parts of the United States; (iv) the onset of the calcium change 1 year after the introduction of chlorinated hydrocarbons into general usage was not a random circumstance; and (v) these persisting compounds are having a serious insidious effect on certain species of birds at the tops of contaminated ecosystems.

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## Plasmodium falciparum: Phagocytosis by Polymorphonuclear Leukocytes

Abstract. *In vitro, the human polymorphonuclear leukocyte can recognize and ingest the free forms of* Plasmodium falciparum. *Digestive vacuoles form about the engulfed organisms. Neither the polymorphonuclear leukocyte nor the monocyte can recognize the parasitized red cell. Phagocytosis of normal or parasitized red cells was not observed.*

Both cellular and humoral elements and events are involved in malaria immunity. The presence of pigment and red cells in the reticuloendothelial cells of the spleen and bone marrow led investigators to consider phagocytosis a most important factor in removing parasites from the blood (*1*). Huff and Weatherby (*2*) first observed phagocytosis of a free schizont of avian malaria

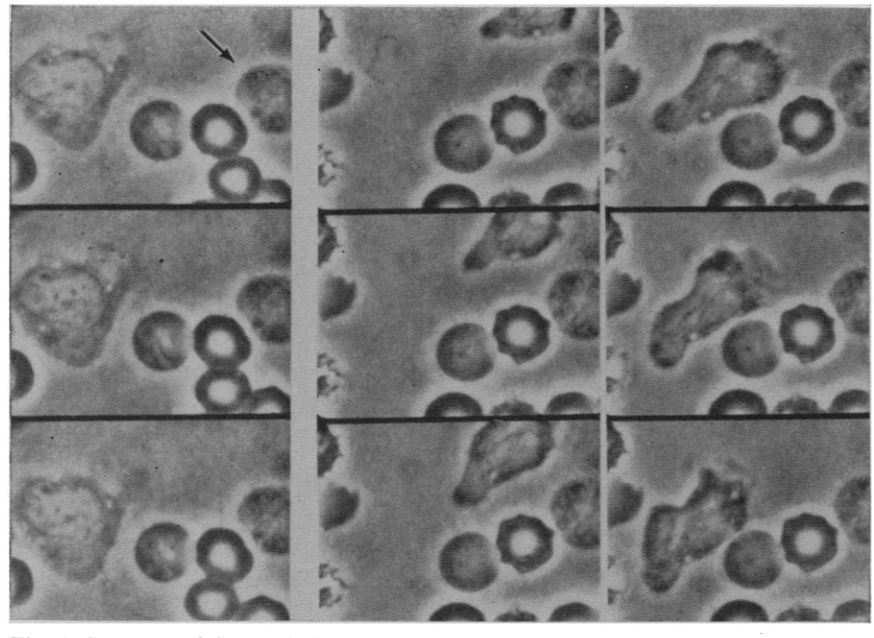

Fig. 1. Sequence of frames is from top down and from left to right. Arrow points to parasitized red cell. In the left column, a monocyte passes the parasitized red cell. In the remaining frames a polymorphonuclear leukocyte moves by the cell. Neither white cell recognizes the diseased red cell (about × 1000).

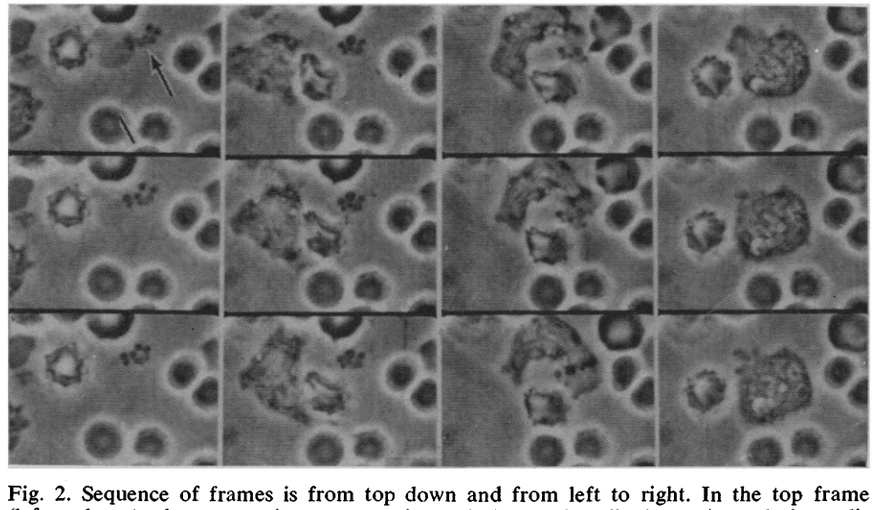

Fig. 2. Sequence of frames is from top down and from left to right. In the top frame (left column) the merozoites escape from lysing red cell (arrow) and immediately attract the polymorphonuclear leukocyte which engulfs and digests them (about × 1000).

by a macrophage in tissue culture. We now report evidence of the phagocytosis of free merozoites of *Plasmodium falciparum* by human polymorphonuclear leukocytes.

