Hematozoa in Endemic Subspecies of Common Kestrel in the Cape Verde Islands

Authors: Hille, Sabine Marlene, Nash, Jon Patrick, and Krone, Oliver

Source: Journal of Wildlife Diseases, 43(4) : 752-757

Published By: Wildlife Disease Association

URL: https://doi.org/10.7589/0090-3558-43.4.752

Journal of Wildlife Diseases, 43(4), 2007, pp. 752–757

Hematozoa in Endemic Subspecies of Common Kestrel in the Cape Verde Islands

Sabine Marlene Hille,[1,4] Jon Patrick Nash,[2] and Oliver Krone[3] [1]Institute of Wildlife Biology and Game Management, University of Applied Sciences, Gregor-Mendel-Strasse 33, Vienna, Austria; [2]EBT Laboratory, Department of Biology, University of Antwerp, 2020, Belgium; [3]Leibniz-Institute for Zoo and Wildlife Research, PO 601103, D-10252 Berlin, Germany; [4]Corresponding author (email: sabine.hille@boku.ac.at)

ABSTRACT: We examined 130 Common Kestrel (*Falco tinnunculus*) representing two endemic subspecies and nine resident island populations on the Cape Verde archipelago between 1996 and 1999 to study diversity, prevalence, and intensity of hematozoa. Hematozoan diversity was very low; we detected only *Plasmodium fallax*, a species that is rarely found in Falconoformes, and, possibly, *Haemoproteus brachiatus.* Moreover, prevalence of *Plasmodium fallax* was low (1.5%) with a mean intensity of infection of 0.05 protozoa/10^{-3} erythrocytes. Only one bird (0.8%) was infected with a gametocyte that was most likely *Haemoproteus brachiatus*; the intensity in this infected bird was 1.5 protozoa/10^{-3} erythrocytes. A single parasite or two parasites were observed in blood smears in four additional birds, but identification to genus was not possible. This is the first record of blood parasites in birds on the Cape Verde Archipelago. The low prevalence of these parasites might be because of arid and less-favorable conditions for the pathogen's vectors. The sedentary nature and high level of isolation of the island kestrel populations are also factors that could decrease the probability of infection.

Key words: *Falco tinnunculus*, *Haemoproteus*, hematozoa, island populations, *Plasmodium*, raptor.

The prevalence of avian blood parasites is expected to be lower in isolated populations when compared to resident mainland or migratory populations (Dogiel, 1949; Steadman et al., 1990; Valkiunas and Iezhova, 1993; Jarvi et al., 2003). In contrast, hematozoa introduced into island ecosystems can have severe negative impacts on forest birds. Such impacts have been documented as a result of accidental introductions of blood-feeding arthropods and avian malaria to resident and isolated bird populations on the Hawaiian Islands (Warner, 1968; Atkinson et al., 1995; Atkinson et al., 2000) and on Samoa (Atkinson et al., 2006). In the tropics, humid warm climates facilitate development and survivorship of potential vectors year-round, which can in turn affect transmission rates (Gabaldon and Ulloa, 1980; Atkinson, 1988). Prevalence and intensity of infection in birds with blood parasites also depend on life history traits and behavior of birds, such as nestling time, body size, nest type, locomotion during nesting, and colonial and migratory behavior (Valkiunas, 2005). Migratory birds have a higher risk of infections with hemoparasites (Markov, 1939; Dogiel, 1949; Valkiunas and Iezhova, 1993), whereas endemic island birds are less commonly exposed to such parasites (Steadman et al., 1990; but see Atkinson et al., 2006). Potential roles of isolation, climate, and human impact on the prevalence of hemoparasites in bird populations in the tropics are not currently understood.

