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***Aedes* spp mosquitoes and emerging neglected diseases of Kenya**

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Abstract

In the recent past, Kenya has seen an upsurge in diseases outbreaks caused by arboviruses especially dengue, chikungunya and riftvalley which have led to economic loss in terms of morbidity and mortality sometimes in both animals and man like in the case of riftvalley fever. Climatic change, unplanned urbanization combined with efficient vectors are some of the factors believed to be driving the emergence. Going by the current trend, an increase in the number of outbreaks is foreseeable in the future. The greatest risk of these arboviruses comes from their ability and potential to adopt from sylvatic zoophilic cycles to urban or peridomestic transmission cycles involving highly efficient and anthropophilic vector, *Ae. aegypti*. To exacerbate the problem, most of these arboviruses have no licensed vaccines and third world countries like Kenya still lack policies for the prevention and control of these diseases. There is also the reason to believe that viruses like Yellow fever and Zika virus have the potential for urban transmission. This could have devastating public health consequences, especially due to the fact that most city dwellers are not vaccinated and thus have no herd immunity. More emphasis therefore needs to be put in understanding the factors for emergence of these diseases in the local environments, their future trends and what actions could be taken to prevent and control them locally.

Keywords: *Aedes sp*, arboviruses, chikungunya, dengue, rift valley fever

Introduction

Aedes spp mosquitoes and especially *Aedes aegypti* are the main vectors of several viruses including chikungunya [1], dengue [2], yellow fever [3] and Zika [4] mainly in the tropical and semi tropical regions of the world. West Nile virus has also been isolated from *Aedes aegypti* in the field [5] whereas various *Aedes spp* the main vectors of rift valley fever virus [6-8].

***Aedes aegypti* origin**

The anthropophilic *Ae. aegypti* that prefers to feed primarily on humans and to breed in domestic (urban) and peridomestic environments [12] is believed to have originated from West Africa where it evolved from the sylvan form, *Aedes formosus* [9-11]. Unlike its Sylvan ancestor, *Ae. aegypti formosus*, that remains in forested habitats where its larvae develop in treeholes and is mainly zoophilic [13, 14], *Ae. aegypti* is not only highly anthropophilic but highly urbanized and has extensively colonized the expanding urban habitat. These factors coupled with the tendency of *Ae. aegypti* to feed on multiple hosts during one gonotrophic cycle [12] make *Ae. aegypti* an efficient vector of various arboviruses.

***Aedes spp* in Kenya**

Aedes spp mosquitoes are widely spread in Kenya, *Aedes ochraceus* are most abundant in the arid northeastern province [15, 135] where they have been implicated in the transmission of rift valley fever [16, 17]. They are also common in Nyanza [15] and Coast counties like Tana-river [16]. *Aedes mcintoshi* another major vector of rift valley fever is also common in North eastern [15, 16, 17] and the coast [17] whereas *Aedes circumluteolus* predominates Western Province especially in Budalangi [15].

Aedes aegypti the mosquito whose vectorial efficiency is believed to have led to the emergence of permanent endemic cycles of urban DENV and chikungunya virus (CHIKV), as well as seasonal interhuman transmission of yellow fever and Zika viruses, is also widespread in the Country [15, 18-23]. It exists in two forms, the anthropophilic domestic, light coloured

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Aedes aegypti aegypti and the and sylvatic, dark coloured *Aedes aegypti formosus* [18, 19]. Whereas *Aedes aegypti aegypti* is common in the urban centers, *Ae. aegypti formosus* is documented to occur in vegetated ecosystems in western Kenya near Kisumu City and in Kakamega forest [19]. Notably, both forms have been found to exist in sympatry along the Kenyan coast at Rabai [18].

