Climate change could shift disease burden from malaria to arboviruses in Africa

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Abstract

Malaria is a longstanding public health problem in sub-Saharan Africa, while arthropod-borne viruses (arboviruses) like dengue and chikungunya cause an underrecognized burden of disease. While many human and environmental drivers affect the dynamics of vector-borne diseases, here we argue that the direct effects of warming temperatures are likely to promote greater environmental suitability for dengue and other arboviruses transmitted by Aedes aegypti while reducing the suitability for malaria transmitted by Anopheles gambiae. Environmentally-driven changes in disease dynamics will no doubt be complex and heterogeneous, but given that current public efforts are targeted to malaria control, we encourage the public health community to consider Aedes aegypti and dengue, chikungunya, and other arboviruses as potential emerging public health threats in sub-Saharan Africa.

Keywords

Africa; malaria; Anopheles gambiae; Aedes aegypti; dengue virus; chikungunya virus; Zika virus; temperature; climate change; transmission

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The global health community has expressed growing concern that climate change will alter the distribution and burden of vector-borne diseases, potentially reversing the gains of control programs and expanding the threat of emerging diseases (1–4). Malaria still imposes a major burden of morbidity and mortality in sub-Saharan Africa (228 million cases and 405,000 deaths in 2018), despite recent intensive control efforts that have succeeded in reducing transmission in many locations (5–7). At the same time, many other vector-borne diseases, including Rift Valley fever, dengue, chikungunya, yellow fever, Zika, o’nyong’nyong, West Nile, leishmaniasis, river blindness, and African sleeping sickness, circulate regularly in humans, wildlife, and livestock in sub-Saharan Africa, although their burden is less well characterized (8–12). For example, over 27,000 cases of arbovirus infections transmitted by *Aedes* vectors have been reported in West Africa since 2007 (13).

It is now well established that temperature has nonlinear effects on vector-borne disease transmission, and that different mosquito and parasite species differ in this response, resulting in differences in their thermal optima and limits (2,4,14–19). As a result, the direction and magnitude of the effects of climate change on transmission of specific vector-borne diseases will differ across geographic regions.

In this Personal View, we summarize and visualize published data to make the case that climate change, in conjunction with urbanization, is likely to drive a shift in most of sub-Saharan Africa from climates most suitable for malaria transmission by rural *Anopheles* spp. mosquitoes to climates more suitable for transmission of dengue and other arboviruses by *Aedes aegypti* mosquitoes, with major consequences for public health and disease control strategies. Specifically, we draw from three lines of evidence: transmission models fit from laboratory thermal performance data; independent data on human infection; and widespread existing distributions of *Aedes aegypti*, dengue, and chikungunya in sub-Saharan Africa. While the drivers of vector-borne disease dynamics are multifaceted and include human mobility, rainfall and water storage practices, urbanization, and others, the increasing temperature suitability for arbovirus transmission merits attention from the global health community, in tandem with ongoing efforts toward malaria control. Therefore, although we cannot conclusively predict changes in disease incidence based on temperature alone, we argue that the effects of temperature change will promote arbovirus transmission and increasingly limit malaria transmission by rural vector species in much of sub-Saharan Africa, acting in concert with urbanization and other changes.

