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Recent epidemics of yellow fever in eastern Africa have stimulated serological surveys in the Karamoja district of Uganda, the northern frontier district of Kenya and the Glinkhar district of Somalia. All sera collected in the surveys were screened for group B arbovirus antibody using the HI test. Yellow fever immunity was confirmed by the mouse-protection test.

No yellow fever immunity was found in sera collected from a residential tribal group at Karamoja but a small number of samples from persons who had previously lived outside the district showed immunity. Immunity was detected in sera from areas of the Northern Frontier district of Kenya. It is thought that this immunity may, in some areas, have resulted from extensions of the Ethiopian epidemic of 1960-62 into the region, but in another area of the district the immunity seems to have arisen from continuing focal transmission within Kenya. Yellow fever immunity was also detected in Somalia.

The recent epidemics of yellow fever in Ethiopia and Senegal have stimulated a renewed international concern with the epidemiology and control of yellow fever in Africa (WHO Chronicle, 1967). In 1959 an outbreak of yellow fever occurred in the Blue Nile region of the Sudan, with 114 cases reported, and the adjacent area of western Ethiopia, with 237 cases (Berdonneau et al., 1961). From 1960 to 1962, the largest epidemic of yellow fever ever recorded in Africa occurred in south-western Ethiopia. The number of cases has been estimated at over 206,000 and deaths at 30,000 (Série et al., 1964). An epidemic of yellow fever took place in Senegal during the last 3 months of 1965; the number of cases has been variously estimated as 2000-20,000 with a 15% mortality (Chambon et al., 1967).

Although yellow fever epidemics have been periodically reported from West Africa since the eighteenth century, the first definite evidence of yellow fever in eastern Africa was found during an immunity survey reported by Sawyer & Whitman (1936). Yellow fever immunity was detected in the Sudan, Kenya, Uganda and Tanzania. Highest percentages of immunity (20%-40%) were recorded in some areas of the Sudan, and subsequently, during 1940, the first yellow fever epidemic in eastern Africa occurred in one of these areas—the Nuba Mountain region—with 1500 estimated deaths (Mahaffy et al., 1941).

In 1939, a yellow fever research station was established in Buumba County, western Uganda, near where high rates of immunity had also been detected by Sawyer & Whitman. During the decade following the Nuba Mountain epidemic, workers elucidated the forest cycle of mosquito–monkey yellow fever transmission in Buumba. Additional human and primate surveys were conducted over much of eastern Africa, and immunity was found throughout Uganda, and in parts of Somalia, Ethiopia and Kenya (Mahaffy et al., 1946; Smithburn et al., 1949; Haddow et al., 1951; Haddow, 1952). The levels of immunity detected were generally low and until 1959 cases of yellow fever were rarely reported in eastern Africa (Ross et al., 1953; Haddow, 1965).

The reappearance of epidemic yellow fever in eastern Africa, after almost 20 years, and the per-

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sistence of virus activity in Ethiopia, indicated a need for a re-evaluation of the distribution of yellow fever immunity in eastern Africa. During 1967, yellow fever immunity surveys were conducted in north-eastern Uganda and northern Kenya. This report details the results of serological tests on these sera and some sera obtained from eastern Somalia during 1966.

MATERIALS AND METHODS

Survey areas

In Uganda and Kenya the serological survey was made in the areas geographically adjacent to Ethiopia. During August and September 1967, sera were collected from patients attending government dispensaries at four locations in the Karamoja district of north-eastern Uganda and at five locations in the Northern Frontier district of Kenya (Fig. 1). The regions sampled are characterized by arid bush savanna, largely inhabited by nomadic cattle herders. Northern Kenya and Uganda are separated by a range of mountains running north to south from the Sudan to Lake Victoria. The Kenya Northern Frontier district is again separated by Lake Rudolf into a western area inhabited by pastoral and nomadic tribes and an eastern, more arid and semi-desert region inhabited by a number of nomadic tribes some of which extend beyond the Kenya border into Ethiopia or Somalia (Fig. 2).