Wherever *Ae aegypti* occurs in the Country, significantly higher numbers are found in the long rains than during the dry season and short rains [19]. They are also found both inside houses [20] and outdoors [21] with the outdoor numbers increasing significantly in the afternoons than in the morning hours [22, 23]. Higher numbers of *Ae. aegypti* are found in the urban areas, upto 3 times more, than in rural areas [22]. Water holding containers especially buckets, drums, tires, pots and jerrycans in the outdoors have been documented to provide high numbers of breeding sites to *Ae. aegypti* in the urban areas [24, 25].

These characteristics have major implications for the possible transmission of arboviral diseases and for the planning of surveillance and control programs [22]. It's clear that arbovirus outbreaks are likely to occur in towns than in the rural areas whereas source reduction efforts targeting the outdoor and indoor containers could be a cost effective way of reducing such outbreaks [24, 25].

In almost 3 decades, Kenya has had multiple outbreaks of arboviruses transmitted by *Aedes sp* mosquitoes resulting in economic and public health distress. These include the yellow fever outbreak in 1992 [26, 27] dengue fever in 2013 [28] 2014 [25] and 2017 [29], chikungunya fever in 2004, [30] and 2018 [31] and Rift Valley fever (RVF) in 1997 [32], 2006-2007 [33], 2014-2015 and 2018 [34]. Worryingly, these arboviruses often remain undetected by health care systems and much remains unknown about the true prevalence of arboviruses in Kenya [35, 36].

Dengue fever

This is an illness caused by infection with any of four types of viruses (DENV-1, DENV-2, DENV-3, DENV-4) belonging to the *Flaviviridae* family [38] transmitted by *Ae. aegypti* [2]. It's characterized by fever, headache, retro-orbital eye pain, myalgia, arthralgia, minor hemorrhagic manifestations, and rash [37]. Although most dengue patients in endemic zones recover within one week, there is a high likelihood of 5 to 10% progressing to severe dengue that presents in the form of dengue hemorrhagic fever (DHF) and dengue shock syndrome (DSS) [39, 40]. These are characterized by thrombocytopenia, plasma leakage due to increased vascular permeability, severe organ involvement, and/or clinically significant bleeding [39, 40]. Unfortunately, 0.1–10% of these severe dengue patients, will not survive.

The disease is endemic in more than 125 countries in the world mainly in Asia, Americas and Africa [41, 38] where approximately 3.6 billion people are currently at risk [37] and an estimated 390 million dengue infections occur annually [42]. It is therefore regarded as the most important re-emerging mosquito-borne disease globally.

The viruses were first isolated in 1943 and 1945 in Japan and Hawaii respectively [43] and the first documented outbreak in Africa occurred in Durban, South Africa in 1927 [44]. Dengue virus isolations in Africa were subsequently reported in

Nigeria (DENV1 and 2) in 1964–1968 [45], in Mozambique (DENV3) in 1983–85 [46], in Sudan in 1984 (DENV1 and 2) [47] and in Senegal 1986 (DENV4) [48]. The disease is currently endemic in 34 African countries [49].

In eastern Africa, the first outbreak was documented in Comoros in 1948 [50] with other outbreaks in the same country being reported in 1983 and 1984 [50]. Another major outbreak caused by DENV2 that affected more than 75% of the population was reported in the Seychelles Islands between 1977 and 1979 [51]. In Kenya, the first documented dengue outbreak also caused by DENV2 occurred in 1982 in the coastal cities of Malindi and Mombasa [52]. It was thought to have spread from the outbreak that had occurred in the Seychelles between 1977 and 1979 [51]. Then after almost 30 years, dengue outbreaks occurred in Mombasa in northern Kenya in 2011 [53] and subsequently in Mombasa city along the Kenyan coast in 2013–2014 [25, 28]. After that, sporadic outbreaks have been reported mainly in the N-Eastern Kenya and Mombasa county including in May 2017 [54].

Apart from the outbreaks, seroprevalence studies have documented the circulation of multiple dengue serotypes (DENV1-3) in Kenya whereby DENV1 and 2 are most dominant [28, 55, 57, 63]. This is consistent with literature suggesting that most epidemics in Africa are caused by serotypes 1 and 2 [49, 59] whereas infection with DENV4 is less common even though it has been documented in parts of Africa [48] and in Europe from travelers returning from Africa [60].

