

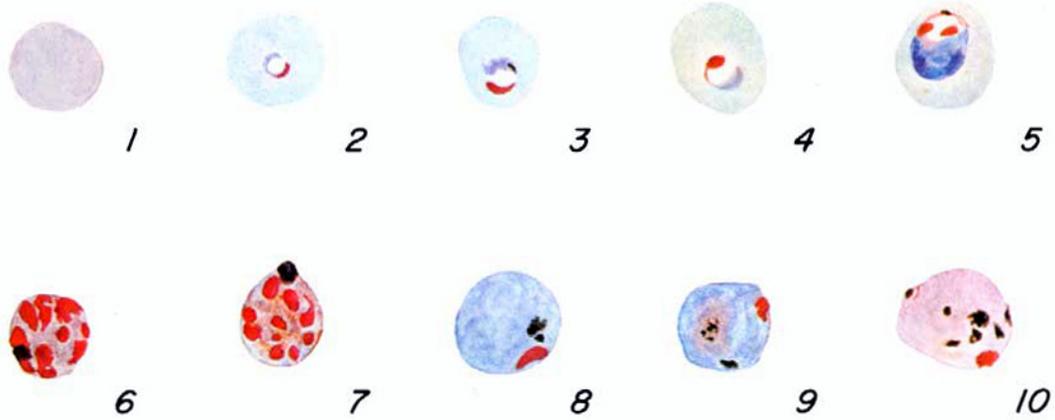
*Plasmodium girardi* Buck, Coudurier, and  
Quesnel, 1952

WORKERS in Madagascar have long suspected that the lemurs were infected with malaria but the parasite was not seen until 1951 when it was discovered in the blood of a *Lemur fulvus rufus*. The parasites were scanty, so the animal was splenectomized. A heavy infection developed 12 days later. The infection was studied daily; after about a week, a second species appeared. Each organism was recognized as a new species. The first one was given the name *Plasmodium girardi* in honor of Dr. G. Girard, the former director of the Pasteur Institute of Tananarive. The other parasite was given the name *Plasmodium foleyi* in honor of Dr. H. Foley of the Pasteur Institute of Algeria. The latter parasite is now considered to be a hepatocystis and therefore it will not be discussed in its entirety here. The description of

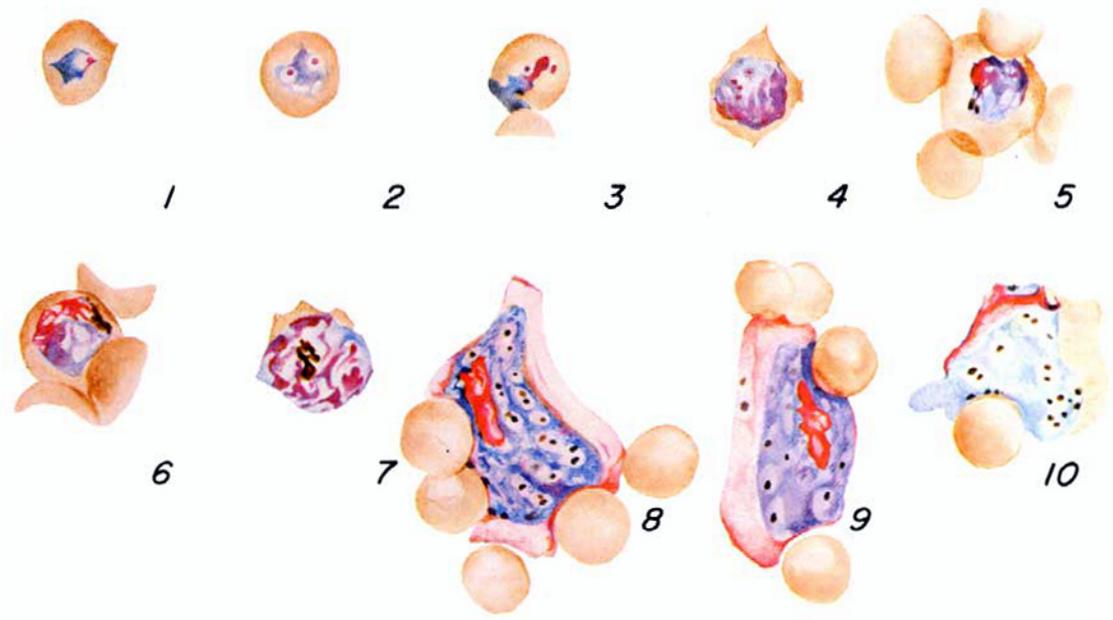
the parasites is accompanied by a beautifully colored plate painted by Dr. Foley.