Blood was drawn from young men, recently returned from Vietnam, who entered the hospital as a result of a relapse of falciparum malaria. Small amounts of heparin (Panheprin, Abbot) which contained no preservative was used as an anticoagulant. A small drop of blood was placed on a cover slip (22 by 40 mm) sandwiched onto a glass slide and sealed with 360 silicone fluid, 350 centipoise (Dow Corning) (*3*). Cells in such preparations remained viable for many hours and afforded good phase-microscopic images.

The slides were studied on a Leitz microscope equipped with phase optics and time-lapse cinephotomicrographic apparatus (Sage Instruments). The temperature of the preparation was maintained at 36°C with an air-curtain incubator (Sage Instruments). The parasitized red cells were observed under a × 90 phase objective and photographed on Plus X negative 16-mm film (Kodak) at eight frames per minute under tungsten illumination with a green-yellow filter at 5.2 volts, requiring 1.25 seconds for exposure.

The parasite is easily made visible in the red cell by phase microscopy and is a highly motile, freely moving flattened structure. The extreme freedom of motion suggests that the interior of the parasitized red cell is fluid or semifluid. The parasite apparently produces no changes in the red-cell surface that can be recognized by the monocyte or polymorphonuclear leukocyte (Fig. 1). Monocytes and polymorphonuclear leukocytes repeatedly pass within a few red-cell diameters from the parasitized red cell, even up to a few minutes before rupture of the diseased red cell. In Fig. 2, a red cell has ruptured, releasing its merozoites. A polymorphonuclear leukocyte which had passed the red cell moments before the rupture rapidly reversed its direction and in a purposeful fashion returned to ingest the released organisms. Digestive vacuoles appear within moments after the phagocytosis of the merozoites.

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## Red Blood Cells: Why or Why Not?

Abstract. *It is commonly stated that, if hemoglobin were dissolved in the blood plasma rather than enclosed in corpuscles, the viscosity of the blood would be greatly increased. We found that when the corpuscles of dog or goat blood were disrupted with ultrasound, giving a solution with the same hemoglobin concentration, the relative viscosity was drastically reduced. It appears, therefore, that the existence of blood corpuscles does not contribute to a reduced viscosity of blood.*

Some animals carry hemoglobin in blood corpuscles, and others do not. Possible advantages of blood corpuscles have been the subject of much speculation. Animals which possess blood of high oxygen-carrying capacity, notably vertebrates, always have hemoglobin located within corpuscles (*1*). It has therefore been inferred that the existence of corpuscles is a necessary prerequisite for a high oxygen-carrying capacity. One reason often stated is that, compared to the more easily flowing fluid obtained by enclosing the hemoglobin in small packages (the red blood cells), a hemoglobin solution with the same oxygen capacity as mammalian blood would be highly viscous, and therefore would require much greater pumping efforts of the heart. This explanation is found at all levels, from specialized monographs to the elementary textbooks (*2*).

We wanted to know whether this contention is reasonable, but a literature search revealed no experimental evidence. We therefore examined the problem by comparing blood with intact red cells and the same blood after disruption of the cells. Comparison of these two fluids, which have identical concentrations of hemoglobin, showed that the assumed effect of the corpuscles on viscosity is wrong.

Blood was taken from the jugular vein of mongrel dogs and domestic goats with sodium heparin as an anticoagulant (10 unit/ml). A range of hemoglobin concentrations was obtained by withdrawing or adding plasma to the samples. Viscosity at 37°C was determined in these samples, before and after hemolysis, by means of Ostwald viscometers (*3*). The corpuscles were disrupted by ultrasound (Branson Sonifier model W-140C) with a Rosett cell used to prevent overheating. Any cell fragments formed remained in the solution, which we will refer to simply as a hemoglobin solution. Treatment with ultrasound had no effect on the viscosity of plasma alone (*4*). Hemoglobin concentration was measured in all samples, both before and after hemolysis, by the cyanomethemoglobin method (*5*).

Disruption of the corpuscles of dog blood by ultrasound caused a conspicuous decrease in the relative viscosity (Fig. 1). However, at very high concentrations of hemoglobin, outside the normal physiological range (hemoglobin more than approximately 20 g/100 ml), disruption of the corpuscles yielded a hemoglobin solution which gelled, and its viscosity could not be measured.

At the normal hemoglobin concentration of dog blood (14.8 g/100 ml), the relative viscosity of blood with cells was 4.7 (Fig. 1, *A*), and disruption of the cells reduced this to a relative viscosity of 3.3 (Fig. 1, *B*). Thus, if the hemoglobin were carried in solution in the plasma, its contribution to the viscosity would be 47 percent less than it is when carried in cells (as related to plasma viscosity, Fig. 1, *C*).

Disruption of the red cells in goat blood (Fig. 2) caused an even greater reduction in relative viscosity than it did for dog blood. This is primarily because the curve for whole goat blood is located above that of dog blood. The curves for plasma and for the hemolyzed blood are approximately the same for the two animals, and the difference in the whole blood must therefore depend on cell characteristics. Another difference is that very concentrated solutions of goat hemoglobin did not gel as dog blood did. Even at a hemoglobin concentration of 25.2 g/100 ml, the viscosity of the goat hemoglobin solution (relative viscosity, 18.2) was