DENV is documented to circulate in 7 of 8 of the previous administrative provinces of Kenya, (all except Nairobi), with higher seroprevalence observed at the Coast and the lowest observed in Western province [56]. DENV1 has been reported to be the major cause of febrile illness amongst children in Western Kenya [61] and evidence of enzootic transmission of DENV has been observed in yellow baboons in Kwale County suggesting possible spillback from humans to baboons [63].

Risk Factors associated with DENV emergence in Kenya

The unplanned urbanization that has accelerated in Kenya and the rest of Africa has enhanced conditions for increase in density of *Ae. aegypti* and for efficient interhuman transmission.. Previous research in Kenya has shown that water holding containers found outdoors in most Kenyan towns provide 75% of the breeding sites to dengue vectors [25, 28] this coupled with the fact that *Ae. aegypti* prefers human habitations as a resting and host-seeking habitat and human blood [12] as both a protein source for oogenesis (egg development) and energy for flight, increases both the probability of becoming infected and the number of hosts infected by bite. For example the isolated outbreak that was reported in Nyali-B –a government dormitory in Mombasa- occurred at a time when residents were storing water in many open container types indoors [25] as the hostels had no piped water. This resulted in high CI, HI and BI increasing DENV outbreak risks.

Lack of sustained mosquito control programs. Unlike for malaria which has spelt out control programme under the National malaria control programme, Kenya lacks vector control programs targeting *Ae. aegypti* the main transmitter of dengue fever.

Globalization and increase in trade and travel, have

undoubtedly facilitated the spread of DENV strains and enhanced hyperendemicity.

Finally, Climatic conditions; Dengue outbreaks in Kenya usually occur during rainy seasons, the April-June 2013 and March-June 2014 dengue outbreaks coincided with the long rain seasons along the coast of Kenya. These rains result to increased aquatic habitats for *Ae. aegypti* breeding [65], thus increasing the vector population density and the risk of dengue transmission. However, drought also promotes vector abundance through increased storage of water in which *Ae. aegypti* mosquitoes breed [66].

Future threats and potential preventive measures to control DENV in Kenya

Currently, vector control and early detection of cases through continued surveillance remains the only viable method of preventing and controlling dengue as there is no licensed vaccine. Therefore, as dengue becomes endemic in Kenya, it's recommended that organized vector surveillance and control programs against *Ae. aegypti* mosquitoes be instituted in the major towns especially in Mombasa where most recent outbreaks have been reported. The activity should target *et al.* 1 container types with the potential to hold water [25, 28, 67]. The national and county governments should also strive to provide a reliable supply of piped water in every household. However, success of these efforts will require legislation and proper inter-agency (health and environment) coordination and funding, with the support of the national and county governments.

Seasonal variation should also be established to identify high risk times and facilitate appropriate public health responses. Also health care providers should increasingly be trained and made aware of the need to quickly detect infection and provide appropriate care to patients. The availability of rapid diagnostic kits at health facilities all over the country is also important.

Chikungunya fever

This is an illness caused by chikungunya virus (CHIKV), a small, enveloped, positive-sense, single-stranded RNA alphavirus in the family *Togaviridae* that was first isolated in Makonde province of the present day Tanzania in 1953 [68, 69]. The word chikungunya meaning "that which bends up" [69] describes the symptoms of the illness, which causes severe and persistent pain in the joints, high fever, and rash leading to significant morbidity with potential substantial effect on labor intensive industries including agriculture, manufacturing and tourism. Although the infection is self-limited and acute symptoms usually resolve within one–two weeks, CHIKV is recurrent in 30–40% of infected individuals and may persist for years [70].

