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Chikungunya fever in Africa: a systematic review

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ABSTRACT

Since the identification of chikungunya virus (CHIKV), sporadic cases and outbreaks were reported in several African countries, on the Indian subcontinent, and in south-east Asia. In the last 20 years, there is a growing number of reports of CHIKV infections from African countries, but the overall picture of its circulation at the continent level remains ill-characterized because of under-diagnosis and under-reporting. Moreover, the public health impact of the infection in Africa is generally poorly understood, especially during outbreak situations. Our work has the aim to review available data on CHIKV circulation in Africa to facilitate the understanding of underlying reasons of its increased detection in the African continent.

KEYWORDS

Chikungunya virus; serology; africa; arbovirus; epidemiology

Introduction

Chikungunya fever: an African viral illness

Chikungunya Fever (CHIKF) is a viral disease, caused by the chikungunya virus (CHIKV), characterized by fever, rash, and incapacitating arthralgia [1]. The infection is asymptomatic in 3–25% of the patients [2], and the case-fatality rate is <1 per 1,000 cases [3]. CHIKF may cause long-lasting joint pain (up to 3 years), especially in older adults [4–7], possibly related to the immune response due to the persistence of viral nucleic acids (but not infectious virus), which could trigger persistent local immunopathology [9]. Moreover, CHIKV may infect human osteoblasts and, thanks to its cytopathic effect, could directly contribute to the joint pathology and erosive disease [10]. Fetal infection is extremely rare, but CHIKV infection may have a direct impact on pregnancy with a higher risk of abortion in the first trimester [1]. Maternal infection close to delivery may facilitate vertical transmission, leading to severe disease (i.e. encephalopathy and long-term sequelae) in half of infected newborns [3,11]. Though some broad-spectrum antivirals such as favipiravir showed some efficacy in mouse models, there is currently no approved antiviral treatment available [12], and the same applies for vaccines [13].

CHIKV is an arbovirus (i.e., arthropod-borne virus), belonging to the *alphavirus* genus of the *Togaviridae* family [1], transmitted to humans by the bite of *Aedes* spp (mostly *Ae. aegypti* and *Ae. albopictus*) female mosquitoes. Although *Ae. aegypti* is the main vector of CHIKV, *Ae. albopictus* (the 'tiger' mosquito) is becoming important in the emergence of CHIKV in temperate areas, where *Ae. aegypti* is not commonly detected. Since the virus identification in the Newala district of Tanzania in 1952–53 [14], three different genotypes of CHIKV have been identified: the Western African, the East-central-south African (ECSA), and the Asian genotype [1,15]. Evolutionary studies have suggested an African origin for CHIKV [16]. The Asian lineage, which caused epidemics in the 1950 s, probably split into two clades: an Indian clade, which likely went extinct, and a South-East Asian clade [16]. The Asian lineage was not identified during the 2005–2006 epidemic in India, consistently with the hypothesis of a lack of sustainability of the human-mosquito cycle at a local scale in the absence of continued importation [16,17].

CHIKV cycle in the tropics

In west and central Africa, CHIKV is maintained in a sylvatic cycle involving wild non-human primates (NHPs) and forest-dwelling *Aedes* spp mosquitoes. However, there is little information on the vertebrate hosts involved in viral maintenance [18]. Both humans and wild NHPs, throughout the humid forests and the semi-arid savannahs of Africa, have significant levels of antibodies against CHIKV, with small-scale outbreaks following a 3–4 years cyclical pattern consequent to the repopulation of susceptible, nonimmune, wild NHPs [19]. In rural regions, outbreaks are more likely in the heavy rainfall season, when sylvatic mosquito density tends to increase. In Asia, the predominant vector is the urban, peri-domestic, anthropophilic *Ae. aegypti* mosquito, which is responsible for large-scale outbreaks characterized by long inter-epidemic periods, which may last several decades [17,18]. Because a

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vertebrate reservoir or a sylvan transmission cycle has not been identified in Asia, it cannot be excluded that the virus persists only in a human-mosquito-human cycle [18]. However, this is still uncertain, since outbreaks of CHIKF in Asia have not been necessarily linked to outbreaks in Africa, which suggests an independent evolution of an African ancestor of CHIKV in Asia and the possibility of a sylvatic cycle maintaining autochthonous virus genotypes in the Asian continent [15].

