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SM Journal of Public Health and Epidemiology

Review Article

Chikungunya Epidemiology: A Global Perspective

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Article Information

Received date: May 02, 2016
Accepted date: May 23, 2016
Published date: May 24, 2016

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Keywords Chikungunya; Aedes; Southeast Asia; Central Africa

Abstract

After being discovered on the borders of Mozambique and Tanzania in 1952, the Chikungunya Virus (CHIKV) is now classified as a category C priority pathogen, as it has spread to over 40 countries worldwide. As the virus circulates through either human or mosquito vectors, it is important to closely monitor its progression within vulnerable countries, as it mimics the tenacity of other dangerous diseases such as the Dengue virus and more recently the Zika virus that has caused an outbreak in Brazil and has spread to other Latin American regions. The purpose of this study is to provide an overview of the Chikungunya viral infection by relating the history with the spread of the disease as well as its impact on global populations. Understanding the transmission of the virus, as well as its current spread (in relation to Central / East Africa), will enable conclusions to be made about which treatment and prevention methods should be implemented in order to target those specific demographics. The demographics that were found to have the highest prevalence of Chikungunya include Southeast Asia and Central Africa. Since this virus has the ability for global spread, containing it and preventing further spread, requires preventative measures that must be undertaken globally.

Introduction

Chikungunya is a disease that was first described by Marion Robinson and W.H.R. Lumsden in 1955 [1]. They were led to this discovery after an outbreak in 1952 along the border between Mozambique and Tanganyika (current part of Tanzania). The specific region of focus was the Makonde Plateau. The virus was detected using the serum of an infected patient within the region.

The virus first established its presence during a 1952-1953 epidemic outbreak in East Africa, more specifically Tanzania [2]. Since then, it has become a global concern, such that it was listed as a priority by the Scientific Leadership Group for the Global Virus Network [3]. The Chikungunya virus has a single stranded positive sense RNA viral genome belonging to the Alphavirus genus of the Togaviridae family [4-6]. The *Aedes* mosquito is largely responsible for the transmission of the virus, with the target cells still widely unknown. Some studies, however, have found some human epithelial cells, endothelial cells, and fibroblasts that are sensitive to the Chikungunya virus [7]. The Chikungunya infection is divided into two phases: an acute phase and a chronic phase. Signs and symptoms of an acute infection include polyarthralgia, high fever, asthenia, headache, vomiting, rash and myalgia [8]. The Chikungunya infection often leads to prolonged joint pain, contributing to the long-lasting disease burden.

The origin of Chikungunya most likely began in Central or East Africa [2]. Researchers who've conducted studies on the genetics of the disease have indicated that a transmission cycle within these regions has surfaced in nonhuman primate hosts [2]. Speculation exists that these cycles could have begun as early as the 18th century [2]. Ocean vessels likely served as carriers of the Chikungunya virus both within infected humans as well as within the responsible mosquito, *Aedes aegypti*. These ships had large water reservoirs for drinking water, which ultimately led to the ability of the mosquito to breed. The *Aedes albopictus* mosquito has also been shown to be a second vector through which the virus can spread. The *Aedes albopictus* mosquito specifically has been linked to increased outbreaks in the Indian Ocean basin, southern Europe, and African regions [2].

Outbreaks of the virus were fairly limited at this time, and large outbreaks between humans were not common. More recently, the virus has had more impact on a global scale. It has been reported in nearly 40 countries around the world [1]. Chikungunya was officially listed as a category C priority pathogen in 2008 by the US National Institute of Allergy and Infectious Diseases [1].

Causative Agent

The causative agent of Chikungunya is an arbovirus belonging to the genus alphavirus under the Togaviridae family [9]. The Chikungunya virus is a single-stranded, positive-sense RNA genome

with a 60-70 nanometer diameter capsid and a phospholipid envelope [10]. The viral RNA genome is approximately 11.5 kb in length and encodes four nonstructural proteins and three main structural proteins: two capsids and two envelope glycoproteins (E1 and E2). These glycoproteins form spikes on the virion surface [2].