During February 1966, sera were collected from three tribal groups residing in the Giohar district of Somalia, north of Mogadishu, and west of the Scebele River (Fig. 1). These tribal groups are also nomadic herders.

Collection and storage of blood and serum

Sterile vacuum tubes were used to collect venous blood. The blood was kept cool on ice until the serum separated; the serum was then stored at $-20^\circ$C, prior to testing.

Serological tests

Haemagglutination-inhibition (HI) tests were performed according to the method of Clarke & Casals (1958) in micro-titre plates. HI tests were performed using 4–8 units of four arbovirus group B antigens: West Nile (MP 22), yellow fever (French neuro-

FIG. 1
REGION OF YELLOW FEVER EPIDEMIC $^a$ IN ETHIOPIA IN 1960-62 AND LOCATION OF IMMUNITY SURVEYS $^b$ IN 1966-67

$^a$ Shaded areas.

$^b$ 1, Moroto; 2, Kangole; 3, Kotido; 4, Kaabong; 5, Lokichokio; 6, Lokitaung; 7, Lodwar; 8, Marsabit; 9, Moyale; 10, Giohar.
tropic), Banzi (H 336) and Zika (MR 766). Complement-fixation (CF) tests were done using West Nile and yellow fever antigens on all sera reacting with one or more of the test HI antigens (group B, HI positive). CF tests were conducted in microtitre plates using 5 units of complement (Casey, 1965).

If sufficient quantities of serum were available, yellow fever mouse-protection tests were done on all sera with detectable group B antibody in the HI test. Adult mice were inoculated intracerebrally with 0.03 ml of an equal volume suspension of test serum and yellow fever virus (French neurotropic, passage 548). The virus challenge was diluted to contain an estimated 100 LD_{50}. Those sera protecting 5/6, 6/6, 4/5 or 5/5 of the mice challenged were considered to be immune to yellow fever. All other survival ratios were considered to indicate absence of immunity.

RESULTS

Karamoja district, Uganda (Table 1)

A total of 63 sera were collected and tested from children and 182 from adults who were members of the major tribal group of the Karamoja district. Only 4 sera had detectable HI antibody and titres were highest to Banzi or West Nile antigens. One serum had West Nile CF antibody; all 4 were negative (non-immune) in the yellow-fever-protection test.

Sera were also collected and tested from 61 persons who had previously lived outside the Karamoja district. Sera from the 11 children were negative in the HI test. Group B HI antibody was detected in 17 of the 50 sera from adults. Five of these 17 sera reacted only with Zika antigen. Four other sera had West Nile and one had yellow fever CF antibody. Of the 17 HI-positive sera, 7 were yellow-fever-immune. The available information on the individuals from whom these sera were collected and the detailed serological results are presented in Table 2.

Northern frontier district, Kenya (Table 3)

Sera were collected and tested from 166 children and 391 adults in the five dispensaries sampled in the Northern Frontier district. HI group B antibody was found in the sera from 35 children and
### TABLE 1
RESULTS OF SEROLOGICAL TESTS PERFORMED ON SERA COLLECTED IN KARAMOJA DISTRICT, UGANDA

<table>
<thead>
<tr>
<th>Tribe</th>
<th>Dispensary</th>
<th>Children (2-15 years)</th>
<th>Adults (16 years)</th>
<th>Total (all ages)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No. tested</td>
<td>Group B HI positive</td>
<td>No. tested</td>
<td>Group B HI positive</td>
</tr>
<tr>
<td>Karamajong</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Moroto</td>
<td>17</td>
<td>1</td>
<td>40</td>
<td>1</td>
</tr>
<tr>
<td>Kangole</td>
<td>19</td>
<td>0</td>
<td>52</td>
<td>0</td>
</tr>
<tr>
<td>Kotido</td>
<td>14</td>
<td>0</td>
<td>46</td>
<td>2</td>
</tr>
<tr>
<td>Kaabong</td>
<td>13</td>
<td>0</td>
<td>44</td>
<td>0</td>
</tr>
<tr>
<td>All other tribes</td>
<td>All dispensaries</td>
<td>11</td>
<td>0</td>
<td>50</td>
</tr>
</tbody>
</table>

*a* Sera reacting with one or more antigens in an HI test and further tested in yellow fever protection test (PT) with 100 LD₅₀ virus challenge.