The infection is endemic in Africa and Asia, where numerous outbreaks have been reported since the 1950s [71-77]. However, the world witnessed epidemics of epic proportions between the year 2004–2011 [87-93] when a CHIKV outbreak affected 75% of communities living in Lamu Island [79, 80] and Mombasa [81] in Kenya and eventually spread across the Indian Ocean to Comoros Island in 2005 [82], India [84] and Southeast Asia, reaching Myanmar in 2010 [80]. Sporadic imported cases and occasional outbreaks of CHIKV fever in other regions outside Africa and Asia such as Italy in 2007 [83]

and in France in 2010 [85, 86] were also reported. In Africa, CHIKV is apparently maintained in a sylvatic transmission cycle involving primates and forest-dwelling *Aedes spp* mosquitoes [87]. These include *Aedes africanus* [88] in East Africa, *Aedes fuscifer*, *Aedes taylori*, *Aedes delzieldi*, and *Aedes luteocephalus* in West Africa [71, 85] and *Aedes taylori* and *Aedes codellieri* in South Africa [89, 80]. However, *Ae. aegypti* is the main vector of CHIKV in urban areas in East Africa [79, 80]. In contrast, transmission of CHIKV in Asia is mainly in the urban area by *Aedes aegypti* and *Aedes albopictus* [91, 92]. In addition to vector transmission, the vertical transmission of CHIKV from mother to foetus has been identified on Reunion Island [93].

Distinct CHIKV genotypes have been identified and comprise the East, Central and South African isolates [East–Central–South–African (ECSA)], West Africa isolates (West Africa), and Asian isolates (Asian) [94]. Isolates that caused the 2004-06 Indian Ocean outbreak form a distinct cluster within the large eastern/central Africa (ECSA) phylogenetic group, in addition to the Asian and West African phylogenetic groups [95].

In Kenya chikungunya outbreaks have greatly affected the Coast and the North Eastern region in the recent past [30,31] even though seroprevalence studies have shown evidence of chikungunya transmission in other regions that have never witnessed outbreaks before as well like Western Kenya [96-98]. Curiously, a certain study detected significantly higher CHIKV infections in the Lake Victoria region than at the Coast [96] even though vector competence has shown the *Ae. aegypti* from the Coast to be more susceptible to CHIKV infection than those from the Western region [98] and Nairobi [99]. Concurrent circulation of DENV and CHIKV have also been documented in Kenya [97] just like in other African [100] and Asian Countries [101, 102].

Risk Factors associated with emergence of CHIKV in Kenya

CHIKV shares many characteristics with DENV, including some of the factors associated with its emergence such as: the use of the highly peridomestic and anthropophilic *Ae. aegypti*, as its principal vector in urban areas this combined with poorly planned urban centers riddled with problems like inefficient supply of tap water and waste management leads to outbreaks. For example, Mombasa is the second largest city in Kenya with approximately 1.2 million inhabitants. The city has a rapidly growing population, and some areas experience overcrowding, numerous open dump sites, inadequate drainage, stagnant water and ample breeding sites for mosquitoes. These factors make Mombasa particularly vulnerable to chikungunya outbreaks.

Future threats and potential preventive measures to control CHIKV in Kenya

It's clear that the increase in human populations in Kenyan cities like Mombasa have improved the conditions for endemic maintenance of chikungunya just like dengue. Therefore, as long as vector surveillance and control programs are not put in place in such towns, outbreaks of chikungunya fever will persist. Also, the risk of transmission to unaffected areas cannot be ruled out. There is a high potential for its establishment especially in inland towns like Nairobi, Kisumu

and Kakamega since Mombasa is a popular tourist destination and a sub-regional transportation hub with connections to Rwanda, Tanzania and Ethiopia, This means there are large numbers of travellers between the inland Kenya and Coastal regions who are likely to help in transmission of Chikungunya leading to the greatest risk of establishment of CHIKV in the inlands, This might cause thousands of cases during outbreaks which might lead to overstretching of the healthsystems and economic losses in future, It's therefore recommended that organized vector surveillance and control programs against *Ae. aegypti* mosquitoes be instituted in the major towns especially in Mombasa where most recent outbreaks have been reported, Such control programs already exist for malaria,