The E1 and E2 glycoproteins play an important role in viral replication. The E2 glycoprotein allows the virus to bind to unknown cellular receptors and initiate entry into the cell via endocytosis. The E1 glycoprotein involves a fusion peptide. When the fusion peptide is exposed to the low pH in endosomes, it initiates the release of nucleocapsids into the host-cell cytoplasm [2].

Un-coating of the virion as well as mRNA transcription, take place inside the host cell cytoplasm. The replication of the virus is not limited to specific tissues or organs; therefore, viral replication may be observed in multiple host organs. The insect host initiates the viral replication. Genome replication takes place in the cytoplasm [1].

Mutations in the E1 glycoprotein have received some recent attention as some researchers have proposed that this mutation may have modified the virus's ability to infect mosquitoes and increased the severity of the illness associated with human infection [11].

Signs and Symptoms

The incubation period after being bitten by an infected mosquito can range from 2-12 days, most commonly between 3-7 days. Of the individuals who become infected, 72-97% of them will develop symptoms [12]. The acute phase of the Chikungunya infection typically lasts from a few days to a couple of weeks, and most patients recover fully. Symptoms characteristic of an acute infection includes biphasic high fever lasting from a few days to a few weeks. Back pain, fatigue, myalgia and arthralgia may or may not present with joint swelling. When present these symptoms usually last for weeks. Headache, insomnia, nausea, and vomiting may also be present. Iridocyclitis, uveitis, and retinal lesions may also occur. When the skin is involved, 50% of patients present with a maculopapular rash. Facial edema, bullous eruptions with pronounced sloughing, localized petechiae and bleeding gums are less common skin manifestations.

Individuals at risk of more severe disease include newborns and older adults (≥65 years). The presence of hypertension, diabetes and heart disease is also associated with an increased severity of infection [1,13,14]. The chronic stage of a Chikungunya infection is characterized by poly-arthralgia lasting from weeks to years beyond the acute stage [13,14]. Other possible long-term or severe complications include prolonged myalgia and fatigue, gastrointestinal upset, encephalitis, depression, lung, kidney and heart dysfunction [1]. Lifelong immunity to the Chikungunya virus is attained by most individuals after infection [13].

Diagnosis

Chikungunya virus infection is likely in individuals who have recently traveled to areas with known virus transmission who are experiencing acute onset of polyarthralgia and fever [15,16]. The patient may present with an acute onset of fever that lasts 3-5 days along with multiple joint pains that may have lasted for the past weeks to months [16]. There are various ways to detect the Chikungunya virus; however, since the presentation of Chikungunya virus infection is similar to the Dengue virus infection, the most reliable way to identify the virus is through a blood test [17]. Serum specimens of the Reverse Transcriptase- Polymerase Chain Reaction (RT –PCR) detect the viral RNA when the patient is in the acute phase of the infection [1,18]. High viral counts generally last 4-6 days after the onset of the illness; therefore, the RT- PCR is a useful diagnosis within the first 7 days [1]. This is beneficial since patients usually see their doctors during the acute phase of the illness [19]. Results of the RT- PCR generally take 1 – 2 days, which is also very efficient for diagnosis [1]. The PCR is both very specific and sensitive for the Chikungunya virus, however, its cost contributes to a decrease in widespread use [19].

Enzyme-Linked Immunosorbent Assays (ELISA) may detect both anti-Chikungunya virus Immunoglobulin IgM and IgG antibodies from either the acute or the convalescent-phase samples [1]. The ELISA test is useful because the antibodies usually develop by the end of the first week, so if the RT-PCR is negative, the virus may still be detected with the convalescent phase sample [15]. The IgG antibodies are detectable after 2 days by the ELISA and the detection may persist several weeks up to 3 months [16]. Serum IgG and IgM are the most for several widely used diagnostic tests, as they are the most economical and the easiest to perform on a patient [19]. The IgM antibody levels are highest 3 to 5 weeks after the onset of illness [18]. The serum IgG and IgM are not very sensitive for an acute Chikungunya infection (4-22%) though, they become highly sensitive after one week (up to 80%). This limitation in sensitivity makes it difficult to detect and differentiate an acute infection from a recent past infection [19]. Approximately 40% of symptomatic patients have persistent IgM antibodies 18 months after the disease onset [16].