### TABLE 2
DETAILED SEROLOGICAL RESULTS ON 7 YELLOW-FEVER-IMMUNE SERA COLLECTED FROM ADULTS IN KARAMOJA DISTRICT, UGANDA

<table>
<thead>
<tr>
<th>Age (years)</th>
<th>Sex</th>
<th>Place of origin</th>
<th>HI a</th>
<th>CF a</th>
<th>YFPT b</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>YF</td>
<td>WN</td>
<td>B</td>
</tr>
<tr>
<td>35</td>
<td>F</td>
<td>Tororo, Uganda</td>
<td>40</td>
<td>40</td>
<td>40</td>
</tr>
<tr>
<td>55</td>
<td>M</td>
<td>Acholi, Uganda</td>
<td>40</td>
<td>10</td>
<td>20</td>
</tr>
<tr>
<td>45</td>
<td>M</td>
<td>Acholi, Uganda</td>
<td>160</td>
<td>20</td>
<td>20</td>
</tr>
<tr>
<td>30</td>
<td>M</td>
<td>Teso, Uganda</td>
<td>40</td>
<td>20</td>
<td>80</td>
</tr>
<tr>
<td>40</td>
<td>M</td>
<td>Kenya</td>
<td>40</td>
<td>40</td>
<td>20</td>
</tr>
<tr>
<td>16</td>
<td>M</td>
<td>Sudan</td>
<td>20</td>
<td>20</td>
<td>20</td>
</tr>
<tr>
<td>23</td>
<td>M</td>
<td>Sudan</td>
<td>160</td>
<td>10</td>
<td>&lt;10</td>
</tr>
</tbody>
</table>

*a* Antibody titres expressed as reciprocal of highest dilution positive. YF = yellow fever; WN = West Nile; B = Banzi; Z = Zika.

*b* Number of mice survived/mice challenged in yellow-fever-protection test (PT) with 100 LD₅₀ virus challenge.

### TABLE 3
RESULTS OF SERA TESTED FROM NORTHERN FRONTIER DISTRICT, KENYA

<table>
<thead>
<tr>
<th>Dispensary</th>
<th>Children (2-15 years)</th>
<th>Adults (16 years)</th>
<th>Total (all ages)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No. tested</td>
<td>Group B HI positive</td>
<td>CF positive a</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>WN</td>
</tr>
<tr>
<td>Lokitaung</td>
<td>25</td>
<td>10</td>
<td>5</td>
</tr>
<tr>
<td>Lokichokio</td>
<td>32</td>
<td>7</td>
<td>4</td>
</tr>
<tr>
<td>Lodwar</td>
<td>34</td>
<td>15</td>
<td>15</td>
</tr>
<tr>
<td>Marsabit</td>
<td>29</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>Moyale</td>
<td>46</td>
<td>2</td>
<td>0</td>
</tr>
<tr>
<td>Total</td>
<td>166</td>
<td>35</td>
<td>24</td>
</tr>
</tbody>
</table>

*a* Complement-fixing antibody titre >1/5 to the antigen listed and <1/5 to the other antigen. WN = West Nile; YF = yellow fever.

