THE INFECTIVITY OF PRIMATE MALARIA INFECTION P.CYNOMOLGI IN ITS NATURAL HOST IS REGULATED BY TRANSMISSION BLOCKING IMMUNITY

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Regulation of the infectivity of malaria infections by naturally acquired transmission blocking immunity was investigated in a natural host-parasite system P.cynomolgi ceylonensis in the toque monkey Macaca sinica. The course of a blood induced infection consisted of a peak of parasitaemia with patency lasting about two weeks. The infectivity of an infection during this period was assessed by directly feeding mosquitoes on the monkey. The intrinsic infectivity of gametocytes during the patent infection, and transmission blocking immunity levels in serum up to about 4 months after infection were assessed in membrane feeding experiments.

Direct feeding experiments revealed that the absolute infectivity was greatest during the second, third and fourth days of patent parasitaemia; thereafter the infectivity declined rapidly despite a rising gametocytaemia. Membrane feeding experiments revealed the presence of infectivity enhancing antibodies in the serum during the early infection corresponding to the period of highest absolute infectivity. Transmission blocking effects were first apparent in the serum about a week after patency and reached peak levels during the next few days. With time, blocking effects of the serum were gradually lost and replaced by enhancing effects again; after about 3 months no blocking or enhancing activity could be detected in the serum.

Transmission blocking immunity was "boosted" and the duration of immunity was prolonged if the monkey was re-inoculated with parasites within 6 weeks, after the primary infection became sub-patent.

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