The Hemagglutination-Inhibition (HI) assay may also be utilized in the detection of a Chikungunya infection [1,19]. The HI assay determines the level of antibodies to the virus present in serum samples [19]. When a Chikungunya viral infection is present, there is a four-fold HI antibody difference in the serum sample, which turns positive within 5 to 8 days after infection [1]. Collection of the samples should take place 3 weeks apart, one from the other, allowing for both the acute and convalescent phases to be captured [1]. A definitive diagnosis can be made with a positive serum sample along with presence of clinical signs and symptoms of infection [1]. See Table 1 for a list of diagnostic criteria for the Chikungunya virus.

Transmission

The Chikungunya virus is transmitted to humans by the bite of an infected female *Aedes* (Ae) genus mosquito. Vertical transmission between mother and fetus however, has been observed in some cases [21]. Transmission often leads to an acute febrile illness associated with an arthromyalgic syndrome [22]. Phylogenetic analyses have demonstrated 3 distinct lineages of Chikungunya strains: West Africa (*Ae. furcifer*, *Ae. luteocephalus*, and *Ae. taylori*), Asia (*Ae. aegypti* and *Ae. albopictus*), and East/South/Central Africa (ESCA) (*Ae. furcifer* and *Ae. cordellieri*) [22]. Strains from the Indian Ocean and India segregate into two independent sub-lineages that presumably derive from an East African ancestral genotype [22]. Until recently, the *Ae. aegypti* mosquito was widely accepted as the main urban vector of the Chikungunya virus. However, the *Ae. albopictus* mosquito was extensively implicated in Chikungunya transmission during the 2005-06 outbreak in Réunion Island [22]. International travel and global expansion of the two main Chikungunya urban mosquito

Table 1: Diagnostic criteria for Chikungunya fever [16].

Suspected case:
A patient presenting with acute onset of fever usually with chills/rigors, which lasts for 3-5 days with multiple joint pains/swelling of extremities that may continue for weeks to months
Probable case:
A suspected case (see above) with any one of the following:
a) History of travel or residence in areas reporting outbreaks
b) Ability to exclude malaria, dengue and any other known cause of fever with joint pains
Confirmed case:
Any patient who meets one or more of the following findings irrespective of the clinical presentation
a) Virus isolation in cell culture or animal inoculations from acute phase sera
b) Presence of viral RNA in acute phase sera by RT-PCR
c) Presence of virus-specific IgM antibodies in single sample in acute or convalescent stage
d) Fourfold increase in virus-specific IgG antibody titer in samples collected at least three weeks apart

RNA: Ribonucleic acid; **RT-PCR:** Reverse transcription polymerase chain reaction; **IgM:** Immunoglobulin **M; IgG:** Immunoglobulin **G**

vectors (*Ae. aegypti*, and *Ae. albopictus*) have enhanced the ability of the virus to spread to new regions where environmental conditions are permissive for viral transmission [23].

In addition to simio-anthropophilic mosquitoes (*Ae. furcifer-taylori*, and *Ae. luteocephalus*), zoophilic mosquitoes (*Ae. dalzieli*, *Ae. argenteopunctatus*, *Culex. ethiopicus*, and *Ae. rufipes*) can carry the virus [23]. Species of the subgenus Aediomorphus, which prefer to feed on cattle, may also facilitate circulation of Chikungunya. Isolates of the Chikungunya virus from zoophilic mosquitoes suggest that it circulates in rodents and cattle [23]. Among wild vertebrates, the virus was isolated from a Cercopithecus aethiops monkey, a *Galago senegalensis* galago, a *Xerus erythropus* palm squirrel and *Scotophillus sp*. Bats [23]. Humans serve as the reservoir for Chikungunya during epidemic periods. Outside these periods, the main reservoirs are monkeys, rodents, bats and birds [24].

