Epidemic Dengue/Dengue Haemorrhagic Fever:  
A Global Public Health Problem in the 21st Century*

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Abstract
Dengue/dengue haemorrhagic fever has been one of the most important resurgent tropical diseases in the past 17 years, with expanding geographical distribution of both the viruses and the mosquito vectors, increased frequency of epidemics, development of hyperendemicity (co-circulation of multiple virus serotypes) and the emergence of dengue haemorrhagic fever in new areas. This paper briefly reviews the changing epidemiology of dengue, discusses some of the factors responsible for the recent resurgence, and reviews the current options available for reversing the emerging trend of the disease.

Introduction
Dengue fever/dengue haemorrhagic fever (DF/DHF) is caused by infection with four dengue virus serotypes DEN-1, DEN-2, DEN-3 and DEN-4, which are closely related to each other antigenically. This results in extensive cross-reactivity in serological tests, but infection with one serotype does not provide cross-protective immunity against the others; thus, persons living in an endemic area can be infected with each of the four dengue serotypes during their lifetime.

Epidemic dengue fever is a very old disease, but it was characterized during most of its history by periodic, often infrequent, epidemics. In the past 17 years, however, there has been a dramatic resurgence of epidemic dengue activity in the tropics worldwide. This increased epidemic activity, which has been caused by all four virus serotypes, has been associated with the

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geographical expansion of both the mosquito vectors and the viruses, the development of hyperendemicity (the co-circulation of multiple virus serotypes in an area), and the emergence of dengue haemorrhagic fever. Hyperendemicity is the most constant factor associated with the evolution of epidemic DHF in a geographical area.

This paper reviews the changing epidemiology associated with dengue viruses and attempts to explain why such changes have occurred in the waning years of the 20th century.

**Transmission cycle**

Infection with dengue viruses is transmitted through the bite of infective female *Aedes* spp. mosquitoes. *Aedes aegypti*, the principal vector, is a small black-and-white, highly domesticated mosquito that prefers to lay its eggs in artificial water-containers commonly found in urban areas of the tropics. Containers found in and around the home, such as those used for water storage, flower vases, old automobile tyres, buckets and other junk items that collect rainwater are examples. The adult mosquitoes are rarely noticed, preferring to rest indoors and to feed on humans during daylight hours in an unobtrusive and often undetected way.

Infection with dengue viruses occurs when a person is bitten by an infective mosquito. After a period of incubation lasting 3 to 14 days (average 4 to 6 days), the person may experience an acute onset of fever accompanied by a variety of non-specific signs and symptoms.

During this acute febrile period, which may be as short as 2 days and as long as 10 days, there is a viremia, which may vary in magnitude and duration. If uninfected *Ae. aegypti* mosquitoes bite the ill person during this febrile viremic stage, those mosquitoes may become infected and subsequently may transmit the virus to other, uninfected persons after an extrinsic incubation period of 8 to 12 days.

**Clinical presentation**

Dengue virus infection in humans of all four virus serotypes causes a spectrum of illness ranging from inapparent or mild febrile illness to severe and fatal haemorrhagic disease. Clinical presentation in both children and adults may vary in severity, depending on the strain and serotypes of the infecting virus, and the immune status, age and the genetic background of the patient. In dengue endemic areas, acute dengue infections are often clinically nonspecific, especially in children, with signs and symptoms of a viral syndrome.

Classical dengue fever is primarily a disease of older children and adults, characterized by a sudden onset of fever and one or more of a number of non-specific signs and symptoms such as frontal headache, retro-orbital pain, myalgias, arthralgias, nausea and vomiting, weakness and rash. Anorexia, altered taste sensation and mild sore throat are not uncommon. Clinical laboratory findings associated with dengue fever include leukopenia, and, in some patients, thrombocytopenia and elevated liver enzymes. Haemorrhagic manifestations may occur, the most common being skin
haemorrhages. Dengue fever is generally self-limiting and rarely fatal, the acute illness lasting 3 to 7 days. Convalescence, however, may be prolonged for weeks with weakness and depression. No permanent sequelae are known, and immunity for the infecting virus serotype is lifelong.