The lemurs of Madagascar are protected animals and consequently there are few opportunities for studying their parasites. However, a few animals were examined by members of the University of California expedition to Madagascar in 1948, and another malaria parasite (*P. lemuris*) was discovered; it is discussed in Chapter 28.

According to Garnham (1966) the original material on *P. girardi* is no longer available. In 1962, at his request, Drs. Raymond and Brygoo splenectomized two animals; one of them exhibited a low-grade infection with *P. girardi* and that material was used for a more extended study of the parasite. Only the blood stages of the parasite have been seen.



*PLASMODIUM GIRARDI*



*PLASMODIUM LEMURIS*

## Cycle in the Blood

### PLATE LIV

The youngest forms are small solid bodies, approximately 1.8  $\mu$  in diameter, with a large nucleus and a small rim of cytoplasm which stains a deep blue. The youngest stages exhibit a granule of dark pigment which the original authors stated did not appear until the parasite occupied about a fourth of the host cell. The pigment body lies on the edge of the cytoplasm. Projections of cytoplasm may occur but for the most part, the parasite is compact and dense even though a vacuole occurs later.

As the trophozoite grows, it may occupy one-half the cell whereupon it loses its vacuole and the parasite as a whole shrinks; the amount of pigment increases and takes up a position on or near the periphery of the parasite. The nucleus becomes enlarged, sometimes diffuse, but always with lighter and darker areas. Very small vacuoles may be seen in the cytoplasm.

With continued growth, the nucleus divides in to two rather large portions, each with a dense core area. Pigment continues to form and now comprises 4 or 5 dark grains arranged on the periphery of the parasite. As division continues beyond the 4-nucleate stage, the host cell becomes pallid, distorted, and develops fimbriated projections. The mature schizont fills the host cell without enlargement, and displays 10 to 12 merozoites; the pigment is still placed eccentrically. Host cell stippling appears to be absent.

The macrogametocyte is spherical and stains a deep blue with granular cytoplasm and heavy dark pigment in an aggregation of distinct grains. The nucleus is deep red and found near the periphery of the cell. The host cell is not enlarged. The cytoplasm of the microgametocyte takes a lilac stain. The pigment is granular to bacilliform and is located in 4 or 5 aggregates of unequal size. The nucleus takes a deep red stain and is located peripherally. The parasite fills the host cell without host cell enlargement.

It is interesting that cirrhosis of the liver of the host animal was known long before malaria parasites were found in them. Whether this cirrhotic change is due to, or is connected with, their malarias is not known. The spleen appears normal when the infection is latent which makes it appear that these animals handle the infection easily. If the spleen is removed, the infection comes to the fore and then again subsides.

*Plasmodium girardi* may have a distinct place in the evolution of the malarias of primates, but the extent is in limbo until more is known about its life cycle. The blood phase of the infection leads one to suspect that it probably belongs with the quartan type parasites and, if so, it is their most primitive example. One thing appears certain, and that is, that the distribution of the pigment in these parasites is not like what is found in the malarias of simians. We shall have to wait for an explanation as to why this difference occurs.

## REFERENCES

- BUCK, G., COUDURIER, J., and QUESNEL, J. J., 1952. Sur deux nouveaux plasmodium observes chez un lémurien de Madagascar splénectomise. Arch. Institut. Pasteur d'Algerie 30 : 240-243.
- GARNHAM, P. C. C., 1966. Malaria parasites and other haemosporidia. Blackwell Scientific Publications, Oxford, pp.1114.

### PLATE LIV.—*Plasmodium giardi* and *P. lemuris*.

*P. giardi* : Figures after Bück *et al*, 1952.  
Fig. 1. Normal erythrocyte.  
Fig. 2-4. Uninucleate trophozoites.  
Figs. 5-7. Schizonts.  
Figs. 8, 9. Macrogametocytes.  
Fig. 10. Microgametocyte.

*P. lemuris* (1000X) : Figures after Huff and Hoostraal, 1963.  
Figs. 1-5. Uninucleate trophozoites.  
Fig. 6. Binucleate schizont.  
Fig. 7. Multinucleate schizont.  
Figs. 8, 9. Macrogametocytes.  
Fig. 10. Microgametocyte.