**Transmission models**

Climate change will affect vector-borne disease transmission because temperature affects vector population size, survival, biting, pathogen incubation rates, and vector competence, with potential additional effects via rainfall and humidity (15–17,20). The physiological effects of temperature on the vector and pathogen traits that drive transmission are well-established from laboratory experiments and field studies (20–24). Both ectotherm physiology theory and data from a wide variety of ectotherm taxa and traits demonstrate that the thermal responses of development, survival, and reproduction are often unimodal, peaking at intermediate temperatures and declining at both low and high temperatures (25–27). Laboratory experiments confirm that these nonlinear thermal responses are pervasive across mosquito and pathogen taxa and traits (15–17,21,28–30). We previously developed...
temperature-dependent $R_0$ models that incorporate empirically measured effects of temperature on mosquito biting rate, immature survival probability, immature development rate, adult lifespan, fecundity, vector competence (probability of becoming infectious following exposure to an infectious blood meal), and parasite development rate, and in turn on mosquito population size, for malaria transmitted by *Anopheles gambiae* (and, where data were not available, from traits derived from other *Anopheles* spp.) (15,31) and for dengue, chikungunya, and Zika transmitted by *Aedes aegypti* (16,17). Because the *Ae. aegypti* temperature-dependent $R_0$ relationships were very similar for all three viruses (16,17), we hereafter focus on results from the dengue model. For both malaria and arboviruses, vector and parasite traits and $R_0$ peak at intermediate temperatures and are suppressed at both low and high temperatures (2,15–18,28). The thermal optima and ranges for transmission vary by vector and parasite species: malaria transmission by *Anopheles gambiae* peaks at 25°C, while arbovirus transmission by *Aedes aegypti* peaks at 29°C (15–17,31) (Fig. 1: lines). Multiple vector and parasite traits contribute to differences in the thermal response of transmission across species (32).

**Independent data on human infection indicate potential for shifts in disease burden**

Field data from both mosquito-based metrics of transmission risk (e.g., entomological inoculation rate) (15,37) and in human incidence and at local and continental scales (16,36) strongly support nonlinear effects of temperature on transmission predicted from laboratory studies and mathematical models. Recent work from our cohort study of febrile children in four villages in Kenya showed a unimodal relationship between blood smear positivity for malaria and temperature, with a peak at 25°C (30-day average temperature, lagged by one month: the time scale at which we expect temperature to affect transmission) and a sharp decline in smear positivity above the optimum temperature (Fig. 1; filled circles) (36). This result strongly supports the independently predicted 25°C optimum from the temperature-dependent malaria $R_0$ model (Fig. 1; solid line) (15). In the same study, non-malarial fever, much of which is caused by dengue and chikungunya, increased with temperature throughout the observed temperature range, supporting the relatively warm thermal optimum of dengue (Fig. 1; dashed line and open triangles). As further evidence for the physiological constraints on malaria and arbovirus transmission, previous studies supported the thermal optima predicted from mechanistic models. First, a study of dengue in 20 cities in Colombia showed a unimodal relationship between incidence and weekly average temperature (multiple time windows and lags were explored) that peaked at a mean temperature of 28°C (38), supporting the model-predicted optimum for dengue transmission of 29°C (16). Second, the predicted unimodal effects of temperature on transmission, peaking at 25°C for malaria and 29°C for dengue, chikungunya, and Zika viruses (also transmitted by *Aedes aegypti*), are supported by continental-scale data on entomological inoculation rate in Africa and human incidence in the Americas, respectively (15–17).
Shifting climate suitability for malaria and *Aedes aegypti*-transmitted viruses

As climate change leads to warming temperatures, the intermediate thermal optima for vector transmission have two immediate implications. First, for all vector-borne diseases, climate change will drive increases in some regions and decreases in others, depending on current and future local climates relative to the optimum and thermal limits for disease transmission. Second, the relative suitability for different vector-borne diseases will shift: the climate may simultaneously become more suitable for some diseases and less suitable for others. In regions where temperatures are regularly between 25–29°C, including much of sub-Saharan Africa, a warming climate will become less suitable for malaria but more suitable for dengue, chikungunya, and other arboviruses transmitted by *Ae. aegypti* (Fig. 2). Specifically, the highest density of people exposed to high temperature suitability for transmission (the ‘risk hotspot’) for malaria is projected to shift toward higher elevations such as the Albertine Rift region and higher latitudes in Southern Africa (Fig. 2A–C, red circles). Meanwhile, the risk hotspot for dengue, chikungunya, and other *Aedes aegypti*-transmitted arboviruses is predicted to expand from West Africa throughout sub-Saharan Africa (Fig. 2D–F).

