OBJECT OF PRESENT STUDY

The present investigation is on the host specificity of an avian malaria parasite. Late Dr. H. N. Ray was very inquisitive about the host ranges of different animals. While examining the blood of locally available birds of different groups, he detected the existence of this plasmodium. The parasite was thereafter, maintained in our laboratory by needle passage for the last seven years. It is not similar in all respects, to the existing species of malaria parasites reviewed by Prof. Garnham (1966). Therefore, a new nomenclature was done (Sarkar and Ray, 1969) and their exoerythrocytic forms and erythrocytic forms including gametocytes and possible vectors was described by Sarkar (1968). Subsequent studies showed that the host range of this parasite is very limited. The parasite is only capable of infecting the birds of the same ganus. Only in one instance, there was a low parasitemia found in a single bird of a different genus, and that too by intravenous injection of a heavy dose of parasites.

From the review of the literature presented so far on the study of host parasite relationship in malaria parasite the following points appear relevant.

The host specificity is due to extreme specialized adaptation of the parasite to its host. Different malaria parasites show specificities for different hosts. The cell to cell adjustment may be conditioned by a) metabolic requirements of the parasite, b) cell

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surface variation of the host and parasite, c) nature and type of immunological responses of the host principally and of the parasite secondarily.

The problem of host specificity from the point of metabolic needs could be conveniently studied if a good cultural condition could be established for the parasites *in vitro*. But with the addition of all possible and impossible nutrients, the *in vitro* culture of malaria parasites has not yielded very promising results. But we know a great deal now, about the special requirements of certain malaria parasites and evidently the host erythrocyte and the host plasma must be capable of supplying them to the parasites for effective colonization.

Hemoglobin is one of the chief sources of nutrition for the erythrocytic stages of parasite. Pinocytotic uptake of hemoglobin occurs in the parasite. Two points are relevant in this connection.

Pinocytosis is a relatively specific process; not all compounds are pinocytosed. Does the variation of the protein globin of the hemoglobin molecule determine the pinocytability of the parasite? Or in other words can the parasite distinguish the globin molecule?

The second point worthy of attention is the subsequent fate of the pinocytozed hemoglobin. Of course, the molecule has to be broken up, digested and the constituent amino acids have to

be built up into the proteins of the parasite. It is hardly likely that the intracellular proteolytic enzymes would be able to differentiate between minor variations in the globin molecule and it is also hardly likely that there would be a disproportion of the individual constituent amino acids, gross enough to affect biosynthesis of parasitic proteins. These considerations, therefore, point out the possibility that if a parasite can utilize a particular hemoglobin and cannot utilize a related molecule, the difficulty must be with the pinocytosis, which is presumably a surface property of the parasite.

Entry of a parasite into the host cell possibly depends on the complementarity of the host cell membrane to the cell membrane of the parasite. When an electron micrograph is taken at the time of approach of a malaria parasite towards the cell of the susceptible host, two peculiar structures a paired organelles and conoid structure are seen in the parasite. These two structures disappear after the parasite entered the cell. It was speculated that these two structures had some role to play in the penetration of the host cell membrane by the parasite. Whether the penetration was effected by the activation of an enzyme within the conoid structure is not known with certainty. It appears probable that such activation, if there be any, would be a secondary event, which occurs after the recognition of the susceptible host cell membrane by the parasite.

Some vertebrates are naturally resistant to some particular parasites. The resistance under such circumstances may be

complete or partial. Naturally occurring resistance may occur if there is present in the serum of the resistant host, some antibodies to the resistant species of the parasite. The parasite under such circumstances may be agglutinated, precipitated, or opsonized. In any of these circumstances, the parasite would be destroyed.

Many of the studies conducted thus far are far from being conclusive. We still do not know the cause of the susceptibility or resistance of a particular species of host to a particular species of malaria parasite. Theories abound but no good experimental evidence exists to support any theory. We have, in the present study, conducted some experiments on the problem of host parasite relationships in a bird malaria. We still have not been able to pin point the cause of the specificity of a particular host to a particular parasite. Most of our results have been noncontributory with some exceptions.

We have conducted the following experiments in this line.

i) Study of the composition of serum and plasma of susceptible and nonsusceptible bird in respect to plasma protein, sodium and potassium.

ii) Characterization of hemoglobin of susceptible and nonsusceptible birds.

iii) Estimation of gamma globulin of sera of susceptible and nonsusceptible birds.

iv) Preparation of antigens from this parasite and to study the presence of a naturally occurring antibody in the non-susceptible bird against this parasite.

v) Detection of complementarity of the cell membrane of the parasite with the cell membrane of the erythrocyte of the susceptible host.