may limit applications in impoverished areas or promote the interior household use of toxic organophosphate insecticides.
Widespread larviciding with monomolecular films and oils that will float on freshwater surfaces, suffocating larvae.	Not as effective for <i>Aedes</i> mosquitoes that prefer to lay eggs in containers near households from upturned bottle caps and tree holes to cisterns and abandoned tires.
Apply topical mosquito repellents, such as 20–30% DEET or picaridin, to exposed skin at all times when outdoors. Consider combining topical mosquito repellents with synthetic pyrethroid-impregnated clothing when outdoors.	Highly effective against all mosquitoes and against other infectious disease-transmitting arthropods, such as ticks.
Biological vector control methods	
Supply freshwater copepods and fish species (<i>Gambusia affinis</i>) that feed on mosquito larvae.	Effective in large water storage containers and in ponds and swimming pools filled with stagnant freshwater after tropical storms and hurricanes. Introduced species could pose a threat to indigenous species.
Widespread biological larviciding with nonpathogenic bacteria that feed on mosquito larvae (<i>Bacillus thuringiensis</i>).	Not as effective for <i>Aedes</i> mosquitoes that prefer to lay eggs in containers near households from upturned bottle caps and tree holes to cisterns and abandoned tires.
Genetic vector control methods	
Radiation-sterilized male mosquitoes cause females to produce nonviable eggs after mating.	Technically sophisticated and expensive; sterilized male mosquitoes have to be repeatedly released in different regions.
Genetically engineering male mosquitoes to pass a fatal gene to offspring.	Technically sophisticated and expensive; the fatal gene could affect important mosquito predators, such as other insects and birds. Genetically engineered male mosquitoes have to be repeatedly released in different regions.
Genetically engineering male mosquitoes to pass a symbiotic bacterium (<i>Wolbachia</i> species) to females, rendering them infertile.	Very highly promising, but technically sophisticated and expensive. Genetically engineered male mosquitoes have to be repeatedly released in different regions.

CONTROL AND PREVENTION STRATEGIES FOR ZIKA VIRUS DISEASE

Although virologists and vaccinologists have learned much more about the immunological mechanisms of flaviviral disease transmission from recent experiences

with investigational West Nile and dengue vaccines, a Zika virus vaccine is far away, especially with federal research funding stalled in Congress. In addition, a Zika vaccine that is rushed from production into mass administration before human trial testing could itself precipitate GBS or be inactivated by cross-reacting

Table 3. Available mosquito repellents: formulations, efficacy, safety, and toxicity¹⁰

<i>Insect repellents (Chemical names)</i>	<i>Formulations (Strength %)</i>	<i>Efficacy against Culicine (flavivirus-carrying) mosquitoes</i>	<i>Safety in children and pregnancy</i>	<i>Toxicity and other adverse effects</i>
DEET (<i>N, N</i> -diethyl-3-methylbenzamide Formerly <i>N, N</i> -diethyl- <i>m</i> -toluamide)	Aerosols, lotions, pump sprays, wipes (5-100%)	+++	FDA: do not use in children under age 2 years. Potential neurotoxicity in children if ingested or if overapplied. American Academy Pediatrics recommends maximum strength of 30%. DEET crosses the placenta. FDA Pregnancy Category N.	Potential neurotoxicity if applied under sunscreen. May damage plastic and some synthetic fabric clothing. Safe for cotton, wool, and nylon.
Picaridin (US) & Icaridin (EU) (2-[2-hydroxyethyl]-1-piperidine-carboxylic acid 1-methylpropyl-ester)	Lotions, pump sprays, wipes (7–20%)	+++	No studies in children, but manufacturers do not recommend use in children younger than 2 years of age. No developmental toxicity in animals. FDA Pregnancy Category N.	Possible skin irritation. No damage to plastics or clothing.
IR3535 (3-[<i>N</i> -butyl- <i>N</i> -acetyl]-amino-propionic acid ethyl ester)	Aerosols, lotions, pump sprays, wipes (7.5–19.7%)	++ EPA: up to 2 hours protection time for mosquitoes.	FDA Pregnancy Category B.	Causes eye irritation. Potential toxicity if ingested or inhaled.
Oil of lemon eucalyptus (<i>p</i> -menthane-3, 8-diol)	Pump sprays (10%–40%)	++ EPA: up to 2 hours protection time for mosquitoes.	May cause seizures or death if ingested. FDA: do not use under age 3 years. FDA Pregnancy Category N.	May damage plastic and clothing. Potential skin irritation in atopic individuals.
Citronella (3, 7-dimethyloct-6-en-1-al) Natural plant oil obtained from <i>Cymbopogon</i> spp. grasses.	Bath oils, candles, lotions (0.5–20%)	+	Potential neurotoxicity if ingested or inhaled. FDA Pregnancy Category N.	May damage clothing. Potential eye irritation, skin irritation, and allergies.
Permethrin (3-phenoxybenzyl (1 <i>RS</i>)- <i>cis</i> , <i>trans</i> -3-(2, 2-dichlorovinyl)-2, 2-dimethyl-cyclo-propane-carboxylate), a pyrethroid insecticide derived from dried, crushed flowers of <i>Chrysanthemum</i> spp.	Sprays for clothing, insect nets, sleeping bags, boots (0.5%)	+++	Potential neurotoxicity if ingested or inhaled. FDA: do not use in children under age 2 years. FDA: Pregnancy Category B.	Not useful on the skin. Possible skin irritation. Pyrethroid resistance is now developing in mosquitoes. No damage to plastics or clothing.