In conjunction with climate change, urbanization is driving widespread changes in habitat, microclimate, and human populations and is occurring more rapidly in sub-Saharan Africa than anywhere else in the world (though these transitions can be complex and diverse) (40,41). Urbanization affects vector-borne disease transmission by altering the availability of vector breeding habitat and contact with humans. *Aedes aegypti* mosquitoes breed in human-made container habitats such as discarded tires, cans, buckets, and water storage containers, all of which increase in density in urban areas but are also present in villages (10,42,43). By contrast, *Anopheles gambiae* and some other African malaria vectors breed in naturally occurring pools of water, which are more common in rural areas, although malaria transmission can also occur in cities (44,45). In addition to affecting breeding habitat, urban areas form ‘heat islands’ with microclimates that are several degrees warmer than surrounding vegetated areas, which can directly influence vector development and survival (46) and may benefit warmer-adapted *Aedes aegypti* over *Anopheles gambiae* mosquitoes. Urbanization may therefore act synergistically with warming climate to promote the shift from *Anopheles*-transmitted malaria to *Aedes*-transmitted arboviruses in sub-Saharan Africa.

Widespread distribution of *Aedes aegypti* and arboviruses in sub-Saharan Africa

Although shifts in climate suitability do not necessarily translate into a shift in disease burden from malaria to dengue and other arboviruses in sub-Saharan Africa, mounting evidence supports this hypothesis. First, for expansions in transmission to occur, *Aedes aegypti* mosquitoes and arboviruses must be present in the region. Growing evidence suggests that the vectors and arboviruses are already widespread and under-recognized in sub-Saharan Africa, in part because of misdiagnosis and a public health focus on malaria.
and Anopheles vectors (Table 1) (10,13,33–35,42,47–53). For example, recent arbovirus surveillance work in Kenya in regions of high malaria endemicity (Fig. 1) showed that ~10–20% of febrile children were positive for dengue virus infection for much of the year (Fig. 3A), and that Aedes aegypti mosquito vectors were abundant in and around households year-round (Fig. 3B; see Supplementary Materials, page 3, for Methods). These data suggest ongoing endemic transmission of dengue in at least four geographically distinct Kenyan populations. Recently, large chikungunya epidemics have also occurred in Mombasa, Mandera, and Lamu, Kenya (54–56) and in the Kassala state of Sudan, where heavy rains flooded a major river, sparking an outbreak (57). Growing evidence suggests that both endemic and epidemic transmission of dengue, chikungunya, and other Aedes-transmitted arboviruses regularly occurs in sub-Saharan Africa, though it may be undiagnosed or misdiagnosed as malaria (Table 1) (13,33,58–61). At the same time that the arbovirus threat is increasingly recognized in sub-Saharan Africa, malaria has declined dramatically in the last two decades (5–7). While the drivers of this decline are undoubtedly complex, and much has been attributed to the success of malaria control programs, it is also possible that some of the decline results from decreasing climate suitability due to increasing temperature. The extent to which warming temperatures have already reduced malaria transmission remains to be assessed because few have recognized that the optimum for malaria transmission is as low as 25°C (62).

**Discussion**

The degree to which changes in climate suitability for transmission translate into changes in the landscape of disease depends on other factors that shape disease dynamics, including pathogen exposure history, housing type, vector control and public health efforts, rainfall, and human mobility (70–73). Exposure history is particularly important because newly occurring transmission in naïve populations may more sharply increase the burden of disease than increases in already endemic populations with some acquired immunity (74,75). Therefore, even if climate change leads to geographic shifts rather than net increases in populations at risk of disease (Fig. 2), these shifts are not neutral from a public health perspective, and may be disruptive to populations, healthcare systems, and economies that have not historically experienced either malaria or arboviral diseases. Within endemic regions, the interannual variability and seasonality of transmission could further change in response to changing rainy seasons and their interaction with temperature (72,76,77). At the same time, changes in demography, population growth, migration, and socioeconomic conditions may mitigate the impacts of climate change on vector-borne disease dynamics (70,78,79). However, climate places limits on where transmission can and cannot occur regardless of population characteristics and may exacerbate effects of changing social vulnerability to disease.

