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THE GROWING THREAT OF ARBOVIRUS TRANSMISSION AND OUTBREAKS IN KENYA: A REVIEW
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THE GROWING THREAT OF ARBOVIRUS TRANSMISSION AND OUTBREAKS IN KENYA: A REVIEW

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ABSTRACT

Objective: **To review the trend in arbovirus outbreaks and activity in Kenya in the last ten years.**

Data source: **Published reports of past outbreak investigations and more recent data available at the Arbovirology and Viral haemorrhagic fevers reference centre, Centre for Virus Research, Nairobi.**

Study selection: **Past and recent outbreaks and active transmission reports of arboviruses of medical importance in Kenya including Yellow fever (YF), Rift Valley Fever (RVF), Dengue and Crimean Congo haemorrhagic fever.**

Synthesis: **Each of the viruses was reviewed providing critical information on classification, incidence, outbreak, and activity in Kenya, mode of transmission, recognition of cases, management and control.**

Conclusion: **There is increased frequency of outbreaks and detection of arbovirus activity in humans and vectors in the last ten years including re-emergence of YF virus as a public health concern in Kenya. The importance of recognition of cases and diagnosis (especially in malaria endemic areas) is critical to management and control. Effective countrywide surveillance backed by diagnostic centres is highly recommended.**

INTRODUCTION

Arthropod-borne viruses, more commonly referred to as arboviruses, are characterised by a biological cycle in which the virus replicates alternately in vertebrate and haematophagus hosts (typically mosquito, tick, or sandfly). Arthropods usually become infected following a blood meal on a viraemic vertebrate host and remain infectious for the duration of their life. After a period of incubation and replication in the tissues of the arthropod host, virus may then be passed to non-immune vertebrate hosts during subsequent feedings. Certain arboviruses are maintained in their arthropod host from one generation to the next by transovarial or transstadial transmission of the virus. Although this mechanism is inefficient, it represents a maintenance survival mechanism of the virus in overcoming conditions that are not favourable for its arthropod host. Arboviruses are classified into three main families: the Togaviridae; the Flaviviridae and the Bunyaviridae, whilst some arboviruses are antigenically distinct from the three major families and are classified separately. Many of the 535 registered arboviruses are the cause of major zoonoses. A zoonosis is an infectious disease, which is transmissible from vertebrate animals to humans. A zoonotic disease can also be enzootic (endemic) or epizootic (epidemic). Humans are accidental hosts which become exposed to virus when the zoonotic cycle is interrupted and an infected arthropod takes a human blood meal. Epizootics occur periodically which can involve thousands of people, for example epizootics of Rift Valley fever virus. Arboviruses cause a range of clinical syndromes in humans depending on the infecting virus and can range from a self-limiting, febrile illness of short duration, to life threatening encephalitis or haemorrhagic fever. However, the majority of infections caused by arboviruses remain undiagnosed and hence the apparent frequency of arbovirus disease, and the public health threat that they pose, is greatly underestimated. A major factor for this situation is the non-specific nature of the clinical signs and symptoms of arbovirus infections, which may be confused with illnesses such as malaria, typhoid, dysentery and bacterial meningitis. The diagnosis of arbovirus disease is usually dependent on a specialised laboratory, which is supported by an active disease surveillance programme.

Arboviruses, because of their dependence on arthropod and vertebrate hosts for successful maintenance, are greatly affected by environmental and demographic changes. Global and local changes to the environment (natural or man-made) exert selective pressure on established ecosystems, which can create conditions that favour the emergence of arboviruses in human communities. This review discusses the arboviruses which pose a particular public health threat to Kenya, their recognition, management and control and will also highlight factors which may be important in governing the emergence and re-emergence of these viruses in the future.