Yellow fever

Yellow fever virus (YFV) is a mosquito borne flavivirus that causes infections in human beings with symptoms ranging from mild non-specific illness to severe disease with jaundice, haemorrhage, and death ^[103], Just like DENV and CHIKV, Yellow fever is a *Flavivirus* indicated by strong historical and phylogenetic evidence to have originated in Africa before spreading to the rest of the world through the slave trade ^[104-107], It's currently endemic in tropical areas of Africa and Central and South America, The virus is primarily transmitted by mosquitoes of the species *Aedes* and *Haemagogus*-in the new world ^[108-110], The mosquito, the true reservoir of YF, is infected throughout its life, and can transmit the virus transovarially through infected eggs, In Africa, YFV is transmitted in the forests and the surrounding savannah between non-human primates (NHP) and a range of sylvatic mosquito species like *Ae formosus*, *Ae africanus* and *Ae simpsoni* ^[108-112], Human yellow fever epidemics on the other hand arise irregularly when the chain of virus transmission overlap with the anthropophilic and fully domesticated *Ae aegypti* on the fringes of the forest, Since *Ae. aegypti* prefer densely populated urban areas, the transmission pattern soon changes from a forest or jungle cycle to an urban cycle characterised by rapid human to mosquito to human transmission ^[111, 112].

In Kenya, a yellow fever outbreak that infected 55 people and killed 34 was reported in Kerio valley from mid-1992 to march 1993, This outbreak came 50 years after the two reported cases in 1943 ^[26, 27, 111].

Entomological investigations showed that this was a sylvatic outbreak transmitted mainly by *Ae. africanus* and *Ae. Keniensis* ^[111], The outbreak involved predominantly young people from local villages who had exposure to the woodlands area of the valley where Vervet monkeys and baboons were found ^[26, 27, 112].

Since the 1992-93 outbreak, no other outbreak has been witnessed in Kenya and seroprevalence studies have also not identified any positive cases, indicating absence of recent YFV transmission ^[112, 113].

However, none vaccinated Kenyans are at a risk of contracting YF if they travel to Yellow fever endemic regions, For example, in March 2016, the National IHR Focal Point of Kenya notified WHO of 2 imported cases of yellow fever (YF) from Luanda Angola ^[114], None of the victims were vaccinated against yellow fever prior to travelling to Angola.

Risk factors associated with emergence of YF in Kenya

Increase in population density and rapid unplanned urbanization; increased population density in the rural areas may lead to encroachment into virgin forest lands by non-immune rural populations, This coupled with extremely rapid unplanned urban migration, to already densely populated cities, where high densities of *Ae. aegypti* – a major vector of YF co-exist with city dwellers who are mostly not vaccinated poses a ready recipe for an epidemic of massive proportion,

Future threats and potential preventive measures to control YF in Kenya

Even though no YF outbreak has been reported in East Africa, the risk that YF could emerge as an urban problem in Kenya should not be ignored, It's evident that Yellow fever epidemics may spread quickly in densely populated urban areas, as reported recently in Angola and the Republic of Congo ^[115],

Therefore mass vaccination campaigns, vector control and surveillance and prompt response to any suspected cases would be of great importance, It's evident that the single-dose vaccine available against YF is efficacious and can help to control and reduce yellow fever virus transmission substantially ^[116-120], Prompt response is also as this could lead to earlier detection of the emerging infection and perhaps limit its extent ^[112], Awareness campaigns; The immediate at-risk population should be educated on mosquito avoidance and control, They should also be made aware of the clinical syndrome of yellow fever while the health workers should be educated on the appropriate early collection of samples for diagnostic confirmation ^[114],