CHIKV spread outside Africa

Since its identification, sporadic cases and outbreaks of CHIKV infection were reported in several African countries, on the Indian subcontinent, and in south-east Asia, where it exhibits a peculiar pattern of spread, with successive epidemics detected along an eastward path [1,20]. CHIKV has recently reemerged, causing a series of large outbreaks, which started in Kenya in 2004 and ravaged the Comoros Islands, the island of La Réunion, and other islands in the southwest Indian Ocean in early 2005, followed by an epidemic in the Indian subcontinent in 2005–2006 [21,22]. In February 2011, CHIKV hit New Caledonia and started to spread in the Pacific, following multiple imported cases [23,24]. In December 2013, autochthonous CHIKV cases were reported for the first time from the Americas, where the Asian genotype caused major outbreaks in Saint Martin and in other Caribbean islands, and then spread to Latin America countries [25]. CHIKV caused sporadic cases, clusters, and outbreaks also in southern Europe: in Italy, more than 300 cases were reported in the summer of 2007, and about 400 in 2017 [26,27]; in the south of France, two autochthonous cases of CHIKF were identified in the summer of 2010, and 12 cases in 2014 [28,29].

Recently, the occurrence of CHIKF outbreaks in Central Africa, which is assumed to be the original niche of CHIKV, has apparently increased. Whether this is a real increase or just the consequence of improved surveillance remains to be defined. The determinants of the phenomenon also need to be discussed.

Materials and methods

CHIKV outbreaks in endemic areas of Africa: search methods

We used the following string search in PubMed and Global Index Medicus: (Chikungunya OR CHIKV) AND (Africa* OR Afrique) and found 677 and 34 items, respectively (last search 31 January 2020). Articles mentioning the CHIKV African (Western or ESCA) lineage spread outside Africa were excluded, and we focused on the last 20 years. After screening, we retained 61 articles about CHIKV in African countries. Any article of interest that was not found by the string search was also considered. We looked for latest information on Chikungunya in Africa in online gray literature including ProMed and all weekly bulletins on outbreaks and other emergencies from the WHO regional office for Africa, available online since beginning 2017 (https://www.afro.who.int/health-topics/disease-outbreaks/outbreaks-and-other-emergencies-updates?page=1).

Results

Epidemiology of CHIKV in the African region

After the first known outbreak in Tanzania in 1952–53, during which the CHIKV was isolated for the first time [14], the virus spread significantly during years '60–80 causing outbreaks in many African countries: South Africa (1956; 1975–77), Zimbabwe (1957; 1961–62; 1971), Democratic Republic of Congo (DRC) (1958; 1960), Zambia (1959), Senegal (1960; then limited and recurrent outbreak until 1997–98), Uganda (1961–62; 1968), Nigeria (1964; 1969; 1974), Angola (1970–71), Sierra Leone (1978), Central African Republic (CAR) (1978–79) [18,19,30]. After this period, there was no evidence of CHIKV circulation in African countries until years 1999–2000, when a large urban epidemic occurred in Kinshasa, DRC, affecting about 50,000 persons [31]: CHIKV isolates belonged to the ECSA genotype, being closer to Central than to East-South African lineage isolated more than 20 years before; this allowed to hypothesize a persistent and unrecognized virus circulation [31], as further confirmed by virological diagnosis of CHIKV infection among clinically suspected (laboratory unconfirmed) yellow fever cases during 2003–2012 in DRC [32]. In 2002 and 2006 few cases of CHIKV infection were reported in Equatorial Guinea [33], as well as during an outbreak of yellow fever that occurred in Sudan in 2005 [34]. In 2004, a large epidemic started on the coast of Kenya with estimates as high as 13,500 infections [35], then spreading to the city of Mombasa and to the Comores, La Réunion, Seychelles and Mauritius islands in the Indian Ocean during 2005–2006 with significant impact on public health [18,22]: in January 2006, a small related outbreak of CHIKV (and Dengue) was also reported from north-east coastal Madagascar (15 confirmed cases/55 tested samples) [36], whereas a retrospective serosurvey conducted among pregnant women during 2009, showed a 5.3% (N = 66/1,244) IgM anti-CHIKV seroprevalence [37]. After 2005, a resurgence of CHIKV transmission was reported from several African countries with outbreaks of chikungunya (and dengue) virus involving major cities, some of them recently colonized by *Ae. Albopictus*, that played a major role in the virus spread in Cameroon and Gabon during the years 2006 and 2007, respectively [38; 39,40]. The 2007 outbreak in Gabon was particularly large,