YELLOW FEVER VIRUS

The mosquito-borne yellow fever (YF) virus is the prototype member of the family Flaviviridae, genus flavivirus. YF virus is maintained by a sylvatic or jungle cycle involving tree canopy breeding mosquitoes and their vertebrate, non-human primate feeding hosts. In Kenya, *Aedes africanus is* implicated as the vector for sylvatic transmission based on the isolation of YF virus from these mosquitoes(1). YF is able to cross into a peri-domestic or rural transmission cycle involving peri-domestic mosquito species and man. Transmission of YF virus to man is associated with a different species of mosquito vector which is often, but not exclusively, *Aedes bromeli* in E. Africa. The risk of YF virus transmission to man is related to the biting preferences of the infected mosquitoes. Urban transmission of YF virus involving the mosquito *Aedes aegypti* is associated with explosive outbreaks of yellow fever that have been documented in West Africa (2). Such massive epidemics of YF have not been recorded in East Africa and may be related to differences in the feeding behaviour of the *Ae. aegypti* populations in this region(3).

Prior to 1992, only circumstantial evidence existed for the circulation of YF virus in Kenya. Most of this data was serological except for a single post-mortem case in a soldier in 1943, although a definitive diagnosis of YF was never made. YF was suspected based on liver pathology(4). Seroprevalence rates for YF in selected communities of Northern Kenya surprisingly revealed exposure rates of between 5-14% Marsabit recording the highest level of sero-positivity(5). The populations of Northern Kenya had not received previous YF vaccination hence the results were likely to represent true exposure of the communities to YF virus. It is currently believed that exposure was related to an extension into Kenya of the large epidemic of YF which occurred in Omo Valley region of Ethiopia in 1960-62(6). In 1992/93 the first confirmed outbreak of YF occurred in Kenya with 54 cases and 29 deaths (a case fatality rate of 53%). The morbidity and mortality figures for this outbreak do not represent the true extent of the outbreak as surveillance was purely hospital based. Approximately 60% of all YF infections are mild and self-limiting and patients may not seek healthcare in hospitals. Even if a patient presented to a hospital with a mild YF infection, the likelihood is that the infection would be clinically diagnosed as malaria or typhoid. Not surprisingly the 1992/93 outbreak of YF in the Kerio Valley was attributed to 'highland malaria' before a definitive laboratory diagnosis of YF was made. Populations at risk of exposure to YF were subsequently vaccinated and approximately one million Kenyans received the live attenuated YF 17D vaccine. A single dose of vaccine confers a solid immunity for a period of at least 10 years and possibly lifelong. The mass vaccination campaign was successful in ending the epidemic transmission of YF virus in the Kerio Valley, Kenya. Between October 1993 to December 1995 a hospital based sentinel surveillance programme detected continued, low level transmission of YF virus with an additional 10 positive cases of YF (confirmed serologically) and three deaths (a case fatality rate of 33%). Importantly, the location of the new cases indicated a southward expansion of the disease(7). Entomology studies during the 1992/93 outbreak were successful in isolating YF virus from field-caught mosquitoes. Virus was isolated from *Ae. africanus* (the principal vector), *Ae. bromeli* and the little known *Ae. keniensis*(1). The YF outbreak in Kenya was a sylvatic outbreak and no cross-over of YF virus to the urban Ae. *aegypti* mosquito vector was recorded. The reasons why YF virus should suddenly emerge in the human population is the subject of many theories. It is currently believed that weather patterns played a significant role in the emergence of YF in Kenya. The outbreak of YF was preceded by a severe drought in 1991-1992 which concentrated primate populations around water sources inhabited by a multitude of mosquito vectors with different feeding preferences(1). Mosquito species such as A*e. bromeli* will readily take blood meals from either primate or human hosts, hence if a mosquito fed on a viraemic primate it is possible that the mosquito could later transmit the virus to man. Studies during the outbreak showed that approximately 37% of blood samples collected from primates were sero-positive for YF(1). Additional plausible theories to explain the emergence of YF virus in the Kerio Valley include the expansion of human settlements and activities (road construction and agriculture) in to forested areas where the sylvatic cycle of YF transmission existed; and the importation of YF in *Amblyomma* ticks (implicated as a potential vector of YF in Central Africa) which were attached to livestock that were driven in to the Kerio Valley in search of grazing pasture and water(1,8).

Attempts to combat YF through vector control have proved difficult to sustain. This problem is compounded when the ecology of the mosquito vector is poorly understood, as is the case in Kenya The most effective approach to the control of YF remains the use of the highly effective 17D live attenuated YF vaccine coupled with the use of active surveillance to rapidly detect YF virus activity(9).