Dengue haemorrhagic fever is primarily a disease of children under 15 years of age, although it may occur in older children and adults as well. Like dengue fever, it is characterized by a sudden onset of fever and non-specific signs and symptoms, and is difficult to distinguish from dengue fever and other illnesses during the acute stage. The critical stage in DHF occurs at the time of defervescence when the patient develops a capillary-leak syndrome, with signs of circulatory failure and haemorrhagic manifestations, primarily skin haemorrhages. Thrombocytopenia (<100,000/mm³) and elevated haematocrit are prominent features. DHF can be a very dramatic disease with the patient's condition deteriorating very rapidly with the onset of shock and resulting in death if the plasma leakage is not detected and corrected with fluid replacement therapy. Leukopenia, thrombocytopenia and haemoconcentration are constant findings; hepatomegaly and elevated liver enzymes are common. Risk factors for developing severe haemorrhagic disease are not fully understood, but, as noted above, include the strain and serotype of the infecting dengue virus, the immune status, age and genetic background of the patient.

Geographical distribution and incidence

Dengue fever and Ae. aegypti mosquitoes have a worldwide distribution in the tropical areas of the world, with over 2.5 billion people living in dengue endemic areas (Figure 1) [13,17,29]. The currently known geographical distribution of the various dengue virus serotypes is shown in Figure 2, and reflects the development of hyperendemicity in most tropical areas of the world in the past 15-20 years. In 1997, DF/DHF has been the most important arboviral disease of humans, with an estimated 50 to 100 million cases of dengue fever and several hundred thousand cases of DHF occurring each year, depending on epidemic activity [13,14,24]. Currently, DHF is a leading cause of hospitalization and death among children in many south-east Asian countries where epidemics first occurred in the 1950s [11]. Epidemic DHF spread to the South Pacific islands in the 1970s, and reached the Caribbean Basin in the 1980s [6,13,14]. The pattern of severe haemorrhagic disease has evolved in the American region in the 1980s and 1990s in a manner similar to the way it did in south-east Asia in the 1960s and 1970s [6,12]. In Central and South America, dengue fever has an economic impact of the same order and magnitude as malaria, tuberculosis, sexually transmitted diseases (excluding AIDS), hepatitis, the childhood cluster (polio, measles, pertussis, diphtheria) and the tropical cluster (schistosomiasis, filariasis, Chagas' disease, leishmaniasis and onchocerciasis) [23].
**Figure 1.** Global distribution of epidemic dengue and the principal vector mosquito, *Aedes aegypti*, 1997

**Figure 2.** Known global distribution of the four dengue virus serotypes, showing areas of hyperendemicity, 1997
Changing epidemiology

The epidemiology of dengue viruses changed with the ecological disruption in south-east Asia during and following World War II. During the war, existing water systems were destroyed, and water storage was increased for domestic use as well as for fire control. War equipment was moved between cities and countries, and large amounts of equipment were also left behind. This material collected rainwater and made ideal larval habitats for *Ae. aegypti*, resulting in the transport of mosquitoes and their eggs to new geographical areas. The result of these ecological changes was a greatly expanded geographical distribution and increased population densities of *Ae. aegypti*. In addition, hundreds of thousands of Japanese and Allied soldiers, most of them susceptible to dengue virus infection, were constantly moving between countries in Asia and the Pacific. This provided ideal conditions for movement of viruses between cities, countries and other regions as well as susceptible individuals for epidemic transmission. The war years were thus responsible for creating the conditions (hyperendemicity and high densities of *Ae. aegypti*) for the emergence of DHF in south-east Asia.

In the years following World War II, unprecedented urbanization of south-east Asia began, with millions of people moving to the cities of the region. Urban centres in most countries expanded rapidly in an uncontrolled and unplanned fashion. Housing was inadequate, and water, sewer and waste management systems deteriorated. The *Ae. aegypti* populations and dengue viruses thrived in this new ecological setting, with increased transmission and increased frequency of dengue epidemics occurring in the indigenous populations of children. Moreover, economic expansion began in the region that continues even today. This led to continued urbanization and increased movement of people (and with them, of dengue viruses) between cities and countries. Those countries that did not already have multiple virus serotypes co-circulating quickly became hyperendemic. The viruses, often all four serotypes, were maintained in a human-*Ae. aegypti*-human cycle in most urban centres of south-east Asia.

