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# A Model For The Ecology Of Avian Malaria*

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**Abstract**

Although studies on *Plasmodium* infections of wild birds have been reported frequently in the literature, our knowledge of the ecology of these parasites remains incomplete. A synthesis of data and ideas from these field studies, and recent experimental work led to the construction of the following hypothetical model for the ecology of avian malaria:

During the late spring, summer, and early fall susceptible birds (young of the year or previously uninfected adults) contract the infection from the bite of an infected mosquito on the breeding ground. The birds migrate or remain in the area and infections become latent over the winter. In the spring, migratory birds return to the breeding area and all birds commence reproductive activity. With the onset of migration and breeding activity, parasite populations become elevated in the birds. This relapse of malarial infections coincides with emergence of vectors. The mosquitoes obtain the parasite, passing it on to susceptibles in the population (whose numbers are simultaneously increasing as the result of reproduction), and the cycle continues. Under favorable conditions, transmission rates equal or exceed a level needed to replace mortality of infected birds. Under unfavorable conditions the parasite is maintained by the bird reservoir, the population of susceptibles increases, and transmission is postponed until favorable conditions return and transmission to the expanded population of susceptibles replenishes the supply of infected adults. Such a cycle, in which the parasite, vector,

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and susceptible host populations reach a maximum in an apparently favorable sequence, with provision for occasional failure of transmission, has obvious survival value.

A more complete understanding of the ecology of avian malaria will be achieved with the investigation of specific problem areas defined in this model. The model may prove of additional value in suggesting an ecological approach to our understanding of the epidemiology of human malarias. It may also have applicability in other disease systems where bird-mosquito relationships are similar, such as certain of the arboviruses.

## 1. Background

Although studies on *Plasmodium* infections of wild birds have been reported frequently in the literature, our knowledge of the ecology of these parasites remains incomplete. Many of the studies dealing with natural malarial infections in wild birds have been surveys and have contributed much to our knowledge of the distribution and incidence of bird malaria. Several authors have considered the ecological implications of their data and have substantially contributed to our understanding of the subject. A synthesis of the data and ideas resulting from these field studies would be useful since it would define certain parameters of the problem and hopefully add to our understanding of the ecology of malaria in wild bird populations. Our discussion deals specifically with the genus *Plasmodium*, and the term "malaria" in the text refers to this genus of parasites.

The objective of this paper, therefore, is to construct a biological model which, although speculative, would outline the present state of our understanding of the ecology of avian malaria and point out major gaps in our knowledge which are yet to be filled. In addition to the existing literature, many of the data used in constructing this model were obtained in an intensive study of a breeding songbird population in a central Pennsylvania woodlot during 1963-1967. Although the model presented here will be specifically applicable to the ecology of *Plasmodium* in summer resident bird populations, a more general application to the ecology of avian malaria in North American songbirds can be inferred. The model may prove of additional value in suggesting an ecological approach to our understanding of the epidemiology of human malarias. It may also have applicability in other disease systems where bird-mosquito relationships are similar, such as certain of the arboviruses.

## 2. Definition of the Ecology of Avian Malaria

Ecology as broadly defined by Allee *et al.*[1] is the interrelation between living organisms and their environment. In the context of this definition the environment of the malarial organism, an obligate parasite, becomes the tissues and organs of the host in which it resides, which for avian malaria in its simplest form consists of birds and mosquitoes. Survival of a malaria population requires frequent transfer

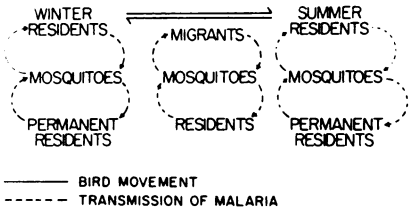


FIGURE 1. *Theoretical potential environment of bird malaria.*

of the parasite from one host to the other, and the biological success of the malarial parasite therefore depends not only on its ability to survive and reproduce within the individual host but equally on its ability to reach new hosts. An understanding of the ecology of avian malaria therefore includes an understanding of the interrelationships between vertebrate, insect and protozoan populations.

If the parameters of time and space are considered in avian malaria transmission, the potential environment of the malaria parasite is quite complex (Fig. 1). Transmission at all possible locations in this potential environment may occur, but all are probably not required for survival of the parasite. However, certain of these pathways must be absolutely essential as the minimal requirements for perpetuation of the parasite species. The ecology of avian malaria may best be understood by identifying the pathways wherein the minimal requirements for survival are met, and the mechanisms by which they are guaranteed.

### 3. Formulation of Model

A review of the basic facts surrounding natural infections of *Plasmodium* in birds is at this point useful. Data from surveys in which monthly samples were obtained indicate that a peak in prevalence occurs in the spring of the year[3,13,15,18,19] (Fig. 2).

