BIRD BLOOD PARASITES – A NEW DIMENSION TO BIRD RINGING

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In the Annual report on the 1991-1992 ringing year (Oatley 1992) there was some speculation as to the reasons for the discrepancy in the survival rate between Olive Thrushes *Turdus olivaceus* and Cape Robins *Cossypha caffra*. From this it was clear that Cape Robins probably have a greater chance of surviving for longer. From a blood parasitological point of view this is very logical. Although the Cape Robin and Olive Thrush very often occur together and are subject to the same probability of playing host to the same species of blood parasites, the Cape Robin is seldom parasitized. Of the 48 Cape Robins checked for blood parasites to date, only 4 (8%) were parasitized, while 28 of the 96 (29%) Olive Thrushes checked harboured blood parasites (Bennett *et al.* 1992). From my own ringing, I have never found a Cape Robin, Whitethroated Robin *Cossypha humeralis* or Scrub Robin *Erythropygia leucophrys* to contain any blood parasites while 69% of all the Olive Thrushes that I handle, both in my garden and in *Acacia* veld, are hosts to blood parasites.

The presence of a *Hepatozoon* parasite in the blood of an individual can be an indication of the diet of the bird. To pick up a *Hepatozoon* infection the bird has to ingest a tick which harbours the *Hepatozoon*. The ingestion of infected ticks can be accidental as is probably the case with the high infection rate of South African Cliff Swallows *Hirundo spilodera* with *Hepatozoon atticorae* where 49% (70/143) were infected. These ticks are probably ingested while preening in the nest as they only feed on the swallows at night. Other species such as the titbabblers *Parisoma* spp. and bush-creeping shrikes, e.g. Crimsonbreasted Shrike *Laniarius atrococcineus*, Southern Boubou *L. ferruginea*, Puffback *Dryscopus cubla* and Threestreaked Tchagra *Tchagra australis*, actively feed on ticks which hide under the bark of trees. All of these shrikes and the titbabblers are highly infected with *Hepatozoon* because of this foraging behaviour. The effect of the parasites on these populations is unknown, purely because of the lack of bloodsmear material from these species. To get back to the Cliff Swallow *Hepatozoon*: while 49% of the birds are infected, 60% of the ticks are infected (Bennett, Earlé, Penzhorn 1992) and there is a yearly mortality rate of nearly 60% amongst the swallows. Is the infection rate of the swallows an indication of the survival of the swallows?

The first arrivals at a Cliff Swallow colony where blood smears were taken on a weekly basis were all parasite free. The first birds infected with *Hepatozoon atticorae* were noted six weeks after the first arrivals and two weeks later 21% of all the swallows trapped were infected. This percentage rose to between 40-50% throughout the season and nearly all of the late departers were infected (88%). It seems as if this parasite had some effect which delayed the departure of some infected birds and probably lessened their chances of surviving their long-distance seasonal migration.

Valkjunas (1989) has firmly established that the timing of migration of individual Chaffinches *Fringella coelebs* in eastern Europe is explained by the severity of their blood parasite infection. "Concentration of infected birds at the end of the migration flow can be regarded as the parasitic filter delaying the birds, which have been seriously ill, from flying away to wintering places at the most favourable periods". Valkjunas (1989) also showed a casual relationship (casual only because of low numbers of recovered birds in the data set) between the heavy spring migration mortality and the intensity of infection of individual birds.

The same might apply to southern African migratory birds, e.g. while some European Swallows *Hirundo rustica* are still in our skies in April, other are already incubating eggs in their northern nests. Is this because these late stragglers are not in peak condition because of their parasitic load?

It has been speculated that the late moult of Knots *Calidris canutus* could be the result of an endoparasite infestation (Underhill *et al.* 1993). The blood parasite load of individual birds might explain, together with other factors, why one always finds some individuals in a population that are way off-line in their timing and speed of moult.