The result of these changes was a dramatically increased dengue transmission and the emergence of DHF. In every country where the disease emerged as a major public health problem, it evolved in a similar manner, first as sporadic cases of DHF occurring for several years, ultimately culminating in a major epidemic. Following the first epidemic, a pattern of epidemic activity was established, with epidemics occurring every 3 to 5 years. Characteristically, succeeding epidemics became progressively larger as a result of geographical expansion of DHF within the country.

The first DHF epidemic ever recorded as such occurred in Manila, Philippines, in 1953-54, although retrospective analysis suggests that outbreaks had occurred earlier as well before the dengue etiology was known. During the first 20 years that epidemic DHF was known, it was localized in several south-east Asian countries where it had become a major cause of hospitalization and death among...
children by the mid-1970s. The 1980s saw a dramatic geographical expansion of epidemic DHF in Asia; it moved west into India, Pakistan, Sri Lanka and Maldive, and east into the People’s Republic of China. There was also a resurgence of the disease in Singapore, which has continued through the 1990s.

Surveillance for DHF is passive, with only severe cases reported to the World Health Organization by most countries. Thus, only the tip of the iceberg is reported, making DF/DHF one of the most under-reported tropical infectious diseases in the past 20 years. Even so, approximately four times as many DHF cases have been reported in the last 15 years (1981-1995) than in the previous 30 years (Figure 3). In 1997, DHF has been a leading cause of hospitalization and death among children in many countries of Asia.

Activities related to World War II also resulted in expanded geographical distribution and increased densities of *Ae. aegypti* in the South and Central Pacific islands. A major regional pandemic of DEN-1 occurred on most islands from 1942 to 1945, affecting both indigenous and military populations. Following the war, the isolation of the Pacific islands and their small human populations resulted in the disappearance of dengue viruses from the area until the mid-1960s when a small outbreak of DEN-3 occurred in Tahiti. In late 1971, DEN-2 was introduced into the Pacific, followed in 1975 by a new strain of DEN-1, in 1979 by DEN-4 and in the early 1980s by a new strain of DEN-3. All virus serotypes caused major epidemics of dengue fever, and some islands experienced severe haemorrhagic disease compatible with DHF. The events in the Pacific have recently been reviewed in detail (16).

Epidemic dengue occurred only rarely in the Caribbean Basin countries after the 1930s, and from 1946 to 1963, there was no recorded epidemic transmission despite the evidence that at least one serotype (DEN-2) was endemic in the region (5). Epidemic dengue never re-emerged as a public health problem in the Americas until the late 1970s. This 40-year quiescence was likely due to several factors, the most important of which was the *Ae. aegypti* eradication programme initiated by the Pan American Health Organization (PAHO) in 1946 to prevent urban epidemics of yellow fever (30,32). The programme was successful and eradication was achieved in most countries of the region. Unfortunately, the programme was discontinued in the early 1970s, and failure to eradicate *Ae. aegypti* from the whole region resulted in repeated reinfestations by this mosquito vector of those countries that had achieved eradication. During the 1970s, support for *Ae. aegypti* surveillance and control programmes waned as these were merged with malaria control programmes in many countries. By the end of the decade, many countries had been reinfested with *Ae. aegypti* (9,10,26). The reinfestation of the region continued during the 1980s and 1990s. In 1997, *Ae. aegypti* had a distribution similar to that in the 1940s before eradication was initiated (Figure 4).
**Figure 3.** Emergence of dengue haemorrhagic fever – Reported cases to WHO, 1950-1995

**Figure 4.** Reinfestation of the Americas by *Aedes aegypti*, 1970-1997
The expanding geographical distribution of *Ae. aegypti* in the 1970s and 1980s coincided with increased movement of dengue viruses both into and within the American region \(^{6,12}\). Prior to 1977, only DEN-2 and DEN-3 viruses were known to be present in the Americas, although DEN-1 was probably present during the early 1940s \(^{6,8,12}\). DEN-3 caused the first epidemics in nearly 20 years in Jamaica and Puerto Rico in 1963, and DEN-2 caused epidemics in 1969 and the 1970s, again in the Caribbean islands that never achieved *Ae. aegypti* eradication. Both of these viruses were maintained in the region as district genetic genotypes \(^{21,22}\), and the DEN-3 caused subsequent epidemics in Colombia and Puerto Rico in the mid-1970s before apparently disappearing from the region \(^{6,12}\). A characteristic of dengue in the Americas from the 1950s through the early 1980s was non-endemicity (no viruses present) or hypo-endemicity (only a single serotype present) in a country \(^{5,6,12,16,26}\).

