

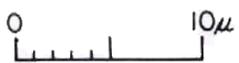
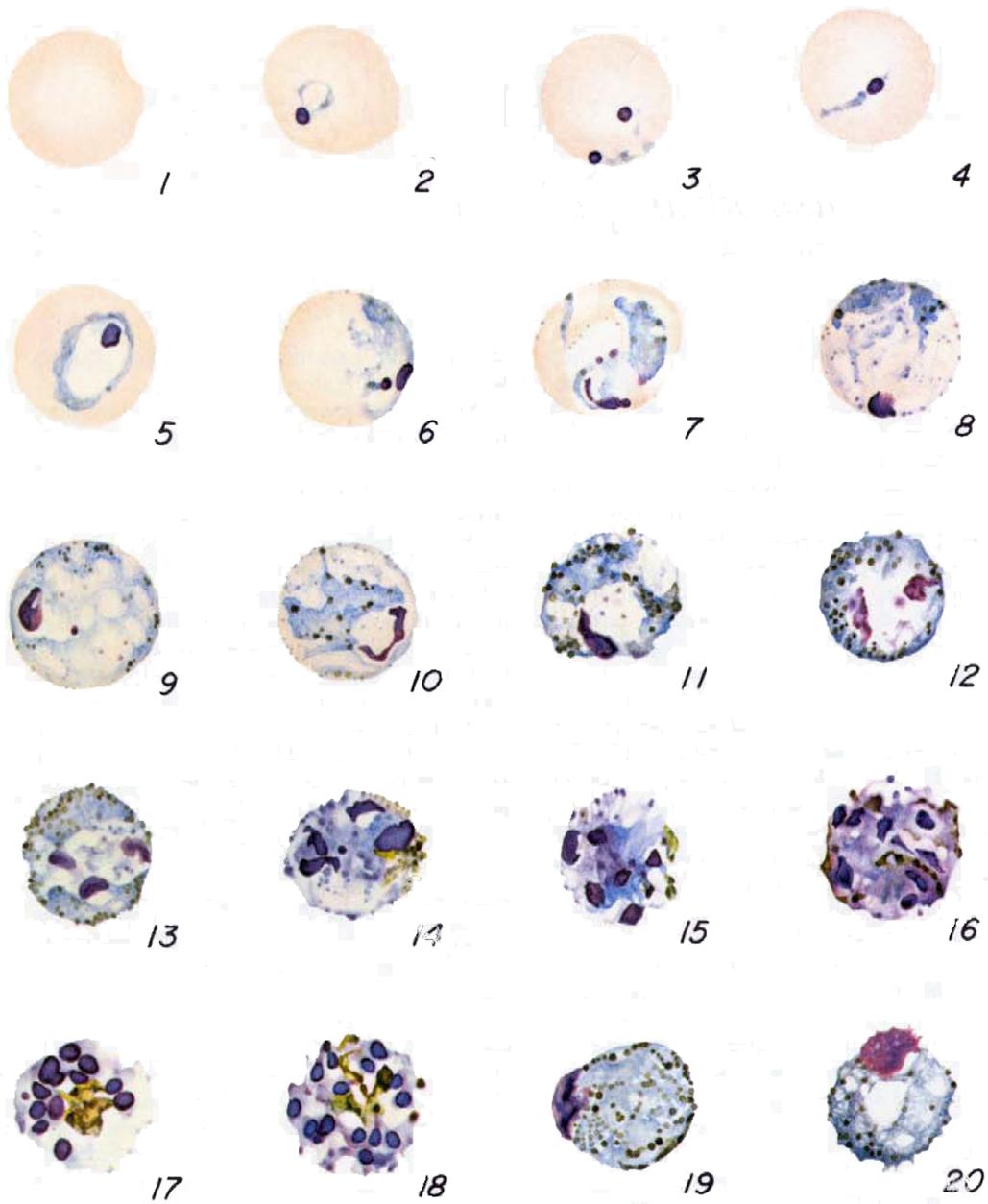
Plasmodium pitheci Halberstaedter and
von Prowazek, 1907

THIS was probably the first true simian plasmodium to be seen and described. Laveran, in 1905, mentions a blood parasite seen in smears taken from an orangutan in Paris. In 1907, Halberstaedter and von Prowazek, working in Borneo, described the parasite they found in the orangutan and named it *Plasmodium pitheci*. Shibayama (1910) saw the parasite in the blood of an orangutan imported to Japan and remarked that Schüffner's dots were not present but, as Wenyon (1926) pointed out, his figures indicate that he used a weak stain which could account for the lack of Schüffner's dots. Dodd (1913) reported the death of an orangutan in the Zoological Gardens of New South Wales as probably due to *Haemoproteus* (= *Plasmodium*) *pitheci*. In 1920, Reichenow put forward the idea, but without conviction, that maybe *P. pitheci* was actually a human parasite although Koch (1900) had failed in an attempt to infect the orangutan and the gibbon with *P. vivax* or with *P. falciparum*. Donovan (1921) carried out a study of blood parasites in simians at the foot of the Nilgiri hills in southern India and reported finding *P. pitheci* in the orangutan. This finding is open to question because, as all authorities agree, these animals occur on Sumatra and Borneo only, although, according to Darlington (1957), they were in India during the Lower Pleistocene era.

The parasite was not seen again until one of us (McWW) visited Borneo in 1966 and while there succeeded in obtaining blood smears from 18 orangutans; ten of the animals harbored *P. pitheci*. The description of the blood stages of

the parasite and the color plate is based on that material.