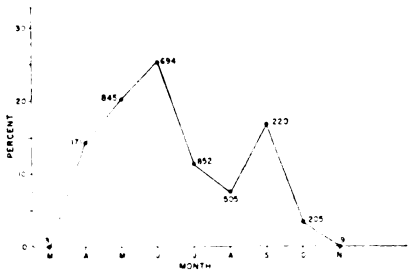


FIGURE 2. *Composite of the monthly prevalence of* Plasmodium *in songbirds captured in a Pennsylvania woodlot, 1963-67, based on a 10-minute examination of blood films. Prevalence is expressed as percent infected per month. Numbers indicate sample size.*

Furthermore, when the prevalence studies are accompanied by monitoring of the potential vectors, it becomes quickly evident that the time of emergence of mosquito vectors in the sampling area is later than the observed peak.[13] For example, the earliest emergence of mosquitoes in our Pennsylvania study area was observed on May 2. For local transmission of parasites by these mosquitoes, the following events must take place: (1) infection of mosquitoes by feeding on an infective bird, (2) sporogony in the mosquito, (3) a second blood meal in infecting a susceptible bird, and (4) development of the infection to the extent that parasites can be detected in a blood film. These events would require at least several weeks under optimum conditions and would be retarded further by the normally cool temperatures of spring,[21,23] so that the earliest demonstrable primary infections could not be expected until June. We may assume, then, that malarial infections in April and May are not the result of transmission by local vectors. Since nonmigratory birds demonstrate the same spring peak,[3] it appears unnecessary to postulate that spring peak of prevalence in migratory species results from transmission in the wintering area or along the migratory route. These observations have led several investigators to postulate the existence of a spring relapse phenomenon in *Plasmodium*.[3,13,15]

Applegate[2] has shown in an experimentally infected wild population of house sparrows that patent infections occurring in the spring of the year with *Plasmodium* are the result of relapse. This observation confirms that of Box[3] who showed that highest prevalence of infections occurred in the springtime in naturally infected captive house sparrows. Applegate's data indicate that a high proportion of infected birds relapse in the spring, and he has termed this phenomenon a "synchronous population relapse."

Our data (Fig. 2) indicate that a second peak in prevalence also occurs in the late summer and fall. Analysis by host age indicates that these infections are primary attacks resulting from local transmission, since the second peak includes predominantly birds in juvenile plumage. While some movement of young in the breeding area does occur, these birds have certainly obtained the infections locally, if not on the study area itself.

In most field surveys where infections by age class were reported, malaria infections in nestlings and juveniles have likewise demonstrated transmission in the breeding area.[3,9,10,18]

Transmission to juvenile birds in the temperate zone indicates that the Mosquito → Summer Residents' and Mosquito → Permanent Residents' portion of the theoretical cycle in Fig. 1 exist in the field. Since overwintering of sufficient numbers of mosquitoes appears unlikely,[21] the mosquitoes must obtain their parasites from infected adult birds before transmission commences, so the Summer Residents → Mosquito and/or the Permanent Residents → Mosquito pathways must also exist in the field. A spring relapse phenomenon which elevates parasite population in adult birds would appear to increase the probability that mosquitoes become infected when feeding on such infected birds.

Data are thus available which consistently demonstrate the existence of a Mosquito→ bird → mosquito cycle in the temperate zone breeding area. Few data are available concerning transmission along the migratory routes or in the wintering grounds. Information from the Pennsylvania study (Table 1) and from others[16,17] indicate

TABLE 1. *A comparison of* Plasmodium *prevalence in the five most common migratory and nonmigratory species of songbirds captured in a Pennsylvania woodlot, 1963-65, based on a 10-minute examination of blood films.*

| | No. observed | No. positive | % positive |
|---|---|---|---|
| *Migratory species:* | | | |
| Robin | 168 | 74 | 44.0 |
| Wood Thrush | 223 | 35 | 15.7 |
| Catbird | 176 | 13 | 7.4 |
| Scarlet Tanager | 60 | 5 | 8.3 |
| Ovenbird | 121 | 7 | 5.8 |
| | 748 | 134 | 17.9 |
| *Nonmigratory species:* | | | |
| Cardinal | 30 | 5 | 16.6 |
| Blue Jay | 36 | 1 | 2.8 |
| Black-capped Chicadee | 72 | 0 | 0 |
| Tufted Titmouse | 73 | 3 | 4.1 |
| Downy Woodpecker | 49 | 0 | 0 |
| | 260 | 9 | 3.5 |

that migratory birds are more commonly infected than non-migratory birds, but the reasons for this are not clear. The importance of the migratory habit of hosts in maintaining the parasite species requires more study.