DENGUE VIRUS

Dengue virus, a mosquito-borne flavivirus, is the worlds most serious arboviral threat to public health with at least 20 million infections recorded worldwide each year. There are four dengue virus serotypes (designated Den-1, -2, -3, -4) which may circulate either as a single virus serotype or as multiple virus serotypes. All four serotypes of dengue virus cause a similar clinical disease which is classically manifested by high fever, headache, severe myalgia, nausea and vomiting and frequently rash (which is often difficult to discern in dark skinned patients). A primary dengue virus infection is usually self-limiting and not usually life-threatening, although the period of convalescence following the acute illness may be prolonged for several weeks. The immune response that follows

infection affords life long protection from secondary infection with the same dengue virus serotype. If all four dengue serotypes are endemic, it is possible for a person to have four dengue virus infections in his or her lifetime. Secondary dengue infection carries with it a possible risk of developing dengue haemorrhagic fever and/or dengue shock syndrome (DHF/DSS) which are severe, life threatening conditions. Why secondary dengue virus infections are sometimes more severe is not well defined and is the subject of much research DHF/DSS is a particular problem in South East Asia and on the Indian subcontinent where multiple dengue virus serotypes are endemic. However, with good supportive therapy and careful monitoring of fluid and electrolyte balances, the mortality rate for DHF/DSS can be reduced to less than 1%. Despite evidence for the presence of all four dengue serotypes in some West African countries (through the isolation of virus from clinical cases or mosquitoes), no case of DHFS has been confirmed. The reasons for this are also unknown. It is however noteworthy that infections clinically compatible with DHF/DSS have been reported in Africa but little work has been done to investigate whether dengue virus was involved(11,12).

Dengue viruses are probably maintained in the environment in a similar manner to yellow fever virus through a mosquito-primate-mosquito transmission cycle. Transmission of dengue virus to man usually involves the *Aedes aegypti* mosquito vector. Where dengue is endemic, this mosquito feeds almost exclusively on man and is very adapted to human settlements where it breeds in and around households often using water storage containers for breeding. Evidence for transovarial transmission of dengue virus in mosquitoes exists and probably represents an additional maintenance mechanism for the virus.

In 1982 a Canadian tourist, who had visited Malindi on holiday, fell ill with a generalised febrile illness. Dengue-2 virus was subsequently isolated from this patient and further investigations led to an additional seven isolations of dengue-2 virus from clinically ill patients(13). The additional isolations were made from Malindi, Mombasa and Diani showing a wide distribution of dengue virus along the Kenya coast. It is almost certain that the dengue virus isolated in 1982 was not a recent introduction to Kenya as clinical descriptions compatible with dengue virus have been reported from East Africa since the 19th century(14). However this was the first confirmation that dengue virus is endemic in Kenya. A more recent isolation of dengue-2 virus has been made from the serum of a febrile child from Kilifi, Kenya with genetic sequencing showing the Kilifi isolate to be almost identical to the dengue-2 virus isolated 17 years earlier in 1982. This latest isolation suggests that active transmission and infection of humans with dengue-2 virus may be more common at the Kenya coast than is currently believed. Whether or not dengue-2 is the only serotype endemic in Kenya needs further study. Given the knowledge that dengue-2 and dengue-3 serotypes have been isolated in Somalia(15) plus the large amount of sea bound commercial traffic between East Africa and the Indian subcontinent (where all four dengue serotypes exist) it would seem highly likely that additional dengue virus serotypes may be endemic in Kenya.

As far as control of dengue virus infection is concerned, there is no vaccine currently available although some putative dengue virus vaccines are in advanced stages of trial. The most effective method available for controlling an outbreak of dengue fever in the community is to remove or destroy the breeding sites of the *Aedes aegypti* mosquito vectors within the domestic environment.