Even the absence of blood parasites in the birds can be of great significance (Earlé & Underhill 1993; Bennett, Montgomerie & Seutin 1992). It has long been debated whether sandgrouse are pigeons with feathered legs or whether they are more closely related to waders. The parasite information indicates that while pigeons are highly infected with various blood parasites both sandgrouse and waders have virtually none (Bennett *et al.* 1992). Earlé & Underhill (1993) have argued that a possible explanation for the lack of blood parasites in the waders is that they probably evolved in an environment free of the vectors – is this also the case regarding the sandgrouse?

The blood parasites of birds can also be used as markers of the movement of bird populations. The vectors of all *Leucocytozoon* species parasitizing birds are blackflies (Simulidae). These flies breed only in running water such as rivers and the *Leucocytozoon* that they transmit would thus occur in bird populations occurring in habitats where streams flow. *Leucocytozoon*, for instance, is the most common blood parasite in mountainous areas such as those in the Lydenburg district where up to 39,7% of Gurney's Sugarbirds *Promerops gurneyi* were infected with *Leucocytozoon*. The occurrence of a *Leucocytozoon* infection in a bird in the Kalahari would indicate that the bird had moved from somewhere else where it was infected, as there is no good habitat for blackflies in the form of running water in the Kalahari. At a ringing site at Buffelsdrift, just east of Pretoria, with an irrigation canal with continuous running water, almost all species and most individuals of these species are infected with *Leucocytozoon* while about 4 km away in the same habitat, the same bird species are seldom infected with *Leucocytozoon*. This is well illustrated by House Sparrows *Passer domesticus* and Cape Sparrows *Passer melanurus* that show a 64% infection rate with *Leucocytozoon* while at the second site not a single sparrow with a *Leucocytozoon* parasite infection has ever been encountered.

Valkjunas & Iezhova (1990) also found that in eastern Europe sparrowhawks

Accipiter spp. are increasingly intensely infected with *Leucocytozoon* from moderate latitudes towards the tropics while the intensity of *Haemoproteus* infections increase from the northwest to the southeast of the hawk's distribution. Bearing in mind that raptors in the northern hemisphere are heavily infected by blood parasites, the occurrence of a certain parasite of a bird can be some indication of the origin of the bird.

In Bloemfontein, large numbers of Cape White-eyes *Zosterops pallidus* and sunbirds *Nectarinia* spp. moved through my garden during the winter months. The high infection rate even during the severe drought years when vectors were few and the possibility of transmission fairly low, indicated that these birds were travelling some distance to spend the winter in Bloemfontein. This was confirmed when a Malachite Sunbird *Nectarinia famosa* ringed in my garden was recovered in Ficksburg.

Probably the greatest importance of having knowledge of the blood parasitological state of the individual is how these parasites might influence the survival of the individual. From earlier work it is clear that individual birds probably retain a blood parasite infection of *Haemoproteus* throughout their lives. However, it also seems as if at least some individuals stay parasite-free throughout their lives (Bennett, Caines & Bishop 1988; Bennett & Bishop 1990). Although the data is a bit thin at present as ringing for blood parasites is less than three years old, the fact that the parasite-free individuals are more often recaptured than the parasite carriers, is becoming a bit too much for pure chance.

When a bird is infected by the blood parasite the parasite first undergoes a developmental stage in the tissues of the bird before being released to invade the blood cells. This tissue stage of multiplication of *Haemoproteus* takes place in all organs but especially in the large muscles such as the breast muscles and heart muscle. On release of the parasites from the cysts, a toxin is also released into the tissue causing necrosis of the surrounding tissue. Thus each time a cyst bursts a number of muscle fibres are destroyed. Although this does not happen often it is known that a heavy infection of *Haemoproteus* in doves can be so damaging to their flight muscles that they cannot fly and may even die (Earlé *et al.* 1993a). The blood parasites probably do not cause the death of their host directly but they might be a major factor in putting the bird under stress and thus causing it to succumb to other infections or to predation. A Grey Lourie *Corythaixoides concolor* brought into my office a few months ago, unable to fly, was discovered to be suffering from a bacterial lung infection with a heavy infection of *Leucocytozoon*. It recovered after a few days of antibiotic treatment and was released. This kind of treatment is not available to birds in the wild and this bird would surely have fallen prey to some predator. Would the bird have been able to fight the bacterial infection successfully if its immune system was not so occupied with the blood parasite infection?

