BRIEF COMMUNICATIONS

ADAPTATION OF A NIGERIAN STRAIN OF PLASMODIUM FALCIPARUM TO PANAMANIAN AOTUS TRIVIRGATUS*

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Abstract. A patent infection (more than 190 days duration) with Plasmodium falciparum, obtained directly from a patient who had been exposed to malaria in Nigeria, was established in a splenectomized Panamanian owl monkey, Aotus trivirgatus griseimembra. Subinoculations from the original monkey recipient into one splenectomized and two normal A. t. griseimembra produced primary patent parasitemias of 18–54 days duration. These results represent the first successful adaptation of P. falciparum from man to a monkey of Panamanian origin.

The failure to establish lines of Plasmodium falciparum directly from man in Panamanian nonhuman primates has been detailed and summarized.1,2 Porter and Young,3 using Saguinus geoffroyi, reported 4 to 15-day patent periods following inoculation of infected human blood, but a single attempt to transfer the infection to a second S. geoffroyi was unsuccessful. However, after as few as two passages in Aotus trivirgatus of Colombian origin, the Panama II strain of P. falciparum proved to be infective for Panamanian A. trivirgatus and subsequently for Alouatta villosa trabeata.4 The present report presents the establishment of a Nigerian falciparum strain in Panamanian A. trivirgatus.

MATERIALS AND METHODS

The monkeys used in this study were A. trivirgatus griseimembra captured in Panama.5 Routine laboratory procedures concerning monkey husbandry and blood smear preparation have been published previously.6

The patient (C.H.), from whom infected blood was obtained, stayed in Lagos, Nigeria from 5 April to 12 April 1979, and in Madrid, Spain from 12 April to 15 April 1979. He returned to Panama on 15 April. No antimalarial drugs were taken during the trip. The onset of symptoms of fever, chills, and headache began on 20 April 1979, and 3 days later a diagnosis of a P. falciparum infection was made by blood film examination. Within 1 hour of the diagnosis, 9 ml of citrated blood (1,900 × 10⁶ parasites) were drawn and inoculated intraperitoneally into a splenectomized A. t. griseimembra (9442).

RESULTS

Data on the course of infections in A. t. griseimembra are shown in Table 1. During the extended patent period in the initial recipient (9442), five peak parasitemias were noted, the highest of which occurred 2 months after the initiation of patency. Subinoculation to a second splenectomized monkey (9382) on patent day 19 produced a less virulent infection than in the donor, as indicated by the shorter duration of patency, lower maximum parasitemia, and fewer recrudescences.

Contrasting parasitemias occurred in two unaltered recipients, monkeys 9445 and 9455 (Table 1). Parasites in 9445 never became countable by the Earle-Perez method, but were detected on thick blood films for 18 days. The parasitemia in 9455 was patent for 10 days longer than in 9445, and achieved a maximum count of 2,730 parasites per mm³. Both animals were splenectomized when parasite-negative on thick blood films; a total of three recrudescences ensued in 9445, the first one beginning on day 13 post-splenectomy. Aotus 9455 died 7 days post-surgery, and remained parasite negative.

Although gametocytes have been observed, they did not appear to be mature.

Accepted 5 July 1980.

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TABLE 1

Infection parameters of Plasmodium falciparum (Nigerian strain) in Aotus trivirgatus griseimembra

<table>
<thead>
<tr>
<th>Monkey no</th>
<th>No. parasites mm3 × 106</th>
<th>Route</th>
<th>Alteration</th>
<th>No. days prepat. and (patent)</th>
<th>Parent period idays</th>
<th>Maximum parasitemia</th>
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<tbody>
<tr>
<td>9442</td>
<td>1,900</td>
<td>ip</td>
<td>Splenectomy</td>
<td>11</td>
<td></td>
<td></td>
</tr>
<tr>
<td>9382†</td>
<td>18</td>
<td>iv</td>
<td>Splenectomy</td>
<td>(9)</td>
<td>192</td>
<td>47,500</td>
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<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>(16)</td>
<td>39</td>
<td>1,020</td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>33</td>
</tr>
<tr>
<td>9445†</td>
<td>0.4</td>
<td>iv</td>
<td>None</td>
<td>1</td>
<td>54</td>
<td>299,340</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Splenectomy‡</td>
<td>(26)</td>
<td>51</td>
<td>234,390</td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>112</td>
</tr>
<tr>
<td>9455‡</td>
<td>48</td>
<td>iv</td>
<td>None§</td>
<td>1</td>
<td>28</td>
<td>317,660</td>
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</table>

* ip, intraperitoneal; iv, intravenous
† Submucosalized from monkey no. 9442
‡ On day 37 post-inoculation
§ Splenectomized on day 31 post-inoculation; died on day 1 post-surgery

DISCUSSION

Collins et al.7 also adapted a Nigerian P. falciparum strain in splenectomized A. t. griseimembra. The primary passage monkey probably originated from Colombia (Collins, personal communication). Infections of our Nigerian strain in Panamanian A. t. griseimembra were less severe than those reported by Collins et al.; none of our monkeys died of malaria-related causes, and chemotherapeutic intervention was not required to sustain the experimental host.

Data presented herein document the first instance of a long term parasitemia in Panamanian A. t. griseimembra of P. falciparum derived directly from a human infection and passage to subsequent recipients.

We are unable to explain the factors contributing to the successful adaptation of an African strain in a Panamanian monkey in contrast to the numerous failures associated with indigenous falciparum strains. A detailed biochemical analysis might reveal differences between strains, leading to adaptation by the former but not the latter.

REFERENCES