As in any endeavor, success or failure may hang by a slender thread. Such might have been the case in the above instance. In 1966, McWW was given a short-term assignment to study the zoonotic aspects of simian malaria in West Malaysia. During the planning of that work, the desirability of his making a side-trip to Borneo in the vain hope of retrieving the malaria parasite of the orangutan, last seen in 1913, was discussed. It was realized that the Malaysia assignment would be difficult because of the limitation in time and funds, but it was agreed that the trip would be made if at all possible. He went to Malaysia and near the end of his tour he let one of us (GRC) know that it would be difficult for him to complete the original work on schedule and therefore he would probably have to abandon the trip to Borneo. It seemed that to miss this chance of finding *P. pitheci* again would be catastrophic--the effort must be made. A cable was sent to him to the effect: "Go to Borneo or don't come back--letter follows". The letter merely reiterated what we both knew as to the importance of the project and urged him to go to Borneo if at all possible. Upon receipt of the cable, he left immediately for Borneo with excellent results as mentioned earlier. It goes without saying that he finished the original assignment in a highly creditable manner and on schedule. One wonders would he have gone to Borneo sans cable? Who can say--the point is: he did!



G. H. Nicholson

PLASMODIUM PITHECI

Cycle in the Blood

PLATE XVII

The youngest parasites consist of a small amount of bluish-gray cytoplasm associated with a deep purple-staining nucleus. The cytoplasm may appear as a ring or only as an elongate smudge (Fig. 4). Double invasion of the cell is rare but it may occur (Fig. 3). As growth proceeds, there is an increase in the amount of cytoplasm with the nucleus remaining essentially unchanged. The cytoplasm becomes amoeboid. Very light stippling appears in the red cell as the trophozoite approaches maturity. Pigment first appears as a coarse, greenish-brown granule. As growth continues, the increase in pigment is by the addition of more greenish-brown granules. The nucleus stains wine-red as it increases in size. There is little or no host cell enlargement.

The cytoplasm of the mature trophozoite is amoeboid and frequently assumes a web formation which almost fills the cell. The nucleus may be distorted. The stippling is sparse and granular with little or no host cell enlargement. The pigment is greenish-black and appears as uniform coarse granules.

The nuclear divisions are not out of the ordinary. The color of the nuclei changes from wine-red in the early stages, to deep purple in the mature forms. The cytoplasm gradually changes from light blue to a reddish-purple and virtually disappears in the mature forms. Pigment granules tend to gather in one part of the cell and then the granular bodies coalesce into one or more greenish-yellow masses (Fig. 17). Stippling remains sparse and is sometimes inapparent since the developing schizont usually fills the entire host cell with little or no host cell enlargement or distortion. The mature schizont normally has 12 to 14 merozoites (Fig. 18).

The macrogametocyte fills the host cell completely and without enlargement (Fig. 19). The nucleus is large, usually oval, dark-staining and generally found near the periphery of the parasite. The cytoplasm is uniform and stains grayish-blue. The pigment is abundant, coarsely granular, and evenly distributed. The microgametocyte also fills the host cell without increasing its size (Fig. 20). The nucleus is larger than its counterpart in the macrogametocyte and it takes a lighter stain. It is oval to circular and stains wine-red. The bluish-gray staining cytoplasm is frequently vacuolated and lacks the uniform consistency seen in the macrogametocyte. The pigment is coarse, granular, and generally less abundant than in the macrogametocyte.

Sporogonic Cycle

There are no data on the sporogonic cycle.

Cycle in the Tissue

No exoerythrocytic stages are known.

Course of Infection

The course of the infection, as described by the original investigators, runs a chronic course with little if any pathology. The temperature in infected animals was within the normal range. In contrast, Dodd (1913) was of the opinion that the death of an orangutan kept for some 13 months in the Zoological Gardens in Sydney, NSW, was probably due to an overwhelming infection with *P. pitheci*. It is unfortunate that a thorough post mortem was not done on the animal, or, if it was, that the findings should have been limited to a single note stating that the bone marrow of the femur, humerus, and ribs was decidedly congested. None of the infected

PLATE XVII.—*Plasmodium pitheci*.

Fig. 1. Normal red cell.

Figs. 2-4. Young trophozoites.

Figs. 5-9. Growing trophozoites.

Figs. 10-11. Mature trophozoites.

Figs. 12-16. Developing schizonts.

Figs. 17-18. Mature schizonts.

Fig. 19. Mature macrogametocyte.

Fig. 20. Mature microgametocyte.

animals examined by McWW showed any clinical evidence of infection.

Host Specificity

The normal host of *P. pitheci* is the orangutan, *Pongo pygmaeus*. According to Halberstaedter and Prowazek (1907) the parasite could be transferred successfully by blood inoculation to other orangutans but not to gibbons or to monkeys. In an attempt to augment the above findings regarding host specificity, blood obtained from three infected orangutans in Borneo was shipped to the United States and given intravenously to a young splenectomized chimpanzee (#31) on 11 July 1967. The infection became patent on the 21st of September and remained so, always with a low parasite count, until 17 October 1967. On 2 October 1967 parasitized blood was transferred to three different monkeys: an owl (*Aotus*

trivirgatus), a pig-tailed (*Macaca nemestrina*), and a rhesus (*Macaca mulatta*). None of these animals became infected. A similar inoculation into a gibbon was not successful.

Also, during the fall of 1967, parasitized blood was given to a second young splenectomized chimpanzee (#1394), which exhibited a patent infection after 18 days. The parasitemia remained low for ten days whereupon it terminated spontaneously. These limited data tend to show that the parasite does not infect other apes well and does not grow in monkeys. It is hoped that opportunities will be found whereby this parasite, whose host is one of our near relatives evolutionarily, can be given the opportunity to infect man.

The natural invertebrate host is not known and as far as we can learn there have been no attempts to find a mosquito capable of accepting infection with the parasite.

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