Two allopatric and endemic subspecies of the Common Kestrel (*Falco tinnunculus neglectus* and *Falco tinnunculus alexandri*; Bourne, 1955) occur on the subtropical and arid sub-Saharan Cape Verde archipelago (Fig. 1; between 14°48′N to 17°22′N and 22°44′W to 25°22′W). These island populations manifest site fidelity, and are isolated and differ in size, with the highest populations occurring on Santo Antão and Santiago (Hille et al., 2003). Following the early work by Wasielewski and Wülker (1918) on the life cycle of *Haemoproteus tinnunculi*, several studies on blood parasites in wild Common (Eurasian) Kestrels (*Falco tinnunculus tinnunculus*) reported infection with *Leucocytozoon*, *Trypanosoma*, *Filaria*, and

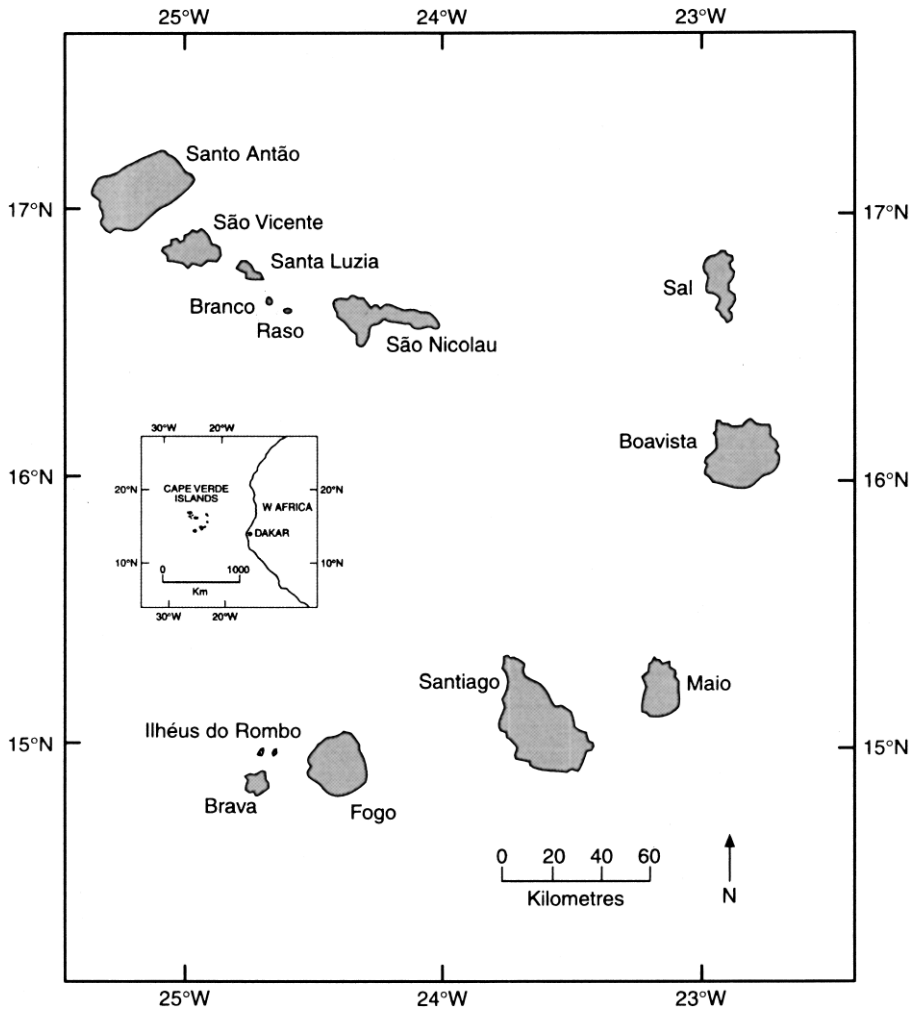

FIGURE 1. Map of the Cape Verde Islands.

Haemoproteus brachiatus (Peirce et al., 1990). The prevalence of *Plasmodium* spp. is generally very low in birds of prey; Kučera (1981) reported a prevalence of 2.3% in Strigiformes ($n=178$), but did not detect the parasite in Falconiformes (see Krone et al., 2001; Wiehn and Korpimäki, 1998). The primary vectors of *Plasmodium* are mosquitoes of the genera *Aedes*, *Culex*, and *Anopheles* (Valkiunas, 2005): The primary vectors for *Haemoproteus* species are blood-feeding arthropods such as simuliid and hippoboscid flies, ceratopogonid midges (genus *Culicoides*; Desser and Bennett, 1993); the latter complete part of their life cycle in free water or moist environments (Mellor et al., 2000). *Haemoproteus* spp. has been reported in kestrels sampled throughout Europe and Africa (Peirce et al., 1983) and the prevalence of *Haemoproteus* in wild bird populations may depend on suitability of breeding habitat for *Culicoides*, which will vary according to rainfall and availability of appropriate freshwater larval habitats (Earlé et al., 1991). In kestrels in Finland, the prevalence of *H.* and *H. brachiatus* was 40% and 13%, respectively (Korpimäki et al., 1995), whereas lower infection rates of *H. tinnunculi* and