It was only discovered recently that the massive seasonal deaths of Pied Currawong *Strepera graculina* in Sydney, Australia, were the direct result of acute infection with *Haemoproteus* (Hartley 1992).

Recently a new species of *Babesia*, a parasite more common to mammals, was described from the African Penguin (Earlé *et al.* 1993). These parasites are very pathogenic in mammals and there is no reason to believe that the same

does not apply to penguins. Penguins are highly susceptible to *Plasmodium* infections (transmitted by mosquitoes) when on the mainland. These *Plasmodiums* are the prime reason for the lack of success in keeping penguins in zoos. Is this a reason why penguins breed on offshore islands where the *Plasmodium*-infected mosquitoes cannot get at them?

Numerous new bird blood parasites have been described from various South African birds over the last two years (e.g. Bennett & Earlé 1992; Bennett *et al.* 1992). Some bird ringers have been named for ever as they collected the smears from which the new species have been described. Dawie de Swardt has *Leucocytozoon deswardti* (which occurs in the Gurney's Sugarbird) named after him (Bennett, Earlé & Peirce 1992) after collecting a very good sample of these parasites. Walter Neser was also named by having *Haemoproteus neseri* from the Chorister robin named after him in recognition for his constant submission of bird blood smears (Bennett & Earlé 1992).

When will the day come when John will boast: "You remember that Malachite Kingfisher that I caught last month? Well, it has *Haemoproteus enucleator*, the first record for southern Africa." To that Pete would reply: "The Red Bishop smears that I took every month show a nice seasonal pattern of infection with *Haemoproteus quelea* which alternates with *Leucocytozoon bouffardi*" and Tom would add: "All the Willow Warblers that I smeared early in the season were parasite-free but the ones moulting late and the late departures were virtually all carrying *Haemoproteus sulvae* and some even *Trypanosoma avium*" ...

On the basis of current bird ringing activity in South Africa, collection by the majority of ringers of blood smears from birds handled by them could quickly answer the following questions:

1. What is the difference in the blood parasites between sunbirds and/or sugarbirds in widely different habitats in the southwestern Cape, eastern and central OFS, Transvaal and in Zimbabwe? In all of these regions ringing studies are already well established and bloodsmears from the Lydenburg area are unusual in that the sunbirds almost exclusively harbour *Leucocytozoon nectariniae* while the generally more common *Haemoproteus sequeirae* occurs rarely. What infestations are carried by the large numbers of sunbirds ringed in the southwestern Cape?

2. Does the infection rate by blood parasites have any effect on the three species of *Pycnonotus* bulbuls at various localities in South Africa? All three of these bulbuls have featured amongst the top 20 most-ringed birds for several years running and are ringed in just about every habitat in South Africa. This question could be answered within a season or two if all ringers made smears.

3. Are blood-parasite-infected birds retrapped as often as birds that are not infected? Is this an indication of the effect of the blood parasite infection? Do infected birds retain their infection at subsequent recaptures? Is the infection thus an indication of the survival possibilities? Commonly ringed and recaptured birds such as Masked Weavers, Cape White-eyes, Cape Sparrows or Laughing Doves can be the subject of such investigations as can be a whole community.

Collection of good smears takes a little practise, but once learnt can be quickly and efficiently accomplished. As indicated above, the results can provide insight into a range of interesting aspects of dispersal and survival. There is, in fact, a whole lot to be learnt from even our commonest birds so what better way to provide additional purpose to your ringing activities?

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