affecting more than 20,000 persons in the city of Libreville [38], and the virus continued to circulate in the country up to 2010 [41], affecting also villages in the southern deep forest region [42]. A prospective study conducted during 2007–2008 in northern Tanzania, among febrile hospitalized patients, detected CHIKV infections in 7.9% (N = 55/700) of the cases [43]. In June 2011 a large CHIKV outbreak (affecting more than 8,000 persons) started in Brazzaville, Republic of the Congo [44,45]. In 2012, a 6-month prospective study performed in Sierra Leone (Bo province) among febrile patients aged >5 years, detected IgM anti-CHIKV in 42.8% (N = 400/932) of the cases [46]. A prospective study conducted during 18 months (Jan 2014-July 2016) in Kenya among febrile children reported CHIKV infection as the cause of fever in 8.3% (n = 32/385) of the cases [47], whereas no CHIKV cases were virologically diagnosed among 489 febrile adults enrolled in a cross-sectional study during 2014–2015 in coastal Kenya [48]. A small outbreak was detected in Senegal in 2006 (6 confirmed cases) and 2015 [49]: a previous large study performed during 2009–2013 among febrile individuals in south-eastern Senegal reported CHIKV as the cause of the fever in only 0.1% (N = 16/13,845) of the cases [50]. Recently, large CHIKV outbreaks were reported in Kenya (the attack rate in Mandera was estimated to 80%) and in the bordering areas of Somalia in 2016 [51]. This was the first time Somalia reported confirmed cases of Chikungunya virus, highlighting improvements in laboratory capacity. Kenya experienced again an outbreak in 2017–2018, with 453 infections in the Mombasa county, including 32 laboratory-confirmed cases [52]. From August 2018, a large outbreak with over 13,000 suspected cases was reported in Sudan. Since November 2018, Brazzaville and Kinshasa, the capitals of the Republic of the Congo and the DRC, respectively, separated only by the Congo river, are experiencing a very large outbreak that has registered over 11,000 suspected cases in the Republic of the Congo, and over 1,000 cases in DRC [53,54]. Most recently, a large outbreak occurred from May to December 2019 in Ethiopia and registered over >50,000 suspected cases [54].

Seroepidemiological studies in Africa

The literature on the seroepidemiology of chikungunya virus in Africa is quite scarce. Since 2000, epidemiological studies based on CHIKV serology have been performed in several African countries. CHIKV serology performed by ELISA (enzyme-linked immunosorbent assay) and/or IF (immunofluorescence) in endemic areas may not be specific because of possible cross-reactivity with other viruses (i.e. O'nyong-O'nyong virus, ONNV); thus, a confirmation with plaque reduction neutralization tests (PRNT) is needed to distinguish CHIKV infection from other arboviral infections. Therefore, only serological studies using PNRT as confirmation test have been taken into account in this review (Table 1, Figure 1). An epidemiological substudy of an HIV-survey conducted in rural areas of Cameroon during the period 2000–2003 showed a high circulation of CHIKV, with 46.5% (N = 119/256) of anti-CHIKV-IgG positivity among adult participants [55], that was confirmed by a smaller cross-sectional retrospective study conducted in 2007 in rural areas of Western Cameroon showing a CHIKV-IgG seroprevalence of 49.5% (N = 52/105) [56]. A 6-month retrospective study conducted among pregnant women who underwent delivery during 2006 in Cotonou, Benin, reported a high seroprevalence for anti-CHIKV IgG (N = 127/351, 36.1%) without a case of vertical transmission, possibly because the infection was not acute or recent [57]. Then, a seroprevalence

Table 1. CHIKV: main seroepidemiological[a] studies in sub-Saharan African countries.