WEST NILE VIRUS

West Nile virus (WNV) was originally isolated in West Nile Province, Uganda in 1937 from the blood of a woman presenting with a mild febrile illness(16). The virus is mosquito-borne and is classified as a member of the Japanese encephalitis virus subgroup of the genus *flavivirus*. Since this date, WNV has been responsible for large epidemics of febrile illness and encephalitis with numerous fatalities being recorded worldwide. WNV has a wide distribution ranging from Africa to Europe. Avian species are an important vertebrate amplifying host for the virus which are infected when they are fed on by mosquitoes. One theory to account for the range of distribution of WNV is the role of migratory patterns of avian species. A great number of Palearctic avians are channelled into the Great Rift Valley during their migration to avoid the European winter and then return with the onset of the following European summer(17,18). Viraemic avian species returning to Europe could unwittingly introduce non-endemic viruses to the continent when they are fed on by native European species of mosquitoes. This theory is supported by evidence that an outbreak of West Nile fever in Romania in 1996 was caused by a virus that is genetically identical to a WNV isolate of Kenyan origin(19). One vector species that is usually implicated with epidemic transmission of WNV to man is the *Culex sp.* mosquito. Although the transmission cycle of WN virus in Kenya has not been studied extensively, it is believed to involve *Culex univittatus* complex mosquitoes as both the enzootic vector and the principal vector transmitting virus to man. This is supported following the recent isolation of WNV from male *Culex univittatus* complex mosquitoes collected at the Turkwel Gorge Hydroelectric station, West Pokot district, Kenya(19). The isolation of the virus from male mosquitoes (which do not take blood meals) was the first evidence of natural transovarial transmission of WNV and likely represents an important maintenance mechanism of the virus. Although epidemics of WN virus have not been documented in Kenya, the fact that WNV was isolated from a small collection of 301 mosquitoes at Turkwel Gorge demonstrates a high level of virus activity in this area.

Clinically, most infections with WNV are sub-clinical and evidence of infection is often only detected during sero-prevelance surveys. However, WNV may also cause

more clinically apparent infections manifested as febrile illness with rash, arthralgia and encephalitis. More severe forms of infection have been recorded in Africa including infection of the liver with resulting jaundice and meningitis or meningo encephalitis in older patients. As with the majority of arbovirus infections, the non-specific signs and symptoms often lead to the infection being attributed to other common disease causing agents and hence the virus, as the aetiologic agent, remains largely undiagnosed.

CHIKUNGUNYA AND O'NYONG-NYONG VIRUSES

Chikungunya (CHIK) and O'nyong-nyong (ONN) viruses are clinically the most important alpha-viruses in Kenya and E. Africa. Both CHIK and ONN are members of the Semliki Forest subgroup of the *Alphavirus* genus, family Togaviridae. CHIK was first isolated from febrile patients during an epidemic in Tanzania in 1952/53(20). The virus is widespread throughout sub-Saharan Africa and South East Asia and serological surveys have shown 20-90% immunity in some communities. CHIK has been isolated from a number of different genera and species of mosquitoes including: *Aedes aegypti, Ae. africanus, Ae. furcifer-taylori* group, and *Ae. luteocephalus* and *Mansonia africana.* During epidemics, CHIK is primarily transmitted by *Aedes africanus* and *Aedes aegypti,* the latter being the typical vector in urban settings. A maintenance cycle for CHIK involving non-human primates and tree canopy breeding mosquitoes has been reported (21) and may be important for virus maintenance in the absence of evidence for transovarial transmission of CHIK virus in mosquitoes.