DEN-1 was re-introduced to the American region in 1977, with epidemics occurring in Jamaica and Cuba in 1977 and in Puerto Rico and Venezuela in 1978 \(^{26}\). In the succeeding four years, this serotype spread throughout the Caribbean islands, Mexico, Texas (USA), Central America and northern South America, causing major or minor epidemics \(^{6,12,26}\). The illness in all of these epidemics was classical dengue fever. In 1981, DEN-4 was introduced into the eastern Caribbean islands \(^{6,12}\). Like DEN-1, this serotype also spread rapidly to other islands in the Caribbean and to Mexico, Central America and northern South America, causing major or minor epidemics in countries that had recently experienced DEN-1 epidemics \(^{6,12}\). Some of these outbreaks (Suriname, 1982; Mexico, 1984; Puerto Rico, 1986; El Salvador, 1987) were associated with the emergence of DHF for the first time, occurring sporadically for the most part, and although DEN-4 was the predominant virus isolated in each of these epidemics, other dengue virus serotypes were also present \(^{6,12}\).

Also in 1981, a strain of DEN-2, new to the region, was introduced into Cuba from south-east Asia \(^{13,20,22,28}\). Unlike the DEN-1 and DEN-4 epidemics, the 1981 Cuban DEN-2 epidemic was associated with thousands of cases of severe haemorrhagic disease (Figure 5); this was the first major DHF epidemic in the Americas \(^{20}\). Although there were an estimated 10 000 cases of DHF, the case fatality rate was low (158 deaths), most likely because of hospitalization and effective management of suspected DHF cases \(^{20}\). In the three-month period of the epidemic, over 116 000 persons were hospitalized and placed on fluid replacement therapy. Although the viruses isolated in Cuba have been unavailable for study, DEN-2 viruses isolated in Jamaica at the time of and shortly after the Cuban epidemic (Gubler DJ, unpublished data) were sequenced and the data suggest that the virus causing the epidemic was a new strain introduced from Asia, most likely from Viet Nam, where several thousand Cuban aid personnel were working at the time \(^{20}\) (Gubler DJ, unpublished data).

The second major epidemic of DHF in the Americas occurred in Venezuela in 1989-90 with over 6 000 cases and 73
The virus serotype responsible is not definitely known since DEN-1, DEN-2 and DEN-4 viruses were all isolated from patients. However, DEN-2 appeared to be most frequently associated with fatal cases (F. Pinheiro, PAHO, personal communication); this virus was the same genotype as the virus thought to have caused the Cuban epidemic in 1981 \(^{22}\). Epidemic DHF of variable intensity caused by this genotype of DEN-2 subsequently occurred in Colombia (1990), Brazil (1992 and 1994), Puerto Rico (1994) and Mexico (1995), but none of these epidemics was of the same magnitude and severity as the Cuban epidemic of 1981.

In 1994, a new strain of DEN-3 was introduced into the American region, causing a major epidemic of DF/DHF in Nicaragua and a small outbreak associated with classical dengue fever in Panama \(^4\). This virus was shown to be genetically distinct from the DEN-3 that previously occurred in the Americas and has been shown to belong to the same genotype as the virus that caused the recent DHF epidemics in Sri Lanka and India \(^{21}\) (Lanciotti R, Quiros I, Clark GG and Gubler DJ, unpublished data). This strain of DEN-3, which apparently was also a recent introduction from Asia, subsequently spread throughout Central America and Mexico in 1995, causing major epidemics. Surprisingly, by early 1997, it was yet to be detected in the Caribbean islands or South America.