Country	Years	Study population, N	Seroprevalence %	Reference
Angola	1970-71	589	13.7%	
Benin	2006	351	36.2%	[57]
Cameroon	2000-03	256	46.5%	[55]
	2007	105	49.5%	[98]
Djibouti	2010-11	914	2.6%	[62]
Democratic Republic of Congo	2015-16	342	26.4%	[58]
Guinea	2006	47	8.5%	[67]
Kenya	2000-03	419	36.5%	[61]
	2009-12	370	0.2%	[64]
	2010-12	500	67%	[59]
	2015-16	385	8.3%	[47]
Madagascar	2010	1,244	5.3%	[37]
Mozambique	2013	55	26.4%	[65]
	2015-16	112	28.6%	[66]
Nigeria	2008	285	50%	[63]
Republic of Congo	2011	517	34.4%	[44]
Senegal	1997-98	447	35.3%	[99]
	2009-13	13,845	0.1%	[50]
Sierra Leone	2012-13	932	42.9%	[46]
Sudan	2016	379	1.8%	[68]
Tanzania	2007-08	700	7.9%	[43]
Zimbabwe	1961-62	44	86%	[100]

[a]Anti-CHIKV IgG positivity confirmed by neutralization tests.

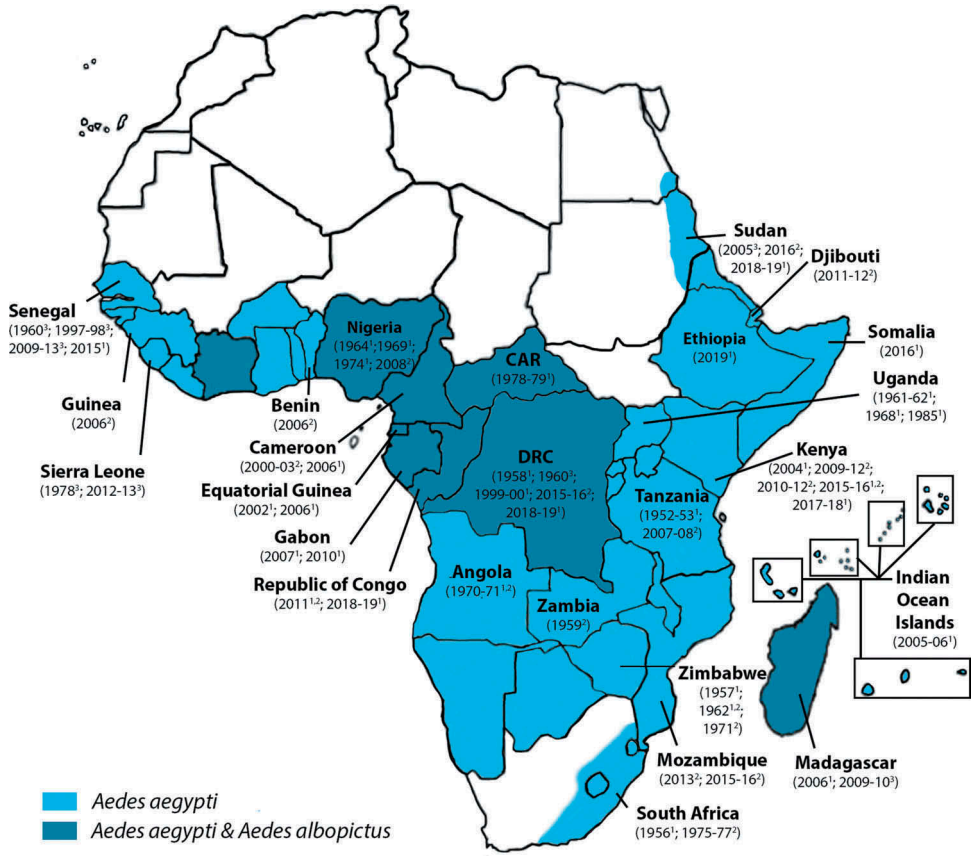

Figure 1. Chikungunya virus in Africa: epidemiology and vector distribution.
[1] CHIKV virology; [2]IgG anti-CHIKV confirmed by neutralization tests; [3]IgM anti-CHIKV only.