On the other hand, the data obtained in our study suggest that the maintenance of malaria can be accounted for by spring relapse of infected birds and summer and fall transmission of infections to susceptibles, all of which occur in the summer breeding area. Note that the available data do not rule out occasional transmission in the wintering grounds or along the migratory route. These data show that it is unnecessary to postulate transmission other than on the breeding ground in order to fulfill the minimum requirements for parasite survival as stated earlier.

TABLE 2. *Prevalence of* Plasmodium *in all birds with juvenile plumage captured in a Pennsylvania woodlot, 1963-67, based on a 10-minute examination of blood films.*

| Month | No. captured | % with Plasmodium |
|---|---|---|
| June* | 38 | 18.4 |
| July | 262 | 15.3 |
| August | 211 | 10.0 |
| September | 66 | 15.2 |
| October | 42 | 4.8 |

* Includes 4 birds captured during the last week in May.

Ideas similar to those discussed in this model have been mentioned in relation to other haematozoa. O'Roke[20] observed a natural relapse for *Leucocytozoon* in ducks and suggested that this was the method of infection for local vector populations. Huff[12] later confirmed the observation of O'Roke for *Leucocytozoon* and extended it to include *Haemoproteus*. Chernin[4] also reported spring relapse in *Leucocytozoon* in ducks and related it to their reproductive activity.

Although the above concept appears warranted, alternative hypotheses are possible, though less attractive. We have stated that survival of the malaria parasite requires transmission from bird to vector to bird. If transmission were continuous, there would be no problem in explaining the survival of the parasite species. However, movement of birds and fluctuation of vector populations suggest that transmission is not continuous. Since a short lapse in transmission under this scheme would result in extinction of the parasite, a more certain mechanism of survival would appear necessary.

The alternative to continuous transmission is the existence of a biological reservoir; in this case either the mosquito vector or the bird. A biological reservoir must consistently provide for survival of the parasite during conditions unfavorable to transmission. It necessarily follows that stability of a host population is essential to establishment of such a reservoir relationship. Thus, the mosquito vector would be an unlikely reservoir because its populations are subject to rapid and unpredictable fluctuations between, as well as within years.[21] Furthermore, the mosquito is short-lived,[23] giving rise to an unstable population structure with rapid turnover of individuals. On the other hand, birds possess attributes which clearly fulfill the above requirements. Population levels of many species of songbirds remain relatively stable from year to year and changes are generally only gradual and long term.[7,8] The turnover rate in bird populations is much lower than that for mosquitoes since individuals commonly live for several years.[6,7] In addition the composition of local populations is stable since migratory birds frequently return to the same breeding area.[11] Finally, the relationship between parasite and host is persistent after it is initially established.[14] The bird, then, would appear to provide a better reservoir population in which the parasite could survive over periods of non-transmission. The demonstration that mechanisms exist which elevate parasite populations within vertebrate hosts in synchrony with the onset of seasonal increase of mosquitoes further enhances their value as a reservoir.

If the basis for survival of avian malaria is maintenance by the bird reservoir with predictable spring relapse, and transmission is a more uncertain event, then we would expect greater annual variation in the fall prevalence than in the spring prevalence. Our data indicate the fall prevalence, which represents primary infections and reflects yearly transmission rates, fluctuates more from year to year (0-25%) than does the spring peak (15-25%), which is due to relapse (Fig. 3). Reeves *et al.*[22] have shown marked differences in yearly infection rates in vectors from the same area. It should not be surprising, therefore, that a small sample of juveniles in a single year would fail to show infection.[16] In years when transmission was inadequate or failed completely, longevity of infected adults with recurring spring relapse would provide for survival of the parasite.

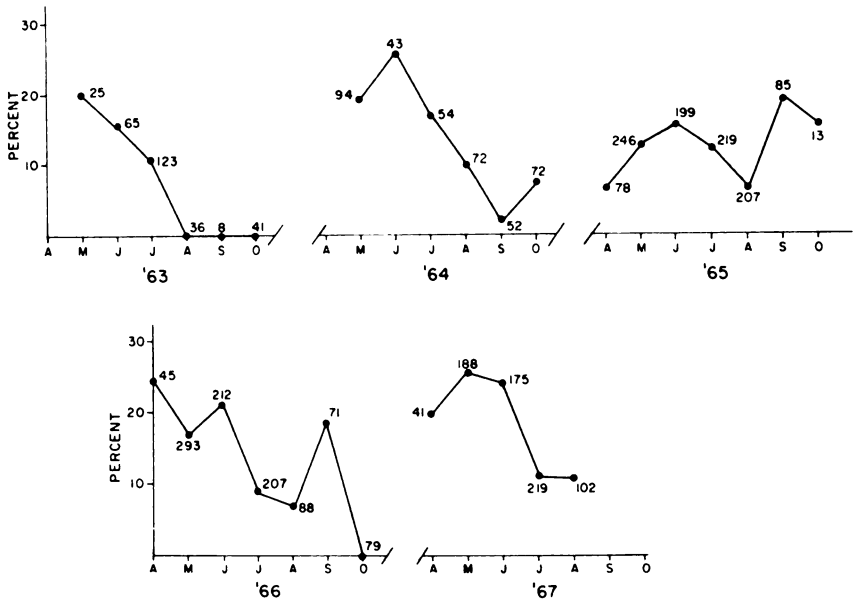


FIGURE 3. *Prevalence of* Plasmodium *by month in birds collected in a Pennsylvania woodlot, 1963-67. Prevalence is expressed as percent infected per month, based on a 10-minute examination of blood films. Numbers indicate sample size.*