The transmission cycle of Chikungunya virus is characterized by a periodicity of occurrence with silent intervals of 3-4 years [23]. These cycles, which characterize the movement of the virus in monkeys, are probably related in part to the immune status of the monkeys and to the percentage of the simian population susceptible to the infection. Following the circulation of the virus, nearly all the monkeys might be exposed to it and therefore become immunologically protected [24]. Isolates obtained from a squirrel, chiropetra, and ticks (*Alectorobius sonrai*), as well as the presence of antibodies specific for the Chikungunya virus in rodents and birds, support the assumption that secondary wild cycles exist [22]. The existence of such cycles could contribute to maintenance of the virus in an endemic region while the simian populations are immunologically protected [23].

A common theme for this second wave of expansion is the role of *Ae. albopictus* as the primary arthropod vector [25]. This is due in large part to a single amino-acid mutation in the Chikungunya E1 glycoprotein (E1-A226V) which increased the vector competence of *Ae. albopictus* approximately 50-fold compared to the more traditional vector *Ae. aegypti* [26]. In 2006, laboratory studies concluded that *Ae. aegypti* and *albopictus* mosquitoes were able to efficiently transmit both the Asian and the ESCA genotype Chikungunya strains [27].

Transmission of the Chikungunya virus appears to be temperature dependent. Incubation of infected mosquitoes at a lower temperature (20 ˚C) displayed decreased virulence of the Chikungunya strain carried by *Ae. aegypti* mosquito [26]. The strain carried by *Ae. albopictus* however, does not exhibit the same decreased virulence when incubated in constant low temperatures. However, it was found that when incubated with daily fluctuations in temperature with a mean value of 20 ˚C, transmission efficiencies and viral loads in *Ae. albopictus* saliva were slightly increased [26].

The infected mosquitoes can be found biting throughout daylight hours, though there may be peaks in the early morning and late afternoon. Both *Ae. aegypti* and *Ae. albopictus* can be found biting outdoors, but *Ae. aegypti* will feed readily indoors [18]. *Ae. aegypti* is currently confined within the tropics and subtropics, whereas *Ae. albopictus* is found more readily in temperate/cold temperate regions [18]. In recent decades, *Ae. albopictus* has spread from Asia and become established in areas of Africa, Europe and the Americas [18]. The *Ae. albopictus* mosquito thrives in a wider range of water-filled breeding sites than the *aegypti* mosquito, which can include coconut husks, cocoa pods, bamboo stumps, tree holes and rock pools, in addition to artificial containers and vehicle tires [18]. The diversity of possible habitats helps to explain the abundance of *Ae. albopictus* in rural and peri-urban areas [18].

Epidemiology

The Chikungunya virus infection has spread drastically over the past 20 years; molecular epidemiology of the strain responsible for this outbreak has indicated it may have originated from Kenya [28]. This is mainly related to travelers who visit countries or areas where Chikungunya is prevalent, and obtain the virus and transmit it when they return to non-prevalent areas [29]. The epidemics of Chikungunya infections that occurred from 2004 through 2008 demonstrated the ease with which this virus is able to spread and infect humans [28]. An abundance of countries has now reported cases of Chikungunya including the United States, India, and La Réunion Island. In 2009, research was done to establish the number of laboratory-confirmed Chikungunya cases in the United States from 1995-2009 [30]. The study concluded that there were 109 laboratory confirmed cases with a median of 26 Chikungunya cases per year between 2006-2009 [30]. The study also displayed that July to September were the most opportunistic months for a Chikungunya

Table 2: List of countries with reported cases of the Chikungunya virus [35].