*b* Sera reacting with one or more antigens in an HI test and further tested in yellow fever protection test (PT) with 100 LD₅₀ virus challenge.
TABLE 4
RESULTS OF SEROLOGICAL TESTS ON SERA COLLECTED FROM CHILDREN IN NORTHERN FRONTIER DISTRICT, KENYA, AND FOUND TO BE IMMUNE TO YELLOW FEVER

<table>
<thead>
<tr>
<th>Age</th>
<th>Sex</th>
<th>Dispensary</th>
<th>HI a</th>
<th>CF a</th>
<th>YFPT b</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>YF</td>
<td>WN</td>
<td>B</td>
</tr>
<tr>
<td>11</td>
<td>M</td>
<td>Marsabit</td>
<td>40</td>
<td>10</td>
<td>10</td>
</tr>
<tr>
<td>6</td>
<td>F</td>
<td>Moyale</td>
<td>20</td>
<td>10</td>
<td>10</td>
</tr>
<tr>
<td>14</td>
<td>M</td>
<td>Lokichokio</td>
<td>160</td>
<td>80</td>
<td>160</td>
</tr>
<tr>
<td>10</td>
<td>M</td>
<td>Lokitaung</td>
<td>20</td>
<td>10</td>
<td>10</td>
</tr>
<tr>
<td>10 c</td>
<td>M</td>
<td>Lokitaung</td>
<td>40</td>
<td>40</td>
<td>40</td>
</tr>
</tbody>
</table>

a Antibody titres expressed as reciprocal of highest dilution positive.
YF = yellow fever; WN = West Nile; B = Banz; Z = Zika.
b Number of mice survived/mice challenged in yellow fever protection test (PT) with 100 LD_{50} virus challenge.
c Originally lived in Ethiopia.

154 adults. The majority of HI-positive sera had at least fourfold higher antibody titres with West Nile and yellow fever than with Zika or Banz.
The highest percentage of group B antibody was found in Lokitaung (57.7) and Lodwar (47.5) west of Lake Rudolf.

There were 71 sera with West Nile and 16 with yellow fever CF antibody. No serum had CF antibody to both antigens. All but one of the West Nile CF-positive sera were collected from the three dispensaries west of Lake Rudolf—Lokitaung, Lodwar and Lokichokio. Approximately half of the group B HI-positive sera from these dispensaries had West Nile CF antibody.

Of the 189 group B positive sera, 53 were immune in the mouse-protection test with yellow fever. All the sera with yellow fever CF antibody were immune; sera with West Nile CF antibody were non-immune. Sera from 5 children in four dispensaries were immune (Table 4). These children ranged in age from 6 to 14 years. One child, a 10-year-old male, had originally lived in Ethiopia. Unfortunately, it was not possible to ascertain when he moved to Lokitaung. Sera from 48 adults were immune and 13 of these sera had yellow fever CF antibody.

The highest percentage of yellow-fever-immune sera was collected in Lokitaung (14.6) and Marsabit (14.3). The percentage for Lodwar (7.5) and Moyale (7.6) was similar, and the lowest rate was in Lokichokio (1.0).

In Lokitaung, Lokichokio and Lodwar, all but one of the positive sera collected were from the dominant tribal group—the Turkana. However, in Marsabit and Moyale, individuals of 13 tribal groups were sampled. The largest single tribal group sampled was the Boran, with 63 sera collected in Moyale and 50 in Marsabit (Table 5). At least 15 sera were collected from three other tribes combining the collections from Marsabit and Moyale. Although the numbers sampled are small from tribes other than the Boran, all the yellow-

TABLE 5
NUMBER OF YELLOW-FEVER-IMMUNE PERSONS IN TRIBAL GROUPS SAMPLED IN MOYALE AND MARSABIT, NORTHERN FRONTIER DISTRICT, KENYA