Rift valley fever

It is an arboviral disease that primarily infects livestock and humans, It's caused by the rift valley fever virus, a negative-sense, single-stranded RNA virus in the family *Bunyaviridae*. genus *Phlebovirus* ^[31], The name is derived from the great Rift Valley of Kenya, where the disease was first recognized, characterized and described after a highly fatal epizootic among sheep in a farm in Naivasha in 1930 ^[121, 122],

RVF outbreaks are usually associated with flooding and El Niño like prolonged and above average rainfall coupled with high humidity and increased vegetation cover as a result of the warming up of the surfaces of the Indian and Atlantic oceans ^[123], This leads to emergence of flood water *Aedes* sp e.g *Ae. mcintoshi* and *Ae. Ochraceus* some of which are infected with RVFV through transovarially infected drought, resistant eggs ^[16, 17 123], The infected female adults then initiate the transmission to nearby animals like goats, sheep and camels thus intensifying the transmission, Outbreaks occur when secondary vectors mainly the *Culex spp* take over the breeding sites and spread the virus ^[16, 123, 124].

Animals mainly get infected through infective mosquito bites whereas humans get severely infected when they come in direct contact with infected bodily fluids or tissues of infected animals during slaughter, food preparation, assisting with animal births, or conducting veterinary procedures or from the disposal of carcasses or foetuses ^[125, 126], This puts occupational groups such as herders, slaughterhouse workers and veterinarians at a higher risk of infection ^[125-128].

In humans, RVF is characterized by retinitis, encephalitis, and

hemorrhagic fever whereas in livestock the disease is characterized by abortions and perinatal mortality [129, 130], Epizootics and epidemics can result in massive loss of livestock, consequent export embargoes, and significant human morbidity and mortality, all of which can be economically devastating to affected areas [129, 130]. To add onto that, RRVFV has been studied as a potential agent of biologic warfare [131, 132] and according to the World Organization for Animal Health (OIE), RRVFV is a high-impact trans-boundary pathogen with potential for bioterrorism and a setback to international livestock trade [133]. Between 1951 and 2007, eleven national epizootics of RRVFV have occurred in Kenya in 4–15-year cycles [123, 124] with an expansion in geographic distribution with each epizootic [33]. This expansion has been driven by environmental factors like rainfall and temperature, the density and movement of livestock and the presence of competent vector species [16, 17, 31].

The last major RRVFV that occurred in Kenya was in 2006/2007 and led to over 150 human deaths and over 700 hospitalised. It also caused losses worth US \$ 32 million in terms of animal herd losses, vaccination costs and trade bans/ value chain ramifications [31, 135, 136]. This outbreak followed another that occurred in 1997–1998 that also affected Somalia, and Tanzania and caused over 450 human deaths in Kenya alone [30].

Apart from Kenya, epizootics have also been reported in other African countries including the 1977–1979 outbreak in Egypt that affected over 200,000 people and resulted in over 600 deaths [134]. In South Africa in 1951 that led to deaths of over 100,000 sheep and half a million livestock abortions [137], in Mauritania [139], Senegal, Sudan [140] and Madagascar. Epizootics have also been reported in the Arabian peninsula; Saudi Arabia and Yemen [138].

Risk factors associated with emergence of RRVFV in Kenya

Weather patterns and animal movements; RRVFV transmission is enzootic during most years between wildlife, such as African buffaloes, and a wide variety of mosquito species (>30 species, 6 genera) [16, 17, 127]. However, epizootic transmission occur in years that after droughts experience above average rainfall as a result of warming in the Indian Ocean linked closely to the El Niño-Southern Oscillation (ENSO) in the Pacific [30, 31, 128]. The outbreaks are predictive following widespread flooding that triggers the simultaneous hatching of large numbers of *Aedes* eggs, rapidly producing a cohort of blood feeding adults, some of which are infectious with RRVFV and able to transmit this infection to ruminants. Since this usually comes after drought, the outbreak happens when livestock are driven by herdsman to these new sources of water and grass, bringing susceptible hosts into close proximity with infectious mammal-feeding *Aedes* mosquitoes [16, 17, 127, 128].