Mosquitoes and parasites are not static threats but evolving organisms that respond to ecological conditions and selective pressures imposed by their changing environments. The potential for mosquitoes to adapt to warming temperatures by increasing their thermal optima and limits remains unknown (80). Mosquitoes quickly and repeatedly evolve resistance to insecticides when vector control programs impose strong selective pressure (81). However, temperature-driven selection on mosquitoes may not align with selection on
the parasites they transmit. At warm temperatures, mosquito longevity is the major limitation on transmission because short pathogen incubation periods and frequent biting cannot overcome declining mosquito lifespans to sustain transmission (4,15,31). But even short-lived mosquitoes may achieve high fitness at warm temperatures if rapid development and high fecundity outweigh the cost of shorter lifespans. As a result, selection may not lead to increased mosquito survival at high temperatures, and therefore evolution may not rescue vector transmission as temperatures exceed current thermal optima.

Even if temperatures become warm enough to drive existing populations extinct or to suppress their ability to transmit disease, warmer-adapted mosquitoes (including *Aedes aegypti* and *Anopheles stephensi*, an urban malaria vector in India (82)) could invade and replace current *An. gambiae* populations transmitting malaria in Africa. Incipient speciation has already occurred in Africa in *Ae. aegypti* subtypes (83,84) and in the *An. gambiae* species complex (85–88), suggesting that both can adapt to changing ecological conditions including urbanization and, potentially, climate. *Aedes albopictus*, another arbovirus vector, is also present in some regions of Africa, and where it co-occurs with *Ae. aegypti* it can be competitively dominant (65–67). Temperature-dependent *R*₀ models suggest that *Ae. albopictus* has a cooler thermal optimum (26°C) and upper thermal limit (32°C) than *Ae. aegypti*, which could limit the expansion and transmission potential of this species under warming climates (16,89). Climate-driven ecological and evolutionary changes in mosquito communities that might alter the direct physiological effects of temperature are therefore highly uncertain.

Although many aspects of the changing environmental and population landscapes that shape disease transmission remain unknown, we have outlined three lines of evidence suggesting that climate change, in concert with urbanization, will drive a shift in disease transmission in sub-Saharan Africa from malaria to arboviruses like dengue and chikungunya in the next few decades (Fig. 2). First, temperature-dependent transmission models predict increased suitability for *Aedes*-transmitted arboviruses and decreased suitability for malaria (Fig. 1) (15,16). Second, large-scale entomological and human disease data and local human incidence data provide evidence that warming temperatures above thermal optima drive declines in transmission (Fig. 1) (15,16). Third, at the same time that malaria is declining in much of sub-Saharan Africa, arboviruses and *Aedes aegypti* already pose an underrecognized public health burden, which could expand under increased climate suitability (Figs. 2–3; Table 1).

Malaria has already declined precipitously in much of Central and South America and the Caribbean in the last three decades at the same time that dengue, chikungunya, and Zika have exploded to cause half a million to >2 million cases per year (www.paho.org) (90–92). The drivers of these disease trends are almost certainly complex and multivariate, including multiple aspects of environmental and human population change. Nonetheless, the correspondence between shifting temperature suitability predicted from laboratory data and models and the observed shifts from malaria to dengue and other arboviruses is striking. For example, in a country-scale analysis of arbovirus transmission in Latin America and the Caribbean from 2014–2016, weekly mean temperatures averaged 25.6°C across the region (range in weekly average temperature from 21.5 – 28.7°C across countries),(16) spanning
the range where malaria transmission peaks and begins to decline while arbovirus transmission increases steeply with temperature (Fig. 1).