<table>
<thead>
<tr>
<th>Tribe</th>
<th>Dispensary</th>
<th>No. tested</th>
<th>No. YF a Immune</th>
</tr>
</thead>
<tbody>
<tr>
<td>Boran</td>
<td>Moyale</td>
<td>63</td>
<td>5</td>
</tr>
<tr>
<td></td>
<td>Marsabit</td>
<td>50</td>
<td>10</td>
</tr>
<tr>
<td>Burji</td>
<td>Moyale</td>
<td>6</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>Marsabit</td>
<td>9</td>
<td>2</td>
</tr>
<tr>
<td>Gabbra</td>
<td>Moyale</td>
<td>10</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>Marsabit</td>
<td>5</td>
<td>0</td>
</tr>
<tr>
<td>Rendille</td>
<td>Marsabit</td>
<td>16</td>
<td>4</td>
</tr>
<tr>
<td>Others b</td>
<td>Moyale</td>
<td>13</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>Marsabit</td>
<td>32</td>
<td>0</td>
</tr>
</tbody>
</table>

a Determined by yellow fever mouse-protection test.
b Includes individuals of the following tribal groups:
Moyale: Somali, Sakuye and Turkana;
Marsabit: Samburu, Masai, Kamba, Somali, Meru, Embu and Achioli.
fever-immune sera from Marsabit were found in members of the Boran (10), Burji (2) and Rendille (4) tribes; in Moyale the yellow-fever-immune sera were collected from members of the Boran (5), Somalia (1) and Sakuye (1) tribes.

**Giohar district, Somalia (Table 6)**

A total of 242 sera from adults were tested from three tribal groups sampled in the Giohar district, Somalia. More than half of the sera had HI antibody to one or more of the test antigens. Most of these HI-positive sera were broadly reactive with all four antigens; however, 7 sera had only West Nile HI antibody, 3 Zika antibody, 3 yellow fever antibody and 1 Banzi antibody.

Of the 129 group B positive sera, there was sufficient serum for the CF test on 88; of these 17 sera had West Nile CF antibody and none had yellow fever CF antibody. Only 71 of the 78 group B HI-positive sera from the Sciavella tribe could be studied in the mouse-protection test; there were 6 immune sera, or 4% of the total collected from this tribe. The 51 group B HI-positive sera from the Abigal and Scidle tribes could not be tested as sufficient quantities were not available.

**DISCUSSION**

In recent years there have been certain technical alterations in the methodology of yellow fever immunity surveys. Originally yellow fever mouse-protection tests were conducted using 10%–20% virus suspensions (Sawyer & Lloyd, 1931) and later a 1% suspension (Mahaffy, Smithburn & Hughes, 1946) inoculated by the intraperitoneal route. The test has since been modified and the virus challenge is diluted to contain 100 LD50 and inoculated by the intracerebral route (WHO Expert Committee on Yellow Fever Vaccine, 1957). In our study, the HI test developed by Clarke & Casals (1958) is utilized to define rapidly those sera with group B antibody, and only these sera are used in the mouse-protection test. Finally, the CF test is used to attempt further definition of the group B antibody detected in the more broadly reactive HI test and to give some indication of the length of time since infection. The end result of the combination of serological test methods is to give more detailed information on the individual specimen; however, the earlier and the present methods should be similar in specificity and sensitivity as used for detection of yellow fever immunity.

There is no documented evidence of yellow fever vaccination in the geographical regions included in this report. Vaccination was done in southern Ethiopia following the 1960–62 epidemic. However, we did not obtain evidence that any of the tribal groups sampled during the survey of the Kenya Northern Frontier district actually were immunized during this campaign. Therefore, the yellow fever immunity detected is felt to be the result of natural infection.

There has been one previous immunity survey in Karamoja; in 1950 samples were collected from 280 individuals and sera from 7 adult members of the Karamajong tribe were immune (East African Virus Research Institute, 1951; and unpublished records). In contrast, there were no yellow-fever-immune sera detected among the 245 members of this tribal group sampled during the survey under discussion. In Karamoja it is probable that human infection with yellow fever has rarely, if ever, occurred in recent years. However, there were 7 individuals from other areas of Uganda, Kenya and the Sudan who were immune, indicating one possible mechanism by which yellow fever could be introduced into Karamoja.

Previous human survey studies in Kenya have been reviewed (East African Virus Research Institute, 1953). Of more than 1000 serum samples collected during 1933–51 in west, central and coastal Kenya only 1 yellow-fever-immune serum was found that could not be attributed to prior vac-
immunity. The Northern Frontier district of Kenya has not been included in previous surveys.