Future threats and potential preventive measures to control RRVFV in Kenya

Wider geographical distribution of RRVFV have been recorded with every outbreak of RRVFV in Kenya since 1951 [31] demonstrating the characteristic ability of RRVFV to escape traditional enzootic areas and invade naive populations. Since this has been attributed to livestock movement and weather

patterns, it can only mean that with the current trend of increased livestock density and global warming, RRVFV will be endemic in most counties of Kenya in the coming years,

Additionally, it has also been established that before the onset of serious clinical disease, humans develop viremias suitable to infect susceptible mosquitoes [128]. This means that increased human travel by buses, air e,t,c which is largely uncontrolled could introduce RRVFV into other Counties like in western Kenya which have never recorded any RRVFV but where susceptible wild and domestic hosts and suitable vector mosquitoes reside,

Since RRVFV outbreaks are predictive, by tracking ENSO in the Pacific, vaccination campaigns implemented by both the county and national governments can help avert impending epidemics. The vaccination must be implemented prior to an outbreak because there is a high risk of intensifying the outbreak especially by animal health workers through the use of multi-dose vials and the re-use of needles and syringes particularly if some of the animals in the herd are already infected and viraemic (although not yet displaying obvious signs of illness),

Restricting and banning livestock movement and sale; this may be effective in slowing the expansion of the virus from infected to uninfected areas,

Establishment of an active animal health surveillance system to detect new cases, this could help provide early warning for veterinary and human public health authorities,

Vector control; Mosquitoes are the initial source of infections during RRVFV outbreaks, therefore larviciding measures at mosquito breeding sites could help especially if breeding sites can be clearly identified and are limited in size and extent. However, during periods of flooding, the number and extent of breeding sites is usually too high for larviciding measures to be feasible,

Public health education and risk reduction; the message should focus on reducing the risk of animal-to-human transmission as a result of unsafe animal husbandry and slaughtering practices. The locals should be discouraged from the unsafe consumption of fresh blood, raw milk or animal tissue. In the epizootic regions, all animal products (blood, meat, and milk) should be thoroughly cooked before eating. Awareness should be increased to practicing hand hygiene, wearing gloves and other appropriate individual protective equipment when handling sick animals or their tissues or when slaughtering animals. They should also be made aware of the importance of personal and community protection against mosquito bites through the use of impregnated mosquito nets, personal insect repellent if available, light coloured clothing (long-sleeved shirts and trousers) and by avoiding outdoor activity at peak biting times of the vector species.

Healthcare workers caring for patients with suspected or confirmed RRVFV should implement Standard Precautions when handling specimens from patients. These should cover the handling of blood (including dried blood), all other body fluids, secretions and excretions (excluding sweat), regardless of whether they contain visible blood, and contact with non-intact skin and mucous membranes.

It should be remembered that with a high potential impact on wildlife, domestic animal and human health, failure to contain a RRVFV outbreak could seriously impact veterinary and human

health in Kenya, Africa, middle East and the rest of the World and have far reaching economic consequences,

Other Arboviruses with the Potential for Urban Emergence

Zika virus (ZIKAV)