There is a potential for epidemic dengue transmission in the United States.
On three occasions in the past 16 years, autochthonous transmission, secondary to importation of the virus in humans, has occurred in Texas (1980, 1986, and 1995). Although the outbreaks were small, they underscore the potential for dengue transmission in the United States, where two competent mosquito vectors are prevalent. *Aedes aegypti*, the most important and efficient epidemic vector of dengue viruses, has been in the country for over 200 years and has been responsible for transmitting major epidemics in the past. Currently, this species is found only in the Gulf Coast states from Texas to Florida. *Aedes albopictus*, an Asian species, was introduced into the continental United States in the early 1980s and has since become widespread in the eastern half of the country. It currently occurs in 678 counties in 25 of the continental states; this species has also been found in Hawaii for over 50 years. However, it has yet to be associated with dengue transmission in the New World. Both *Ae. aegypti* and *Ae. albopictus* can transmit dengue viruses to humans, and their presence in an area increases the risk of autochthonous dengue transmission, secondary to imported cases.

The sequence of events associated with the changing epidemiology of dengue in the Americas in the 1970s, 1980s and 1990s was nearly identical to that which occurred in south-east Asia in the 1950s, 1960s and 1970s. Thus, re-invasion of Central and South America by *Ae. aegypti* in the 1970s and 1980s, combined with increased urbanization, increased movement of people and, with them, of the dengue viruses, resulted in most countries evolving from non-endemicity (no viruses present) or hypo-endemicity (one virus present) to hyperendemicity (multiple virus serotypes co-circulating). This resulted in increased frequency of epidemic activity and the emergence of DHF as a major public health problem. Several countries (Cuba, Venezuela, Brazil and Nicaragua) have had major epidemics of DHF in recent years. Moreover, outbreaks with sporadic or small numbers of cases of DHF have occurred in Nicaragua, Honduras, El Salvador, Guatemala, Mexico, Colombia, French Guiana, Suriname, Aruba, St. Lucia and Puerto Rico, and sporadic cases of DHF have been confirmed in the Dominican Republic, the U.S. Virgin Islands, Panama and Costa Rica. In 1980, DHF was not considered endemic in any American country. Between 1981 and 1997, however, there was a dramatic emergence of DHF, with 17 countries reporting laboratory-confirmed DHF that met the WHO case definition. This disease is now endemic in most of those countries where multiple dengue virus serotypes co-circulate and the number of cases reported to PAHO have increased dramatically (Figure 5). If the disease pattern continues to evolve in the Americas as it did in south-east Asia, the first ten years of the 21st century will bring more frequent and larger epidemics of DHF.

Surveillance for dengue in Africa has been poor during this century. Prior to the 1980s, the last recorded epidemic was in Durban, South Africa, in 1927-28. Endemic transmission of DEN-1 and DEN-2 was documented in Nigeria, but outbreaks were not reported. Although surveillance has not improved, reports of
epidemic dengue fever have increased dramatically since 1980 (Figure 6). Limited outbreaks have occurred in West Africa (Angola, 1986 and Senegal, 1990), but the most recent epidemic activity has occurred in East Africa and the Middle East, including the Seychelles (1977), Kenya (1982), Mozambique (1985), Sudan (1985), Djibouti (1991), Somalia (1982, 1993) and Saudi Arabia (1994)\(^\text{16}\). All four dengue serotypes have been involved, but to-date, epidemic DHF has not been reported in Africa or the Middle East. However, sporadic cases of the disease clinically compatible with DHF have been reported from Mozambique, Djibouti and Saudi Arabia.

**Factors responsible for global resurgence of dengue**

The reasons for the dramatic resurgence of epidemic DF/DHF in the waning years of the 20th century are complex and not fully understood, but are most likely associated with demographic and societal changes that have occurred over the past 50 years\(^\text{13,16}\). Several important factors can be identified. First, major global demographic changes have occurred, the most important of which has been the unprecedented population growth, primarily in tropical developing countries. Coincidental with this has been the uncontrolled and unplanned urbanization in these countries. These changes have resulted in large, crowded human populations living in urban centres in substandard housing with inadequate water, sewer and waste management systems, creating ideal conditions for increased transmission of mosquito-, rodent- and water-borne infectious diseases. Second, most consumer goods
are packaged in non-biodegradable plastic or cellophane materials, which are discarded into the environment where they collect rain-water and provide ideal larval habitats for the vector mosquito. Also, making ideal larval habitats are used automobile tyres, the number of which has increased dramatically in the past 20 years, and which are very difficult to dispose of from the environment. All these factors have contributed to the expanded geographical distribution and increased population densities of the principal mosquito vector *Ae. aegypti*. Third, effective *Ae. aegypti* mosquito control is virtually non-existent in most dengue-endemic countries. Emphasis over the past 25 years has been placed on ultra-low-volume space sprays of insecticide for adult mosquito control (10). This has been shown to be ineffective in controlling *Ae. aegypti* (10,25). Thus, hundreds of millions of people in urban centres of the tropics are living in intimate association with large populations of an efficient epidemic mosquito vector of dengue viruses.