AFRICA	ASIA	AMERICAS	
Benin	Bangladesh	Anguilla	Panama
Burundi	Bhutan	Antigua and Barbuda	Paraguay
Cameroon	Cambodia	Aruba	Puerto Rico
Central African Republic	China	Bahamas	Saint Barthelemy
Comoros	India	Barbados	Saint Kitts and Nevis
Dem. Republic of the Congo	Indonesia	Belize	Saint Lucia
Equatorial Guinea	Laos	Bolivia	Saint Martin
Gabon	Malaysia	Brazil	Saint Vincent & the Grenadines
Kenya	Maldives	British Virgin Islands	Sint Maarten
Madagascar	Myanmar (Burma)	Cayman Islands	Suriname
Malawi	Pakistan	Colombia	Trinidad and Tobago
Mauritius	Philippines	Costa Rica	Turks and Caicos Islands
Mayotte	Saudi Arabia	Curacao	United States
Nigeria	Singapore	Dominica	US Virgin Islands
Republic of Congo	Sri Lanka	Dominican Republic	Venezuela
Reunion	Taiwan	Ecuador	
Senegal	Thailand	El Salvador	**OCEANIA/PACIFIC ISLANDS**
Seychelles	Timor	French Guiana	American Samoa
Sierra Leone	Vietnam	Grenada	Cook Islands
South Africa	Yemen	Guadeloupe	Federal States of Micronesia
Sudan		Guatemala	French Polynesia
Tanzania	**EUROPE**	Guyana	Kiribati
Uganda	France	Haiti	New Caledonia
Zimbabwe	Italy	Honduras	Papua New Guinea
		Jamaica	Samoa
		Martinique	Tokelau
		Mexico	Tonga
		Montserrat	
		Nicaragua	

infection [30]. The median age of individuals infected was 48 years, with a range of 20-78 years. The study also identified 94 cases that were reported from different states and districts [30]. In total, the specimens that tested positive for Chikungunya were submitted from 25 states in addition to the District of Columbia.

In other areas of the world such as the La Réunion Island in the Indian Ocean, attack rates of the Chikungunya virus had reached 80-90% between 2005-2006 [31]. Prevalence of the disease on the islands as well as the prevalence among pregnant women were of special interest. In July of 2006, the prevalence rate in La Réunion was found to be 34.3%, which was determined by a regional surveillance-system managed by the Cellule Interrégionale d'Epidémiologie [31]. Attack rates were observed with the upsurge of the La Réunion Island outbreak, either using a clinical declaration of suspected cases (16.5%), or a rapid serum survey in pregnant women (18.2%) [31].

In India, specifically the state of Orissa, outbreaks of Chikungunya were recorded from 13 of the 30 districts of Orissa spanning 33 revenue blocks and 78 villages [32]. Attack rates ranged from 0.4 to 50.76% in the different villages. The first major outbreak occurred during the spring, but the majority of cases were reported from September to November (2006 and 2007). These outbreaks coincided with late monsoon season [32]. This correlation was brought up to signify that rainy season attracts more mosquitoes, which may have increased the incidence of the virus. The data was collected by age and sex distribution of the cases in the state of Orissa. The data confirmed the emergence of the Chikungunya virus infection in the state of Orissa in 2006 and its spread to a wider geographic zone in a short period of time. This spread was facilitated by the presence of the *Aedes* vector species [32]. The likelihood of global spread is increasing, and measures must be taken to improve the recognition and control of the disease. Education among health care providers and the public is essential [28].

Disease Burden

The Chikungunya virus has been identified in nearly forty countries [33]. See Table 2 for a list of countries with confirmed Chikungunya cases. Chikungunya was first identified in Tanzania in 1952, and has led to numerous outbreaks across Africa, Europe and the Americas [33]. The most recent outbreaks were documented in Réunion and Mauritius, India and coastal Italy [33]. To date, there has been a total of 11,792 confirmed cases of autochthonous

transmitted cases of Chikungunya in the Americas [33]. In addition to those, 666 cases have been reported through importation [34]. The growing prevalence of the Chikungunya virus is adding to the already substantial disease burden. According to Centers for Disease Control and Prevention (CDC) the average length of illness from an acute Chikungunya virus is 7 days [35]. The acute illness is followed by a substantial potential for continued morbidity. The time lost at work and the extra health care expenditures to treat the virus and the lingering co-morbidities equates to a huge burden on the societies and individuals affected.