ONN was first isolated during an outbreak of fever with severe joint pain that swept through the Lake Victoria basin region of Uganda and Kenya, and as far as Tanzania and Malawi in 1959/60. More than two million people were infected with ONN in Uganda alone during the outbreak(22). Lack of surveillance in E. Africa for ONN probably reflects an underestimate of virus activity in the region. Unlike CHIK virus, ONN has not been reported in large epidemics outside E. Africa. The major vectors of ONN are *Anopheles gambiae* and *Anopheles funestus* mosquitoes(22,23). During 1996/97, south central Uganda experienced the second major ONN epidemic to be documented in East Africa(24). The epidemic was focussed near lakes and swamps where it was associated with high attack rates in the communities. At the time of this outbreak a large amount of discussion centred on where the virus had been sequestered for the past 37 years when there was no reported outbreak of ONN. Studies to find an animal reservoir for the ONN or evidence of a transmission cycle involving lower primates had been unsuccessful. One single isolation of ONN virus had been made from a pool of *Anopheles funestus* in Kenya in 1978, but this was not associated with an ongoing epidemic(23). Had ONN really disappeared for 37 years? The answer to this question came from a study to determine the cause of 'fevers of unknown' origin in Western Kenya. In the study, conducted in 1994, serum samples from patients with a non-descript febrile illness were collected. At the time of the study ONN and CHIK viruses were not included in the screening profile of infectious agents. Following the 'return' of ONN in Uganda in 1996/97, the serum samples were examined for evidence of ONN and CHIK infection. A total of 74 acute ONN virus infections and 5 CHIK virus infections were diagnosed in addition to 43 unspecified alphavirus infections from a total of 398 samples (7). It therefore appears that ONN transmission in Kenya, and perhaps in other countries where the ONN virus is endemic, actively occurs during inter-epidemic periods and it is the introduction of the virus in to an immunologically naive community that leads to the epidemics described above.

The illness caused by CHIK or ONN viruses are characterised as an acute self-limiting febrile illnesses with severe joint pain (arthralgia) and rash. The arthralgia associated with infection is the most significant feature and occurs in approximately 70% of cases. Infection with ONN virus also gives rise to a generalised lymphadenopathy which has been used as a diagnostic marker (together with fever, joint pains and rash) during times of epidemic virus activity. Whilst the arthralgia usually resolves over a period of approximately one week, it is not uncommon for pain to remain for many weeks to months. Rare haemorrhagic manifestations associated with CHIK virus infection have been reported during epidemics in India but this has not been reported in Africa. There are no available control strategies for ONN and CHIK as there are no available vaccines and mosquito control is still an elusive solution for many of the arboviruses. In times of outbreaks, people can be advised to avoid mosquito bites by use of mosquito nets at night and adaptation of environmental sanitation practices.

RIFT VALLEY FEVER

Rift Valley fever (RVF) virus was first described as a disease causing agent in 1931 at Lake Naivasha, Kenya(25). The mosquito-borne RVF virus is a member of the *phlebovirus* genus of the family Bunyaviridae and affects both domestic animals and humans throughout sub-Saharan Africa. Widespread losses of livestock as a result of RVF infection (sheep, cattle and goats suffer between 10-30% mortality and a large percentage of pregnant animals abort) have a significant economic impact. The epizootics of RVF virus in Kenya is strongly linked to extreme climatic events of prolonged and heavy rainfall that result in the flooding of dambos (depression on land with imperfect drainage) followed by hatching of numerous *Aedes* (Neomelaniconion) eggs commonly referred to as flood water mosquitoes.

A proportion of these eggs are transovarially infected by RVF virus(26,27). If flooding is persistent, the enzootic cycle of RVF virus is initiated by infected *Aedes* mosquitoes feeding on nearby livestock, followed by secondary *Culex sp.* mosquitoes feeding on the viraemic livestock. It is the infection of some *culex* mosquitoes (*Culex pipiens, Culex*

zombaensis; Culex *antronatus)* which have cosmopolitan host feeding preference, that initiates an epizootic in which the human population becomes involved(27). Recently, the use of satellite remote sensing in being able to predict potential outbreaks of RVF by measuring the normalized-difference vegetation index (NDVI), as a marker of photosynthetic capacity has been applied in RVF endemic parts of Kenya(28). Using data from the advanced very high resolution radiometer (AVHRR) on National Oceanic and Atmospheric Administration (NOAA) satellites, it is now possible to predict outbreaks of RVF upto five months in advance. All outbreaks of RVF that have occurred in Kenya since 1950 could have been predicted in advance by satellite remote sensing NDVI data. This is potentially of great value in the timely implementation of public health measures to avert or contain future outbreaks of RVF in Kenya. In north-eastern Kenya alone during the 1997/98 RVF epizootic an estimated 89,000 new cases of RVF occurred making the outbreak possibly the largest ever in East Africa(29).