Protective Efficacy Scale: 0, no protection provided; +, minimal level of protection; ++, moderate level of protection; +++, maximal level of protection.

FDA Pregnancy Categories: A, human studies have demonstrated no evidence of risk to the fetus; B, animal studies have demonstrated no evidence of risk to the fetus; C, animal studies have demonstrated adverse effects on the fetus; D, investigational or marketing experiences or human studies have demonstrated adverse effects on the fetus, but potential benefits may warrant use of the drug in pregnancy despite the risks; X, studies in animals or humans have demonstrated fetal abnormalities; N, FDA has not classified the drug.

antibodies from prior flaviviral experiences with dengue, West Nile virus, or even the yellow fever vaccine.⁹ Therefore, the only effective national strategies to immediately prepare the United States for the Zika virus invasion are vector control measures and personal protection from mosquito bites. In addition to a basic knowledge of the *Aedes* mosquito vectors and their biting behaviors, an understanding of simple vector control measures and the selection of the best chemical and physical mosquito repellents will be required to prepare citizens to repel the Zika threat.

MOSQUITO VECTOR CONTROL STRATEGIES

Mosquito vector control strategies may be classified as physical, chemical, biological, and genetic. The most effective combinations of vector control strategies should be well matched to the habitat, egg-laying preferences, and the biting behaviors of the targeted mosquito vectors. Although state and local governments typically provide for regional chemical, biological, and genetic vector control programs, every citizen has a personal responsibility for implementing simple physical vector control strategies, such as removing yard debris, unblocking clogged drains and roofing gutters, and emptying or covering all potential freshwater containers. Table 2 describes the 4 types of mosquito control strategies and their most effective applications in vector control programs targeted at *Aedes* species vectors.

PERSONAL PROTECTIVE STRATEGIES TO PREVENT MOSQUITO BITES

In addition to some simple physical control strategies, such as window screens and air-conditioned households, personal protective strategies to prevent mosquito bites include wearing light-colored, long-sleeved shirts and long pants and applying effective mosquito repellents throughout the day. Mosquito repellents may be divided into two basic chemical classes: 1) synthetic chemicals, such as DEET, picaridin, and IR3535 (Avon Skin So Soft); and 2) plant-derived oils and synthetics, such as oil of lemon eucalyptus, oil of citronella, and permethrin.¹⁰ Table 3 describes the range of mosquito repellents and insecticides available worldwide as stratified by their active ingredients, formulations, strengths (%), efficacies against mosquitoes, precautions, and adverse effects.¹⁰ Although recommendations to combine clothing impregnated with insecticides, such as permethrin, with mosquito repellents, such as DEET or picaridin, are not supported by randomized controlled trials, the most effective uses of mosquito repellents are to apply a topical repellent on exposed skin and to wear permethrin- or other synthetic pyrethroid-impregnated

clothes that act as contact insecticides and provide better and longer lasting protection against mosquito bites.¹⁰

CONCLUSIONS: WHAT CAN WE EXPECT? WHAT CAN WE DO? WHAT PREVENTIVE MEASURES WILL WORK OR FAIL?

The local mosquito-borne transmission of Zika virus disease in the United States is inevitable as more viremic persons arrive from Zika-endemic areas during the summer Atlantic coastal mosquito-breeding and tropical storm seasons. Although aerial insecticide spraying of freshwater wetlands can control West Nile virus-transmitting mosquitoes, it is less effective for *Aedes* mosquitoes that prefer peridomestic habitats, not marshlands, with humans rather than animals available for blood feeding and accessible containers everywhere for egg laying. Policing properties and public spaces for potential larval-breeding containers and frequently emptying or eliminating them combined with local insecticide spraying of households (pyrethroids only) and public spaces will be essential. Although promising, genetic vector control strategies are technically sophisticated, very expensive, and need to be repeated frequently as new areas become Zika-endemic. Slow and inadequate (\$800,000 to \$1.9 billion) federal funding will delay not only needed research and implementation of genetic vector control strategies, but also human vaccine development and efficacy testing. Unlike Ebola and West Nile viruses, the new Zika virus strain has no apparent animal reservoir and even asymptomatic Zika-infected persons can spread the virus via mosquitoes or sex, with unknown potential for postinfectious central nervous system complications, such as GBS and microcephaly.

Mosquito-borne transmission will be magnified by sexual transmission from asymptomatic males to their sex partners as Zika remains capable of replication for months in semen. Protective measures will include not only physical and chemical vector control (Table 2) and personal protection with pyrethroid-impregnated clothing and topical insect repellents (Table 3), but also proper condom use for prolonged periods during sex (throughout pregnancy and up to 9 months) by males who have had Zika virus disease or visited Zika-endemic areas. The defensive lessons learned now and combined with ongoing research in flaviviral immunology and genetic mosquito vector control will better prepare us for the next arthropod-borne pandemic in our changing world ecosystem.

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