DHF Epidemics in Cuba, 1981 and 1997:
Some Interesting Observations

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Abstract
Three main dengue outbreaks have been reported in Cuba, one in 1977, produced by DEN-1 and
categorized as classical dengue fever, and two DHF epidemics, one in 1981 and the other in 1997,
caused by DEN-2. Epidemiological observations revealed that secondary dengue infection was the most
important risk factor for DHF/DSS after a gap of 16 to 20 years, the severity of the disease was more
during the 1997 epidemic as compared to 1981, and people of both black and white races were equally
infected. However, black people were at a reduced risk to manifestations of DSS as compared to white
people.

Key words: Dengue Haemorrhagic Fever, Dengue Shock Syndrome, Aedes aegypti, Cuba

Introduction
In the last few years, dengue and dengue haemorrhagic fever have been recognized as
important emerging infectious diseases in the tropics and subtropics around the globe(1).
There are four dengue serotypes, viz. DEN-1, DEN-2, DEN-3 and DEN-4, in circulation
worldwide. Persons living in dengue endemic areas are at risk of four dengue infections
during their lifetime. Some characteristics of the vector, the virus and the host have been
identified as risk factors for the development

of the severe disease, i.e. dengue
haemorrhagic fever (DHF), but the most
important of all is the sequential infection(2,3).

DHF epidemics of 1981 and
1997 in Cuba
Cuba suffered an extensive epidemic of
classical dengue in 1977-78 caused by DEN-1
(not a single case of DHF). This was followed
by a DHF outbreak in 1981, caused by
DEN-2. The 1981 epidemic was the first and the most severe DHF epidemic ever to be recorded in the American region. No DHF or fatal cases were observed in children 1 and 2 years of age. These children were born after the 1977-1978 epidemic and hence was the only group in the Cuban population that did not suffer a secondary infection. Secondary infections were demonstrated in almost 98-99% of individuals (children and adults) with DHF.

After the epidemic was controlled, a campaign to eradicate Aedes aegypti from Cuba was launched. Extensive environmental and chemical measures succeeded in reducing the house index (% houses infested by Aedes aegypti) from well above 10.9% at the beginning of the intensive control operations in August 1981 to 0.007% by April 1984. The degree of control of vectors in Cuba was apparently high enough to interrupt all transmission of dengue. There was no viral activity between 1982 and 1996 in the entire country as evidenced by an effective surveillance system for this disease.

In January 1997, a DEN-2 outbreak was detected in Santiago de Cuba municipality (in an eastern Cuban province). Several risk factors for the re-emergence of dengue were identified. These were: high vector infestation, increased migration of people from endemic countries to the municipality, limited water supply, deficiencies in solid waste disposal, and inadequate vector control activities in the municipality.

A total of 17 259 febrile cases were initially considered to be dengue suspected cases; however, serological studies confirmed the infection in 3012 individuals. Only DEN-2 was recovered by viral isolation on C636 cell line and PCR from the patients studied. Of the serologically confirmed dengue cases there were 205 DHF cases with 12 fatalities. All these DHF cases were classified according to the Guideline for Prevention and Control of Dengue and Dengue Haemorrhagic Fever in the Americas. Fever (100%), haemorrhagic manifestations (100%), headache (91.2%), abdominal pain (86%) and myalgia (75.1%) were observed in the 205 DHF/DSS (dengue shock syndrome) patients. Thrombocytopenia and shock were observed in 99% and 15.1% of the cases respectively, and 100% of the cases presented haemocoagulation. Around 68.7% and 30.2% of the cases presented ascites and pleural effusion. Shock was observed in the 12 fatal cases. 51.2% of cases had hepatomegaly and 38% vomits. The youngest case was 17-year-old and the oldest 66 years. One hundred and two (102) patients were female (49.7%).

The epidemic was completely controlled by August 1997 although the last case was reported in November 1997. May and June were the months with the highest incidence. The vector control measures arrested the spread of the disease to other parts of the country and the epidemic was confined to this municipality only. (Cuba had at that moment 30 out of 169 municipalities where Aedes aegypti was present.)