Three dispensaries were sampled in that part of the Northern Frontier district which lies between Uganda and Lake Rudolf. The serological pattern of the sera collected in this region indicates that the population has been exposed to at least two group B viruses, West Nile and yellow fever. Yellow fever immunity was detected in the sera of children at each dispensary indicating that yellow fever infection has occurred in these populations recently.

The percentage of yellow fever immune sera is almost twice as high in Lokitaung (14.6) as in Lodwar (7.5) and is lowest in Lokichokio (1.0). Lokitaung is situated at the north end of Lake Rudolf close to the Ethiopian border. Following the yellow fever epidemic in Ethiopia, serological surveys were carried out in various directions away from the epicenter focus in the Omo River valley (Serié, personal communication). Yellow fever immunity was detected as far south as Kalam, which lies close to the Omo River and is less than 100 miles (160 km) north of Lokitaung. It appears likely that the immunity detected at Lokitaung, and possibly that found in the other two dispensaries, resulted from extension of the Ethiopian yellow fever epidemic into this region of Kenya. Consideration must also be given to the possibility that yellow fever virus activity occurred previously near Lake Rudolf.

The group B antibody detected in the sera collected from the two dispensaries east of Lake Rudolf—Marsabit and Moyale—was primarily due to yellow fever. In this region of the Northern Frontier district tribal affiliation largely determines the geographical limits of population movements.

Referring back to Fig. 2, it may be seen that the geographical boundary of the Boran tribe extends into Ethiopia. It is therefore possible that some of the yellow-fever-immune sera collected in Marsabit were from individuals who were infected in Ethiopia. However, it seems unlikely that all of the immunity detected in this tribe occurred in this manner as this would necessitate a large and distant migration which to our knowledge does not occur. Although the Burji tribe is Ethiopian in origin, those members of this tribe sampled in Marsabit migrated from Ethiopia at least 10 years ago, and these people do not travel in large numbers across the international border. The Rendille tribe is located entirely within Kenya and separated from Ethiopia by another tribe, the Gabbra. Therefore, although individual members of these tribes may have crossed into Ethiopia and perhaps even have transported yellow fever virus back into northern Kenya, the majority of the yellow fever immunity detected was most likely the result of infection acquired in Kenya. It is not possible to determine from the available information whether natural yellow fever infection was acquired in the immediate vicinity of Marsabit or in some other area of this large region. The number of yellow-fever-immune persons in Moyale was smaller and could have resulted from yellow fever virus infection in Ethiopia or in Kenya.

The only previous yellow fever immunity survey in Somalia was reported by Mahaffy, Smithburn & Hughes (1946). No immunity was detected from small numbers of sera collected at Mogadishu and Kismayu on the coast. One serum was immune from among 27 collected from adults at Villagio, which is near the area of the present survey in the Giohar district. Therefore, the present survey confirms these earlier findings, although without sera from children and adequate amounts of serum for complete serological testing, it is not possible to state how recently yellow fever infection has occurred.

Thus, in summary, interpretation of the findings in the Northern Frontier district is difficult as a number of possibilities have to be considered and there is no earlier survey for comparison. Firstly, although clinical yellow fever has not been reported from the Northern Frontier district, it is our opinion that the immunity detected should be considered as caused by yellow fever virus in view of the close correlation of the three test techniques and in the absence of any other known group B arbovirus that may give a similar immunity. Secondly, while some of the yellow-fever-immune individuals may have received infection as migrants in Ethiopia it is very unlikely that the high levels of immunity at Lokitaung and Marsabit could have been entirely due to this. Thirdly, in the absence of an earlier survey it is impossible to decide whether the immunity in northern Kenya is associated solely with an extension of the 1960–62 Ethiopian yellow fever epidemic or with separate and long-standing yellow fever virus activity in Kenya. Nevertheless, in view of the high level of immunity—the highest recorded in Kenya—the detection of CF antibodies and immunity in children, as well as the information about tribal immunity, it seems reasonable to conclude that there has been recent virus activity in Kenya as far south and east as Marsabit.
It is evident, therefore, that further information is needed on several points related to the current problem of yellow fever in eastern Africa. Of immediate interest are further serological surveys in the Northern Frontier district to define more precisely the distribution of yellow fever immunity and to discover if there are foci of active virus transmission.