From the facts discussed above, it is possible to postulate the following hypothetical model of the ecology of avian malaria (Fig. 4).

During the late spring, summer, and early fall susceptible birds (young of the year or previously uninfected adults) contract the infection from the bite of an infected mosquito on the breeding ground. The birds migrate or remain in the area and infections become latent over the winter. In the spring, migratory birds return to the breeding area and all birds commence reproductive activity. With the onset of migration and breeding activity, parasite populations become elevated in the birds. This relapse of malarial infections coincides with emergence of vectors. Mosquitoes obtain the parasite, passing it on to susceptibles in the population (whose numbers are simultaneously increasing as the result of reproduction), and the cycle continues. Under favorable conditions, transmission rates equal or exceed a level needed to

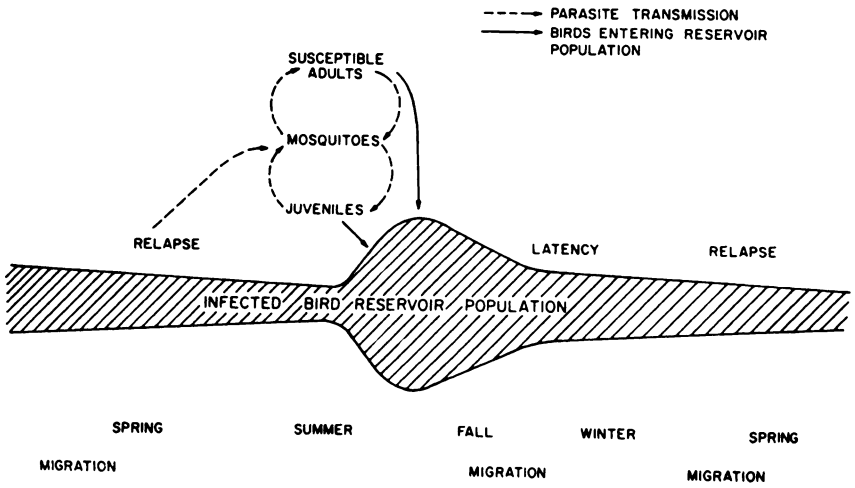


FIGURE 4. *Essential elements for survival of avian malaria. Hatched area represents relative size of reservoir population.*

replace mortality of infected birds. Under unfavorable conditions the parasite is maintained by the reservoir, the population of susceptibles increases, and transmission is postponed until favorable conditions return and transmission to the expanded population of susceptibles replenishes the supply of infected adults. Such a cycle, in which the parasite, vector, and susceptible host populations reach a maximum in an apparently favorable sequence, with provision for occasional failure of transmission, has obvious survival value.

### 4. Questions Raised by the Model

A functional model collects the available facts into a concise theory. More important, the model points out areas where more information is needed. Several such areas where additional information would add to our understanding of the ecology of avian malaria are outlined below.

The model assumes that spring relapse is accompanied by increased infectivity to mosquitoes. Likewise, it assumes that a bird whose infection is "latent" by microscopic examination is less able to infect mosquitoes than one with demonstrable parasites. Additional work with laboratory transmission should be performed to clarify these points.

Migratory birds had a higher infection rate than nonmigratory birds in the Pennsylvania study (Table 1) and in other studies.[17,18] The reasons for this may lie in host-specificity, behavioral differences in birds, or a significant amount of transmission occurring off the breeding area. Solution of this problem would strengthen the model or favor alternate hypotheses.

An intensive study of an infected nonmigratory population is needed, including monthly prevalence data established by a subinoculation test. A study population which was somehow isolated from migrants would be ideal. The hypothesis that malaria can be maintained without annual introduction by migrants should be tested.

The mechanism of spring relapse should be studied further. It is unclear whether this phenomenon results from an environmental stimulus mediated by the physiology of the host, as suggested by Chernin[4] for *Leucocytozoon*, or by an innate rhythm in the parasite, as appears to be the case in vivax malaria.[5]

It is obvious therefore that our understanding of the ecology of avian malaria is incomplete. A more complete understanding should be achieved with the investigation of specific problem areas defined by this model.

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