ZIKA virus is a flavivirus (*Flaviviridae: Flavivirus*) closely related to YFV and DENV, It was first isolated from a Rhesus monkey in the Zika forest of Uganda in 1947; a year later it was isolated from *Ae. africanus* at the same location [141], After that, it was sporadically isolated and occasionally associated with human disease in African countries like Gabon [142], Central African republic [143], Uganda, Nigeria, Senegal, Egypt and in Asian countries like India, Malaysia, Thailand, Viet Nam, the Philippines and Indonesia [144, 145], The virus is mainly transmitted by *Ae. aegypti* and *Ae. albopictus*, that also transmit other viral infections, including dengue virus (DENV), chikungunya virus (CHIKV), and yellow fever virus (YFV) [4, 147], It has also been isolated from *Ae. africanus* [141, 148] and *Ae. luteocephalus* in Africa [149], Disease symptoms of ZIKAV include flu-like illness associated with high fever, malaise, dizziness, anorexia, retro-orbital pain, edema, lymphadenopathy, and gastrointestinal manifestations and rash [150, 151], Even though seroprevalence of ZIKAV in endemic areas can be as high as 56-75% of the population, most of the infections remain asymptomatic [145, 146], However, the association of ZIKV infection with microcephaly, a condition that results in small heads and underdeveloped brains in infants and neurological complication (Guillain–Barré syndrome); yet, no specific treatment or vaccine for the disease exists [151, 152] has led to the latest early declaration of “Zika as a public health emergency of international concern” (WHO) [150, 152] So far, no case of Zika virus has been detected in Kenya despite the country’s proximity to the source of the pathogen transmitted by mosquitoes and primates [153], it is however, not known whether the current situation is the result of preventive measures or lack of diagnostics,

Risk factors

Globalization, with consequent increased travel and trade, rapid urbanization and growing weather variation events due to climate change has contributed to the recent unprecedented Zika virus (ZIKV) pandemic,

Future threats and potential preventive measures to control ZIKAV

Even though to date no outbreaks of ZIKAV have been reported, it’s important to note that ZIKAV originated from Africa which has the right ecological requirements for the virus like appropriate climatic conditions, flora and Fauna, However, in the case of an outbreak timely and practical Zika diagnosis and management might not be possible in most countries as health professional are ill [154, 155] prepared and there is limited guiding content on possible serological and other molecular markers arrays to enhance real time epidemiological vigilance [155-158], Therefore, there is need for capacity strengthening with focus on the laboratory facilities and human resources to be able to implement epidemiological surveillance and disease control, carry out accurate diagnosis and offer quality case

management during outbreaks, Also just as for the other arboviruses, eliminating breeding spots of the main vector *Ae. aegypti* in urban centers will not only control a potential ZIKAV outbreak but DENV and CHIKV as well,

Attention is also needed in the main entry points like bus stations, airports and seaports as these are gateways from the infected areas, The public should also be educated on prevention and control of ZIKAV, Pregnant women in any trimester should be discouraged from travelling to endemic areas, and guidelines on the symptoms and actions to take on onset should be provided to individuals who must travel to these zones,

Conclusions

For this review, we selected emerging human pathogenic arboviruses that are transmitted by *Aedes spp* because they are the primary arthropod vectors of arboviruses that have led to outbreaks in Kenya in the recent past, Arboviruses in Kenya have a well-documented history of emergence through several mechanisms, including geographic expansion aided by livestock movement for Rift valley fever, spillover from non-human primates to humans in the case of Yellow fever and enhanced amplification in the domesticated *Ae. aegypti* in the case of dengue and chikungunya aided by water storing containers in the urban areas, Whatever the mechanism, it’s clear that these arboviruses threaten to increase in the future due to increased rural to urban migration, human travel and global commerce as well as deforestation, Global warming also has the potential to increase the distribution of vectors and to enhance transmission potential,

The greatest risk of these arboviruses comes from their ability and potential to adopt from sylvatic zoophilic cycles to urban or peridomestic transmission cycles involving highly efficient and anthropophilic vector, *Ae. aegypti*, This coupled with the explosions in urban human populations in the tropics Kenya included, have a potential to enhance the transmission of diseases such as DEN and CHIK which unfortunately have no licensed vaccines, There is also the reason to believe that viruses like Yellow fever and Zika virus have the potential for urbanization which could have devastating public health consequences, especially due to the fact that most city dwellers are not vaccinated and thus have no herd immunity, Research on how to devise intervention strategies to facilitate their control especially in the urban areas is therefore necessary and timely,

Author contributions

EAO wrote the article

Competing interest

The author declares that she has no competing interests,

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