Disease control strategies that are effective against malaria—such as long-lasting insecticide-treated bednets, indoor residual spraying, and artemisinin combination therapy—are ineffective against dengue, which uses the day-biting and container-breeding Aedes aegypti mosquito as its primary vector (93,94) and currently has no specific drug therapy or broadly effective vaccine available (the development and roll-out of the Sanofi-Pasteur dengue vaccine has had mixed results (95,96)). A shift from malaria to dengue in sub-Saharan Africa would therefore require public health efforts to retool to control an ecologically different vector and pathogen, a shift that has already taken place throughout much of the Americas. In particular, the development of accurate point-of-care diagnostics for dengue and chikungunya viruses and community-based vector control will be increasingly important for targeted care and prevention of arboviruses (13,34,35,53). While malaria eradication efforts remain critical, given the year-round circulation of dengue and chikungunya and abundance of Aedes spp. mosquitoes in Africa, public health efforts should also prepare for a potentially growing threat of arboviral disease in Africa.

**Supplementary Material**

Refer to Web version on PubMed Central for supplementary material.

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Search strategy and selection criteria

This Personal View primarily summarizes evidence from our own work and that of collaborators. The references in Table 1 were selected based on our own reading of the peer-reviewed literature in English, suggestions from two anonymous reviewers, and Google Scholar searches of “arbovirus,” “dengue,” “chikungunya,” “Aedes aegypti,” or “Aedes albopictus,” and “Africa” performed in May 2020. They were chosen based on providing some evidence of arbovirus or vector presence in Africa, and are not an exhaustive list.
Key Messages

- Malaria transmission by *Anopheles gambiae* peaks at 25°C while dengue transmission by *Aedes aegypti* peaks at 29°C, based on mechanistic transmission models parameterized and validated with laboratory and field data. Warming temperatures in the tropics are expected to favor transmission of dengue over malaria.

- Independent data on human infections of malaria and dengue support the predicted nonlinear effect of temperature on disease incidence. In tropical regions, where temperatures regularly hover around 25°C, warmer temperatures correspond to a decrease in malaria incidence and an increase dengue and chikungunya incidence.

- Dengue, chikungunya, and their *Aedes aegypti* mosquito vector are already widespread but under-recognized in Africa, based on studies of vector abundance, human serology, and acute infections from across Africa. As climate suitability increases for arboviruses, these diseases could expand and overtake the public health burden of malaria.

- While malaria control efforts remain critical, arbovirus control through increased surveillance and testing capacity and vector control of container-breeding, day-biting *Aedes aegypti* is a critical emerging public health need in Africa. Testing and diagnostic capacity for arboviruses, as well as awareness of vector ecology and exposure risk, lag well behind that of malaria in most of sub-Saharan Africa, where climate change is expected to promote dengue and other arboviruses.
Figure 1. Malarial and non-malarial fever among Kenyan children from 2014–2018 versus temperature, overlaid on basic reproduction number curves for malaria and dengue. Points represent proportion of children with positive malaria smears (filled circles) and proportion of children with non-malarial fever (open triangles) over temperature. Land surface temperatures at each participant visit were calculated as 30-day mean temperatures lagged by one month (the time window in which we expect temperature to affect transmission), specific to each of the four clinic sites. Proportions were calculated at 1°C intervals of temperature (x-axis) at each of the four different outpatient clinic sites in western and coastal Kenya where children with undifferentiated fever were recruited, for up to four points per temperature bin (12,33–36). Lines represent predicted basic reproduction number ($R_0$ rescaled to range from zero to one) for malaria (solid line) and dengue (dashed line) as a function of temperature from ecological models based on laboratory mosquito and parasite data (15–17). For methods detail, see Supplementary Materials, pages 2–3.
Figure 2.
Temperature-driven malaria risk hotspot (red circles; top row [A-C]) shifts to high elevations in East Africa while *Aedes aegypti*-transmitted arbovirus risk hotspot (red circles; bottom row [D-F]) expands throughout sub-Saharan Africa from current (left column [A, D]) to 2050 (middle column [B, E]) to 2080 (right column [C, F]). Color scale indicates the number of months per year predicted to have highly suitable (relative $R_0 > 0.5$) temperatures for transmission, multiplied by population density (log(1 + population density)), for a scaled index of person-months of high risk for transmission. Temperature suitability for transmission is based on the upper 50th percentile of relative $R_0$ from temperature-dependent $R_0$ models (15,16). All climate projections are based on the business as usual climate scenario RCP 8.5, using the HadGEM2-ES General Circulation Model. The red circles indicating hotspots are shown to ease visualization of the areas of highest person-months of risk. An aridity mask (gray) blocks out regions that are too dry for malaria transmission (39). This figure is intended to illustrate one possible scenario of temperature-driven risk, rather than making a specific prediction about future disease burden, which additionally depends on moisture availability, human population growth and mobility, and other factors. For Methods details, see Supplementary Materials, pages 4–8.
Figure 3. High rates of dengue virus infection in febrile children (A) and consistently high abundance of *Aedes aegypti* mosquitoes (B) in four villages in Kenya suggests that arboviruses are an underrecognized public health burden.