A fourth factor which has had a great impact on the emergence of DF/DHF is the increased travel of people by jet airplane. The reinfestation of the American tropics by *Ae. aegypti* placed at risk for dengue infection large numbers of susceptible individuals living in permissive urban areas. The numerous epidemics and increased transmission of dengue that subsequently occurred there and in Asia and the Pacific provided increased opportunity for the viruses to move between countries, both within and between the regions. Air travel by humans, who are incubating the virus, provides the ideal mechanism for transporting dengue viruses between population centres of the tropics and results in a constant exchange of dengue viruses and other pathogens. An illustration of the increased human air travel is seen in the U.S. Department of Transportation data from 1983 to 1994 (15); the number of international departures from U.S. airports doubled from 20 to nearly 40 million, with over 50% of those departures destined for tropical areas.

Finally, the public health infrastructure required to deal with epidemic vector-borne infectious diseases has deteriorated during the past 30 years in most countries. Limited financial and human resources, and competing priorities for those resources, have resulted in a 'crisis mentality' among public health officials. The emphasis has thus been on implementing emergency control measures in response to epidemics rather than on developing programmes to prevent epidemic transmission (10). This approach has been particularly detrimental to dengue prevention and control because in most countries surveillance is very poor; the passive surveillance systems relied on to detect increased transmission are dependent upon reports by local physicians, who often have a low index of suspicion and do not consider dengue in their differential diagnosis of dengue-like illness. As a result, the epidemic has often reached or passed peak transmission before it is detected and emergency control measures are implemented, too late to have any impact on the course of the epidemic (10).
Prospects for the future

There is currently no vaccine for DF/DHF. Although live, attenuated vaccine candidates for all four virus serotypes have been developed\(^2\), it will likely be at least 10 years before they are available for general use. Prospects for reversing the trend of increased epidemic DF/DHF must rely on mosquito control, which are not promising in the near future. New dengue virus strains and serotypes will likely continue to move between areas where \textit{Ae. aegypti} occurs in infected air travellers, resulting in continued hyperendemicity, increased frequency of epidemic activity and increased incidence of DHF if effective prevention programmes are not implemented early. This will require changing the emergency-response mentality of government officials, public health professionals and the public to one of epidemic prevention.

Effective, sustainable prevention programmes for DF/DHF must have several components\(^1\). First, an active, laboratory-based surveillance system that can provide early warning for epidemic activity is essential. Moreover, there must be effective information exchange and international cooperation. The second component is a rapid-response contingency plan to prevent an incipient epidemic when the surveillance system predicts increased dengue transmission. Political support to implement this rapid response in a timely manner is critical to its success. The third component of a sustainable prevention programme is education of the medical community. Experience has shown that case fatality rates can be kept acceptably low if physicians and nurses understand the pathophysiological changes that occur in DHF; therefore, early diagnosis and effective management are the key to preventing fatalities in this disease\(^1\). The fourth component is community-based, integrated \textit{Ae. aegypti} control. Sustainability of the prevention programme will depend on decreasing the reliance on government mosquito control agencies and the transfer of more responsibility for \textit{Ae. aegypti} control to the inhabitants in urban areas where most dengue transmission occurs. This will require community participation and community ownership of the programme. Lastly, there is also a great need for research and improved public health infrastructure. Research is desperately needed to develop more effective prevention strategies, including new mosquito control technology and dengue vaccines, and on the epidemiology and disease pathogenesis of DF/DHF. Only with an improved public health infrastructure to support community-based prevention programmes will it be possible to reverse the trend of emergent epidemic DF/DHF.

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