The Cuban experience is probably unique. In two different epidemics, 16 years apart, which occurred in an immunologically defined population, there was a clear
demonstration that second dengue infections were the most important risk factors leading to DHF. Yet another epidemiological observation revealed that in the 1997 epidemic, DHF cases were observed only in adults. Only one DHF case occurred in an individual younger than 16 years of age. In Cuba, only adults were at risk of a secondary dengue infection because dengue transmission was interrupted in 1981. Correspondingly, all children were at risk of a primary dengue infection. Part of the adult population of the Santiago de Cuba municipality was immune to DEN-1 and therefore at risk of secondary DEN-2 infection. Serological studies demonstrated secondary IgG antibody response in 91.6% (11/12) of the fatal cases and in 98.2% of the 113 DHF studied cases. One interesting and new observation was the possibility that DHF could still occur in an individual who had acquired a secondary infection almost 20 years following a primary infection. To our knowledge, this kind of finding has not been reported before. Data from a dengue surveillance system plus the known low vector mosquito density rates demonstrated that no dengue viruses had circulated in Cuba between 1982-1996 as no flavivirus IgG antibodies were detected in sera from children studied in the dengue surveillance system. From 1989 to 1996, more than 9000 paired sera obtained from persons with febrile illness were tested and no dengue cases were confirmed.

We must emphasize the high severity of both the 1981 and 1997 Cuban epidemics when compared with some others which occurred in the American region. However, when both the epidemics in Cuba are compared, the severity of the disease during the 1997 epidemic is more pronounced. The case-fatality rate (CFR) during the 1997 epidemic was 0.40/100 DF cases and 5.8/100 DHF/DSS cases. On the contrary, the CFR during the 1981 epidemic was 0.046/100 DF cases and 1.5/100 DHF/DSS cases. The rate fatality of 16-year-interval DHF infections (1997 epidemic) was higher than for the 4-year-interval secondary infections (1981 epidemic), suggesting thereby that the risk for DHF persists for many years (perhaps for life) after the first dengue infection; which means that all individuals with previous dengue infections should be aware of the increased risk of DHF when they travel to dengue-endemic areas. Of course, other risk factors for DHF such as race, chronic diseases, the types of viruses that produce both the first and second infections, and, even more important, the genetic characteristics of these viruses, can modulate this risk. In the Cuban experience, in both the epidemics, the sequence of infections was the same, DEN-1 followed by DEN-2. Although the genotype of the second virus was different, both had their origin in Asia.

Finally, another interesting observation that could explain the high severity of these epidemics was the ethnic groups of the people involved. During the 1981 DHF epidemic, white people were considered a risk factor for the severe form of the disease. DHF/DSS, both among children and adults, was significantly higher among the Whites (p<0.01). In the second epidemic, in 1997, once again, Whites were a risk factor for the severe form of the disease. Of the total 169 DHF/DSS cases reported, 46.7% were Whites, 35.5% Mulattos and only 16% Blacks. Racially, the population ratio in
Santiago de Cuba is 30.1% Whites, 42.8% Mulatto and 26.8% Black. Among the 12 fatal cases, two were Blacks (16.6%)\(^9\).

Since 1997, no dengue circulation has been documented on the island. A strong entomological and seroepidemiological surveillance system has been established and only imported dengue cases have been reported. The campaign to control and eradicate Aedes aegypti is being maintained. Finally, we want to record that the 1997 epidemic which occurred in a municipality of 475,580 inhabitants was thoroughly studied. Clinical records were reviewed by experts. The table 1 includes key observations from this outbreak.

**Table 1.** Summary of the main observations of dengue in Cuba (1977-97)

<table>
<thead>
<tr>
<th>Year</th>
<th>Total number of cases</th>
<th>DHF/DSS cases</th>
<th>Fatalities</th>
<th>Viral agent</th>
<th>Genotype</th>
<th>Population at risk of secondary infection</th>
<th>Interval of infection</th>
</tr>
</thead>
<tbody>
<tr>
<td>1977</td>
<td>500 000</td>
<td>None</td>
<td>None</td>
<td>DEN-1</td>
<td>American</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>1981</td>
<td>344 203</td>
<td>10 312</td>
<td>158 (101 children)</td>
<td>DEN-2</td>
<td>South Asia</td>
<td>44.46% (^*)</td>
<td>4 years</td>
</tr>
<tr>
<td>1997</td>
<td>3 012</td>
<td>205</td>
<td>12 (adults)</td>
<td>DEN-2</td>
<td>South Asia</td>
<td>18.5% (^**)</td>
<td>16-20 years</td>
</tr>
</tbody>
</table>

\(^*\) Of the Cuban population  
\(^**\) Of the Santiago de Cuba population

Finally, we want to call the attention of the international community to the fact that it is possible to control dengue when the principles established by PAHO/WHO for the purpose are strictly followed. Cuba has done it twice.

**References**