RVF in humans is characteristically an acute febrile illness with fever, severe headache, myalgia of short duration (2-3 days for most patients although symptoms may persist longer). A small proportion of patients (approximately 1%) may develop a more severe illness characterised by haemorrhagic events involving gastrointestinal bleeding and jaundice. Encephalitis has been recorded in RVF patients and is another serious and often fatal manifestation of the disease which may occur after apparent recovery (symptoms may develop upto one to two weeks later). Patients complain of headache, fever and a range of neurologic signs which may progress to coma and death. Notable sequelae of RVF infection include visual problems, as a result of retinal haemorrhage, with some patients suffering permanent bi-lateral blindness.

Control of RVF virus is possible through the use of an inactivated vaccine for livestock. This removes the important primary amplification of virus in livestock as a prelude to subsequent human infection. A vaccine is also available for human use but this vaccine remains experimental and is only available in limited quantities for workers at high risk of infection e.g. laboratory workers. An additional possibility for control or prevention of outbreaks of RVF may now be possible by using the predictive algorithms for epizootics based on satellite remote sensing data and weather patterns. Should conditions prevail that are favourable for RVF transmission, heavy and prolonged rainfall leading to the flooding of dambo sites, insecticides may be used in the breeding sites to control mosquito larvae and stop the emergence of transovarially infected mosquitoes.

CRIMEAN-CONGO HAEMORRHAGIC FEVER

Crimean-Congo haemorrhagic fever virus (CCHF) is a member of the *Nairovirus* genus of the family Bunyaviridae. Of the viruses known to cause haemorrhagic fever (Ebola, Marburg, Lassa, yellow fever, dengue, Rift valley fever), the haemorrhaging associated with CCHF infection is the most severe. The virus is maintained in nature by trasstadial and transovarial transmission within the tick population (primarily *Hyalomma sp.)* and by horizontal transmission between the ticks and various domestic and wild animals. CCHF has a wide distribution and occurs throughout much of Africa, the Middle East, Crimea, Pakistan and Western China. The distribution of CCHF virus possibly reflects movement of infected ticks with migratory birds and wild life and the import and export of livestock. Man may be exposed to CCHF virus through a number of different routes but it usually involves one of the following: the bite of an infected tick, the crushing of an infected tick which may allow the virus to gain access through abrasions of the skin or mucous membranes, contact with the blood or fluids of an infected animal during slaughtering, or contact with the blood and body secretions of a patient suffering from CCHF infection. The first two modes of transmission are typical of rural settings where livestock farming is practised. The recent mass movement of livestock throughout Kenya by pastoralists in search of grazing land carries with it the potential for dispersal of tick-borne viruses including CCHF virus. Slaughter-house workers are at risk of exposure to CCHF virus not only from infected ticks which will vacate the hide of a slaughtered animal to seek a fresh host but also from the blood of viraemic animals during the slaughtering process(30). Nosocomial transmission in hospitals is commonly seen with CCHF and poses a great risk to healthcare workers. Transmission in hospitals is associated with direct, unprotected contact with the blood and secretions of acutely ill patients. There is no direct evidence for aerosol transmission of CCHF virus in hospital settings but the remote possibility cannot be ruled out. Laboratory infections with CCHF virus have also been recorded with fatal outcomes on occasion. Typically, following exposure to CCHF virus the first signs and symptoms of infection appear after an incubation period of between 3-12 days (shorter incubation times are typical of nosocomial infection). The illness manifests itself by sudden onset of headache, fever, severe muscle/joint pain, nausea, vomiting and diarrhoea. As the illness progresses haemorrhagic manifestations often become apparent with skin haemorrhaging forming large ecchymoses, epistaxis, malena and haematemesis. Neurological involvement has also been recorded and is associated with a poor patient prognosis and severe liver necrosis is observed in fatal cases. The mortality rate for CCHF infection may be as high as forty per cent.

Serological studies have revealed evidence of CCHF virus activity in Kenya and in addition, the virus was isolated from a febrile cow in Nakuru in 1965 and from *Rhipicephalus pulchellus* ticks taken from a dying sheep at the Kabete Laboratories in 1975(31). More recently, a fatal human case of CCHF was diagnosed in Western Kenya which fortunately remained an isolated case despite the patient entering a large healthcare facility for supportive treatment.