The disease burden of the Chikungunya virus exists for both individuals affected, and the communities they live in. The Chikungunya virus causes a wide range of acute manifestations, such as arthralgia, rash, fatigue, fever and myalgia [31]. In addition to those acute manifestations listed, the virus can also present with cerebral disorders including but not limited to: altered mental status, encephalopathy, disrupted behaviour etc. These manifestations have the potential to greatly affect the quality of life of those infected. Research has shown that two years after acute infection with the Chikungunya virus, 43%-75% of infected people reported prolonged or late-onset symptoms that were highly attributable to the Chikungunya virus [36]. The long-term health implications of the virus show that the disease burden to the individuals affected lasts well beyond the acute manifestations.

As discussed above, the Chikungunya virus has the ability to cause substantial long-lasting morbidity for those infected. This reality puts a burden not only on the health care system, but also the work force in the communities affected. There are four categories of cost to take into consideration when estimating disease burden [37]. These categories include public health costs, individual and household costs, business costs and national development costs [37]. Public health costs are costs associated with disease prevention, public health education and information, public health inspections, as well as the costs associated with spraying or fogging for vector control. Individual costs include those costs incurred as a result of disease treatment, subsequent morbidity management and lost wages, in addition to the costs associated with purchasing material for personal prevention of vector-borne illnesses. Business costs are attributed mostly to loss of productivity related to sick time or modified workload assignments to match employee capacity. National development costs can be explained as the income lost due to the Chikungunya virus. An example of this would be the loss of expected revenue from tourism in Caribbean countries where the Chikungunya virus is gaining national attention [37]. The tourism industry has consistently put emphasis on health, security and safety as key factors in the promotion of profitable and sustainable tourism [38]. The loss of health and safety related to risk of disease may have the ability to impact National development to a significant degree.

Recent studies have shown that the Disability Adjusted Life Years (DALYs) lost during the 2006 epidemic in India totalled 25,588 with an overall burden of 45.26 DALYs per million people [39]. Among the DALYs reported, persistent arthralgia accounted for 69% of the total DALYs. The financial burden to families and companies affected during these epidemics is substantial. In the same study, it was noted that the financial burden of the productivity loss due to income foregone was estimated at 391 million Indian Rupees converted to over 6 million USD in India alone [39]. This figure may seem insignificant when the economy of India as a whole is taken into perspective, but is certainly worth noting considering the potential for the economic burden it can pose to smaller and low income countries which can be enormous.

The potential for the Chikungunya virus to spread across major parts of the world is frightening yet realistic possibility. The Dengue virus, another vector-borne illness has created a substantial economic burden, and may give researchers insight into what to expect with Chikungunya. Worldwide, estimates of the cost of medical care, surveillance, vector control, and loss of productivity related to the Dengue virus infections have totalled $39 billion USD [40]. The recent arrival of the *Aedes aegypti* mosquito in the Americas proves that the Chikungunya virus can no longer be considered a third world disease, but a global problem that requires the attention of physicians, scientists and public health authorities to prevent further disease burden [40].

Prevention

Although vaccinations for the Chikungunya virus do not exist in the market today, there are various other preventative measures that can be taken in order to minimize the spread of the virus. The only action to be taken in regards to prevention of this disease is vector control. Vector control is very difficult to attain but remains the most important method used to prevent the rapid spread of Chikungunya [41,42]. Vector control includes not only preventing mosquito bite but also ensuring that the infected person does not get bitten by mosquitoes within the first week of infection. CDC puts heavy emphasis on treatment of an infected patient in the first week of the viral infection. During the first week of infection, an individual has the potential to infect any subsequent mosquito that bites them, increasing the probability of further human transmission through the newly infected mosquitoes.