retrospective study conducted during the 2011 outbreak in the Republic of Congo, among blood donors (N = 517), showed that more than one out of three (34.4%) individuals was CHIKV-IgG positive, suggesting previously unrecognized circulation of the virus in the country [44]. A similar estimate (26.4%) was found in neighboring Kinshasa, Democratic Republic of the Congo in 2015–2016 [58]. A cross-sectional study conducted in rural Western Kenya, involving 500 asymptomatic individuals (50% adults, 50% children <14 years) recruited during the years 2010–2012, showed a very high prevalence of anti-CHIKV-IgG positivity (N = 335/500, 55.8%), with adults having higher seroprevalence than children (78.7% *vs* 42%) [59]: the study area had not been affected by CHIKV outbreaks in the 10 years before, allowing to hypothesize a sustained virus transmission during interepidemic periods [59]. A household survey involving 1,864 individuals conducted in coastal Kenya in 2009 showed a seroprevalence of anti-CHIKV IgG of 1.3% (N = 25/1,864), with 168 (9%) individuals having positivity to both anti-CHIKV and anti-ONNV IgG for which PRNT could not distinguish between CHIKV and ONNV exposure [60]. Furthermore, a previous retrospective study on serum sample of pregnant women collected during 2000–2003 from a coastal Kenyan district (close to Mombasa) showed an anti-CHIKV IgG seroprevalence of 36.5% (N = 153/419) [61], allowing to hypothesize the CHIKV circulation (together with other arboviruses) in coastal Kenya also before the 2004-CHIKV outbreak. A household arboviral seroprevalence study conducted in 2010–2011 in Djibuti showed an anti-CHIKV IgG seroprevalence of 2.6% (N = 24/914) [62].

Seroprevalence of anti-CHIKV IgG among symptomatic individuals has been performed in few studies conducted in Africa. In 2008, a 6-month retrospective study among febrile individuals conducted in the Borno State, Nigeria, reported a 50% (n = 143/285) seroprevalence of anti-CHIKV IgG, with 91.6% (n = 131/143) of co-infections (malaria, typhoid fever, other flaviviruses) [63]. A cross-sectional hospital-based study conducted during the years 2009–2012 among febrile patients in three different sites in Kenya reported CHIKV infection as the cause of fever in only 0.2% (N = 1/379) of the cases [64]. Two cross-sectional seroprevalence studies conducted in Mozambique, among febrile patients recruited in Maputo (2013) [65] and in eight different localities in central and northern Mozambique (2015–16) [66], showed similar anti-CHIKV-IgG prevalence (26.4%, N = 55/208 [65]; 28.6%, N = 112/392 [66]), suggesting a significant and unrecognized virus circulation in all the country. Similar results, even though characterized by lower prevalence, were found also in peripheral cities of Guinea-Conakry, where 8.5% (N = 4/47) of the febrile

patients resulted positive for anti-CHIKV-IgG [67]. Lower seroprevalence of anti-CHIKV IgG (1.8%; N = 7/379) were found among febrile outpatients from eastern and central Sudan during 2012–13 [68].