No vaccine is available against CCHF virus and the treatment of cases is supportive with strict barrier nursing procedures. Intravenous administration of the antiviral drug ribavirin has shown promise in the treatment of CCHF but therapy must be started as early as possible during the course of infection for maximum benefit. The control of primary CCHF infections relies on the avoidance of practices that are of potential risk. Livestock should be dipped regularly to control tick infestation and protection should be provided to slaughterhouse workers. In the hospital setting, strict barrier nursing procedures, safe disposal methods for contaminated waste and safe burial practices are paramount.

ENVIRONMENTAL FACTORS AFFECTING THE EMERGENCE AND RE-EMERGENCE OF ARBOVIRUSES IN KENYA

Of the approximately 535 registered arboviruses, roughly 100 of these have been associated with human disease of which 50% are mosquito-borne. However, in countries where malaria is either endemic or hyper-endemic, outbreaks of disease of arboviral origin often go unrecognised. What governs the emergence and/or re-emergence of arboviruses as a health threat is complex but five important variables may influence this rate, namely: vector, virus, wild vertebrate host, humans and environmental factors. Environmental factors have a most profound effect on the rate of emergence of arbovirus disease by altering vector dynamics and ecology. In the case of irrigation and cultivation schemes for example, the presence of water supplies provide increased opportunities for vectors with aquatic larval stages to breed for prolonged periods and are no longer dependent on seasonal rainfall. The presence of human settlements as a consequence of economic prospects from irrigation schemes also enhances mosquito vector diversity by providing additional breeding habitats through potable water storage. This practice increases the chances of promoting colonies of mosquitoes that are highly adapted to feeding and breeding close to human settlements including important vectors in the transmission of yellow fever and dengue (*Aedes* aegy*pti* mosquitoes). The practice of deforestation is strongly linked to the emergence and re-emergence of arbovirus disease as farmers and livestock are exposed to new arthropod vectors and the diseases that they carry. Population movement has long been associated with the sudden appearance of outbreaks of disease as immunologically naive people enter a disease endemic area. In a similar manner, large-scale movement of livestock, or importation of animals from neighbouring countries, carries with it the threat of importation of new arthropod vectors and the diseases they carry. An outbreak of CCHF virus in slaughterhouse workers in Saudi Arabia in 1996 was traced to the importation of livestock from Somalia(30). Importation of new arbovirus diseases through commercial travel must also be considered. Container vessels can transport mosquitoes for huge distances across the globe and non-endemic arbovirus diseases have the potential to emerge at any time. A particular case in point was mentioned in the section dealing with dengue fever. Numerous commercial vessels cross the Indian Ocean from Kenya to the Indian subcontinent and vice versa. The four dengue virus serotypes are endemic in India and importation of dengue virus infected *Aedes sp.* mosquitoes to Kenya through commercial vessels is a significant possibility. The climate along the Kenya coastline and the *Aedes aegypti* population present is clearly suitable for dengue virus transmission as dengue-2 virus is already endemic in Kenya, hence the potential for outbreaks of dengue fever due to the importation of a new serotype. Global warming is exerting a profound impact on the emergence and re-emergence of arbovirus disease as mosquitoes which were originally restricted to warmer climates are now increasing in their distribution as global temperatures rise. As the mosquitoes expand in to new environments, new diseases are recognised in communities that were once disease free. Another effect of global warming is a reduction in the extrinsic incubation period (EIP) of the virus in its mosquito vector host. The EIP represents the time from ingestion of virus by the mosquito vector to the time the vector is able to transmit the virus through subsequent feeding. A reduced EIP means that enhanced transmission of arboviruses may be possible during the short lifetime of the mosquito and the potential for epidemics of arbovirus disease increases.

CONCLUSION

The re-emergence, increased outbreak frequency and growing endemicity of some of the important arbovirus disease agents in Kenya is a real problem that requires more attention than it currently receives. It is important that these infections be recognised and proper and continued surveillance and control strategies be put in place in order to deal with future outbreaks.

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