Furthermore, in dry country areas, such as the Northern Frontier district, the cycle of yellow fever virus circulation may be different from that of the monkey–mosquito cycle found in the forest. Hadlow, Williams & Mims (1955) reported on primate surveys in the Karamoja district of Uganda. Yellow fever immunity was not detected in 28 monkeys but 23 out of 147 bush-babies (*Galago senegalensis Geoffroy*) were immune. Different host and possibly different vector systems may result in new problems related to virus survival and dispersion; the epidemiology of yellow fever in dry country areas needs to be re-evaluated in the light of recent evidence of human infection.

More serological surveys are needed in Kenya and Somalia to redefine the distribution of yellow fever immunity outside the areas of the present survey. The original purpose of yellow fever immunity surveys in eastern Africa was to determine if this disease could spread from Africa to the more densely populated areas of Asia. Therefore, particular attention should be given to evaluation of yellow fever immunity in the eastern and coastal regions of Kenya and in Somalia.

An effective yellow fever prophylactic vaccine has been available for many years. There is, however, still a lack of the epidemiological and virological information that it needed for most effective use of this vaccine. The international control of yellow fever remains one of the major medical challenges of this century.

ACKNOWLEDGEMENTS

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RÉSUMÉ


Des sérum d'habitants ont été examinés en épreuve d'inhibition de l'hémagglutination (IH) en présence de 4 arbovirus du groupe B: virus amaril (souche neurotrope française), virus Banzi, West Nile et Zika. Tous les spécimens réagissant en IH avec un ou plusieurs de ces antigènes ont fait l'objet d'une réaction de fixation du complément avec les virus amaril et West Nile. Enfin, dans la mesure du possible, on a également pratiqué des épreuves de protection de la souris.

Dans le district de Karamoja (nord-est de l'Ouganda), sur 63 sérum d'enfants et 182 sérum d'adultes, 4 seulement renfermaient des anticorps IH pour les virus du groupe B. Les épreuves de protection sont restées négatives. L'examen de séums prélevés chez 11 enfants et 50 adultes ayant séjourné en dehors du district a montré la présence d'anticorps IH pour les virus du groupe B chez 17 adultes. Sept séums positifs vis-à-vis du virus amaril seul donnaient aussi un résultat positif en épreuve de protection de la souris.

Dans un district du nord du Kenya, 35 séums d'enfants sur 166 et 154 séums d'adultes sur 391 présentaient des anticorps IH pour les virus du groupe B. Parmi les 189 séums positifs, 53 faisaient preuve d'un pouvoir protecteur chez la souris. Les pourcentages les plus élevés de séums immuns pour le virus amaril ont été relevés à Lokitaung (14,6), à Marsabit (14,3), à Moyale (7,6) et à Lodwar (7,5).

Dans le district de Gihor (Somalie), plus de la moitié (129) des séums prélevés chez 242 adultes renfermaient des anticorps IH pour un ou plusieurs virus du groupe B.
Les épreuves de protection pratiquées sur 71 sérums se sont révélées positives dans 6 cas (4%).
Selon les auteurs, la présence d'une immunité anti-amarile dans certains villages du nord du Kenya est vraisemblablement due à la pénétration du virus à partir de l'Ethiopie lors de la poussée épidémique observée dans ce pays en 1960-1962. Dans la région de Marsabit, dont les habitants n'ont aucun contact avec la population de l'Ethiopie méridionale, tout plaide en revanche en faveur d'infections amariles récentes acquises au Kenya même.

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