Molecular diversity of CHIKV in Africa

RNA virus populations such as CHIKV do not consist of a single genotype but an ensemble of related sequences, termed *quasispecies*. Exploration of sequence space is most of the times detrimental for the virus (e.g. leading to error catastrophe) but few times leads to beneficial mutations that may enhance the epidemic potential of a specific variant [8]. CHIKV life cycle includes the virus/vector interaction, where anatomical barriers in the mosquito may pose population bottlenecks [69]. These bottlenecks in the mosquito can be overcome thanks to frequent recombination events in the 3′-untranslated region (UTR), which is made of sequence repeats that are conserved but whose numbers vary by viral lineages. These recombination events accelerate CHIKV adaptability and therefore may favor host switch [70,71]. To better understand these complex interactions, virus evolutionary trajectories can be retrospectively characterized after the emergence of new epidemic variants. CHIKV variants that caused a large epidemic since 2004, spreading from Kenya toward Indian Ocean Islands, the Indian subcontinent, and south-east Asia, belong to the ECSA genotype [1,72,73]. During this epidemic, two different lineages were identified – the Indian Ocean sublineage and the India sublineage that emerged independently from coastal Kenya and later spread to Southeast Asia, Italy, and France -, suggesting independent introductions of CHIKV strains from Kenya into Indian Ocean Islands and India [15,74]. A viral variant, presenting a substitution of the amino acid alanine with valine in the position 226 of the E1 glycoprotein (E1:A226 V), was selected during the epidemic that originated in the Indian Ocean and became predominant in specific areas where *Ae. albopictus* was the most commonly detected *Aedes* species, such as La Réunion and the Kerala district in south-India [75]. Overall, the presence of the E1:A226 V mutation is associated with CHIKV transmission mainly mediated by *Ae. albopictus*, whereas when absent *Ae. aegypti* remains the main vector [76]. Thus, a single amino acid substitution may have influenced vector specificity, increasing the fitness of CHIKV for specific vector species and, consequently, for its transmission [77]. Interestingly, this mutation – shown to have emerged through convergent evolution in at least four occasions [78] – has never been detected in any Asian genotype of CHIKV strain, possibly because of a negative epistatic interaction with a threonine at position E1-98 [79]. This threonine is invariant in Asian-lineage strains, whereas all ECSA strains have an alanine at the same E1 position, which has a neutral effect on the fitness of the E1:A226 V substitution [80]. Other studies suggested the role of a second mutation (E2:I211 T), together with E1:A226 V, in the virus adaptation to *Ae. albopictus* vector [15]. CHIKV isolated during the 2016 outbreak in Kenya, where *Ae. aegypti* is the main vector involved in viral transmission, two mutations in E1 and E2 viral glycoproteins (E1:K211E and E2:V264A) have been detected in the background of the *wild type* E1:226A virus [81]: both mutations have been previously associated with increased infectivity, dissemination and transmission in *Ae. aegypti* from north-India [76], with no impact on virus fitness for the *Ae. albopictus* vector [76,78]. Outbreaks in 2016 and 2017 in Pakistan, India and Italy revealed a divergent variant of the Indian Ocean sublineage, with wild type phenotype at position 226 of the glycoprotein E1, but other adaptive mutations such as two above mentioned mutations E1:K211E and E2:V264A, and in addition, E1:I317 V and E2:G205 S, possibly playing a role in conferring competence to *Ae. Albopictus* [82; 83,84].

Phylogenetic analyses of CHIKV from African countries show that the virus circulation is mainly restricted to ECSA genotype: in particular ECSA1 circulating in Southern-East Africa, ECSA2 in Central Africa, and ECSA3 in Kenya and Indian Ocean Islands [53]. From 2000, many CHIKV variants belonging to the ECSA2 lineage were reported, among others, from DRC, Cameroon, Gabon and Equatorial Guinea [33,40,56,85]. Their close genetic relationship over a vast territory suggests the continuous circulation of CHIKV in central Africa. The CHIKV ECSA2 strains may be phylogenetically distinguished in two groups: the first (Group A), isolated in Angola and CAR prior to 1984, and the second (Group B), isolated from Gabon (2007), Cameroon (2013), and Republic of the Congo (2011) [53]. The CHIKV ECSA2 Group B possesses the recently derived E1:A226 V mutation, whereas Group A seems to lack this mutation, with the exception of the variant isolated during the ongoing outbreak in Republic of the Congo (RC_Diosso_2019 strain) [53]. The ECSA3 genotype isolates from Kenya and Comores during 2004–2005 were related to strains from Tanzania, South Africa, CAR and DRC [72].

Discussion

The epidemics of CHIKF that occurred in Africa and elsewhere since 2004 demonstrated how easily the virus could spread, causing major public health implications at national and international levels. Several factors likely contributed to the explosive spread, including the changing distribution of the vectors responsible for transmitting CHIKV in Africa, with a dominance of *Ae. albopictus* over *Ae. aegypti* in some rural/peri-urban and, to a lesser extent, urban areas (i.e. Yaoundé, Cameroon [86]), and viral adaptation to the competent vectors. In West and Central Africa, CHIKV is maintained in an enzootic cycle involving wild NHPs and forest-dwelling *Aedes* mosquitoes such as *Ae.*