Preventative measures such as sleeping with long-sleeved shirts and long pants, sleeping with mosquito nets covering your bed, or sleeping with the air conditioning cooling system turned on can also help to decrease the transmission. These measures are recommended as they are effective and easy to do. Likewise, bug sprays and various mosquito repellents are exceptional in preventing mosquito bites while enjoying the outdoors. Repellents that contain ingredients such as DEET (Diethyl-meta-toluamide), picaridin, IR3535, lemon and eucalyptus oil and paramethane-diol products provide long lasting protection [43]. Education also plays an important role in disease prevention. Knowledge of when mosquitoes infected with Chikungunya tend to be most active is helpful when planning outdoor activities. Day time biting has been related to an increase in the risk of an outbreak and also makes it more difficult to keep the infection confined to one demographic. The last preventative technique worthy of mention includes checking the surroundings of homes and buildings to make sure that there is no stagnant water surrounding the premises where people live. Mosquitoes require water in order to progress through their life stages, therefore eliminating these sources can help to decrease the prevalence of mosquitoes around your property. Mosquito eggs may hatch in as quickly as one or two days therefore, constant vigilance is required. The *Aedes aegypti* mosquito can also be identified by its white markings on the legs, as well as a marking in the form of a lyre on the thorax [44-48].

Recommendations for Health Policy and Practice

Development of health policy and practice guidelines could

yield positive benefits by incorporating preventative measures in a coordinated manner. Although prevention may seem simple, the World Health Organization (WHO) concluded in its report titled "Guidelines for Prevention and Control of Chikungunya Fever" that "there is no available expertise or standard guideline for the proper surveillance, clinical case management, control and prevention of Chikungunya fever" [9,49]. As a result, the WHO has proposed six steps, needed to implement further understanding of the epidemiology of Chikungunya, and also for implementing an improved policy on its prevention and regional containment [52]. The six components listed include strengthening surveillance system for prediction, preparedness, early detection and response to Chikungunya outbreaks; improvement in early case detection and case management of Chikungunya fever; Integrated Vector Management (IVM); social mobilization and communication; partnerships; and operational research [51,52].

With the hopes of creating a viable preventative technique that can diminish the prevalence of Chikungunya, the goals of the World Health Organization is clearly stated. Whether these measures will be successful in preventing the disease will be unknown for years. Currently, steps should be taken to continue viral surveillance and containment. The implementation of preventative measures will hopefully, decrease the incidence of Chikungunya, along with the added benefit of protecting the population from another mosquito borne illness such as Dengue and most recently the Zika virus. According to the CDC, there have been 544 cases of the Zika virus in the United States to date [53]. However, all 544 reported cases are travel-associated and there is no locally acquired vector-borne cases [53]. Regional prevention measures, as with the Chikungunya virus, should be incorporated into the United States health system in order to prevent the possibility of the Zika virus disease becoming a locally acquired vector-borne disease. The Zika virus, like Chikungunya and Dengue viruses, is transmitted through the *Aedes aegypti* and *Aedes albopictus* mosquitoes [54]. The estimated range of the *Aedes aegypti* and *Aedes albopictus* ranged from the Southern United States to the Midwest and North Eastern United States [55]. Based on this information the recommendations discussed above should be incorporated in all regions throughout the United States, as well as all areas of the globe within the range of the *Aedes aegypti* and *Aedes albopictus* mosquitoes.

Conclusion

Chikungunya has spread to over 40 countries worldwide since the discovery and there have been explosive outbreaks globally which remains predominantly confined to Southeast Asia and Central Africa. Public health global initiatives should be focused on these areas in an effort to decrease the spread of the virus to neighboring continents. Although still confined, the threat of the Chikungunya virus has increased, due mainly to global travel. Although the infection is self-limiting, morbidity is high in major outbreaks. A large outbreak would have widespread personal, financial and societal implications. Transmission can be decreased mainly through vector control. Currently, no vaccine to prevent the disease exists. Routine educational campaigns to help spread awareness of the Chikungunya virus should focus on the early signs of the infection as well as the preventative measures that will help to reduce transmission. Community empowerment is crucial for the prevention and control of the virus as well as the containment of future outbreaks.

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