The rates of dengue positivity (A) are measured as the percentage of children <18 years of age with undifferentiated febrile illness attending outpatient care who tested positive by PCR or IgG ELISA for dengue virus infection (69). Data were compiled from four different clinics in western and coastal Kenya during each calendar month between 2014 and 2018. *Aedes aegypti* abundance (B) was measured as the monthly average number of *Aedes aegypti* eggs per household recovered from ovitraps placed in and around houses. Error bars indicate standard errors of the mean. For Methods, see Supplementary Materials, page 3.
Table 1.
Evidence for *Aedes aegypti* vectors, arbovirus transmission, and over-diagnosis of malaria across sub-Saharan Africa.

| Location                        | Evidence                                                                 | Reference |
|---------------------------------|--------------------------------------------------------------------------|-----------|
| Kenya (western)                 | Dengue infection in children                                             | (33)      |
| Kenya (coastal)                 | Dengue and West Nile virus transmission in children and adults           | (63)      |
| Kenya                           | Acute flavivirus and alphavirus infection in children                    | (35)      |
| Kenya                           | Serological evidence of arboviral infection in children                  | (51)      |
| Kenya (coastal)                 | O’nyong Nyong virus and chikungunya virus transmission                   | (52)      |
| Kenya (western)                 | O’nyong Nyong virus and chikungunya virus transmission                   | (64)      |
| Kenya                           | Chikungunya infection in febrile children                                | (53)      |
| Kenya (Mombasa)                 | Chikungunya outbreak                                                     | (54)      |
| Kenya                           | *Aedes aegypti* breeding sites in rural and urban, coastal and western locations | (43) |
| Tanzania                        | Severe febrile illness and overdiagnosis of malaria                      | (49)      |
| Tanzania                        | Rift Valley Fever and alphavirus seroepidemiology                       | (12)      |
| Uganda (rural)                  | Febrile patients and overdiagnosis of malaria                            | (48)      |
| Uganda (Zika Forest)            | Arbovirus serology in endemic population                                | (61)      |
| East African Community Region   | Arbovirus infection                                                      | (11)      |
| Cameroon                        | Flavivirus seroepidemiology                                              | (12)      |
| Cameroon                        | *Aedes aegypti* and *Ae. albopictus* present                             | (65)      |
| Cameroon                        | *Aedes aegypti* and *Ae. albopictus* present                             | (66)      |
| Central African Republic        | *Aedes aegypti* and *Ae. albopictus* present                             | (67)      |
| Mozambique                      | Dengue, chikungunya, Rift Valley fever, West Nile, and Zika virus seroepidemiology | (12) |
| Côte d’Ivoire (southeast)       | *Aedes* mosquitoes present in an arbovirus-endemic setting               | (42)      |
| Sierra Leone                    | Rift Valley Fever virus, flaviviruses, and alphaviruses                 | (68)      |
| West Africa                     | Expansion of DENV-3                                                      | (60)      |
| West Africa                     | Dengue, chikungunya, and Zika outbreaks and *Aedes aegypti* and *Ae. albopictus* presence | (13) |
| Africa                          | Dengue virus infection                                                   | (59)      |
| Africa                          | Overdiagnosis and co-morbidity of severe malaria                        | (50)      |