furcifer, Ae. taylori, Ae. luteocephalus, Ae. africanus and *Ae. neoafricanus* [19,87,88]. In 1990, *Ae. albopictus* was first detected in the African continent [89] where *Ae. aegypti* was already present [90]. Then, *Ae. albopictus* rapidly appeared and spread in several African countries [91,92], possibly because of its capacity to predominate in the competition with *Ae aegypti* where both mosquitoes circulate [93,94]. This changing vector distribution overlapped with the observation of increased CHIKV transmission, including in urban and peri-urban areas in Africa. Overall, CHIKV circulation in Africa in the last 20 years is restricted to ECSA genotype that has shown a significant genetic plasticity, leading to the appearance of mutations associated with increased vector fitness according to the *Aedes* species circulating: the CHIKV E1:A226 V variant associated with increased fitness to *Ae. albopictus* (with no impact on fitness to *Ae. aegypti*) [77], and the mutations E1:K211E and E2:V264A associated with increased fitness to *Ae. aegypti* (with no impact on fitness to *Ae. albopictus*) [76,78]. Interestingly, the CHIKV E1:A226 V variant caused the outbreak propagated by *Ae. albopictus* in north-eastern Italy in 2007 [26]; however, a CHIKV strain *wild type* E1:226A caused the 2017 outbreak in Central Italy and the subsequent secondary outbreak in the south of the country [27]. Surprisingly, no difference in vector competence and fitness was found by experimental infection studies conducted on Italian *Ae. albopictus* mosquitoes, using different CHIKV variants (i.e. those with and without the E1:A226 V mutation) [95]. Thus, there is a need to increase the availability of virologic data, coupled with vector competence studies, from different parts of the world where CHIKV is circulating.

In the last 20 years, there is a growing number of reports of CHIKV infections from African countries, possibly depending on improved surveillance systems, including increased laboratory capacity, that has been observed in few African countries. Despite these advances, the understanding of CHIKV circulation is likely to currently be underestimated at the continent level. The diagnosis of CHIKV infection remains challenging in many resource-limited settings, including African countries. In fact, the clinical picture of the CHIKV infection may overlap with other endemic diseases in these countries, and molecular studies to detect adaptive mutations of CHIKV variants from Africa are often lacking. In fact, the clinical picture of the CHIKV infection may overlap with other endemic diseases in these countries, including malaria and other arboviroses (e.g. Dengue). Moreover, the self-limitation of the majority of the clinical cases, as well as the poor access to diagnostic laboratory exams (serology and virology), may allow under-diagnosis and consequent under-reporting in African settings. Furthermore, although the increasing number of CHIKV outbreaks worldwide, data on vertical transmission and fetal diseases are scarce and lacking from African countries, with the exception of La Réunion and Mayotte islands [96]. A systematic review and metanalysis on mother-to-child-transmission of CHIKV showed a risk of vertical transmission of 15.5%, with a pooled risk of symptomatic neonatal disease and death of 15.3% and 2.8%, respectively, mainly related to intrapartum transmission [96]; moreover, newborn infection may be symptomatic or asymptomatic, with long-term neurodevelopmental delays occurring in 50% of neonatal symptomatic infections [96]. Overall, the public health impact of CHIKV infection, especially during outbreak situations, is difficult to measure in many African countries where the vast majority of the economy is informal, there is a lack of well-organized national blood-transfusion systems, and diagnostic capacity is limited, and the infant mortality and morbidity are already high because of other endemic diseases as well as poor quality of health access and services.

The occurrence of CHIKV outbreaks in multiple areas of Africa, along with the global spread of the virus, which has caused epidemics in several areas of the Old and the New World raises questions about the usefulness of safe and effective vaccines to contrast an illness characterized by low mortality but very high morbidity, especially in tropical areas. Several vaccine candidates are now under human trials [13]. Whether a vaccine against CHIKV should be considered a priority to decrease the burden of disease also in Africa is surely a matter of debate.

Conclusions

The increased occurrence of large CHIKF outbreaks in Africa (where the virus is endemic), although globally under-diagnosed and under-reported, might be explained, to some extent, by a series of factors, such as urbanization, increased number of susceptible people, changes in vector ecology and distribution, virus adaptations to the main local vector, as well as improved surveillance activity in some areas.

Disclosure statement

We have no competing interest to declare.

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