Leucozytozoon toddi have been reported for several African species of Falconidae that were sampled in arid regions (Haiba, 1949; Peirce and Cooper, 1977; Bennett et al., 1992). The goals of this study were to identify and determine blood parasite prevalence and intensity in nine nonmigratory and isolated Common Kestrel populations endemic to the Cape Verde Archipelago.

The study includes data from four field seasons completed during April and May 1996–99. A total of 130 adult kestrels (69 males and 61 females) were sampled at randomly selected breeding locations for nine island populations. *Falco tinnunculus alexandri* were sampled on Sal ($n=7$), Boavista ($n=28$), Maio ($n=6$), Santiago ($n=15$), Fogo ($n=13$), and Brava ($n=27$), and *Falco tinnunculus neglectus* were sampled on São Nicolau ($n=1$), São Vicente ($n=2$), and Santo Antão ($n=31$) using bal-chatri traps (Bub, 1986). We could not exclude all possible sampling artifacts such as potential seasonal stress and immune suppression associated with the rearing season, but based on the work of Valkiunas (2005) we felt that this represented the best period to detect blood parasites using traditional microscopic examination of blood smears.

Smears were prepared from blood obtained by brachial venipuncture. Blood was taken from adult birds during the breeding period when nestlings were 3–4 wk old in each population to control for phenological differences between populations (see Weatherhead and Bennett, 1992). Smears were air-dried, fixed for 5 min in pure ethanol, stained with Giemsa solution, and scanned at 100×, 400×, and under oil immersion (1,000×). The scanning time for each blood smear was 10 min. During this period, they were scanned at 25× for 0.5 min, 100× for 1.5 min, 200× for 2 min, and 400× for 6 min. The oil immersion (1,000×) was only used when the blood smear contained parasites. Measurements were performed with a Zeiss Axioplan microscope (Oberkochen, Germany). Intensities of parasites were calculated by counting parasites in fields of 300–400 erythrocytes in 10 fields.

We determined parasite prevalence (proportion of individuals infected in the population) and parasite intensity (mean number of respective protozoa per 10,000 erythrocytes) across all individuals sampled per population.

From 130 individuals sampled from nine island populations, only six individuals, (4.6%) were infected with blood parasites; the identification of parasites was only possible for three of these birds. Two (1.5%) kestrels were infected with *Plasmoodium fallax* one kestrel (0.8%) was infected with *H. brachiatus.* These birds occurred on Santo Antão and Santiago, the islands with the two largest kestrel populations in the archipelago (Hille et al., 2003). We detected *P. fallax* in two individuals on Santo Antão (6.5% of the birds sampled on the island; Fig. 2); an intensity of 0.04 organisms/10^{-3} erythrocytes was observed in one kestrel and 0.06 organisms/10^{-3} erythrocytes in a second kestrel. Only one falcon was infected with *H. brachiatus* (intensity of 1.5 organisms/10^{-3} erythrocytes). This kestrel was caught on Santiago (6.7% of birds sampled on the island). Higher prevalence and intensity estimates might be obtained by polymerase chain reaction diagnostics or serological methods (Jarvi et al., 2002; but see Jarvi et al., 2003).

Assuming that the identified *Plasmodium* and *Haemoproteus* and their vectors are similar to those on the mainland, diversity, prevalence, and intensity of blood parasites in the island populations were lower than reported for kestrels on the European mainland (Korpimäki et al., 1995). One reason for low prevalence and intensity of hematozoa on Cape Verde could be the geographic isolation of the resident island populations, which rarely come in contact with kestrels from other populations (Hille et al., 2003). According to Manwell (1935), high mobility and migration patterns of the host species

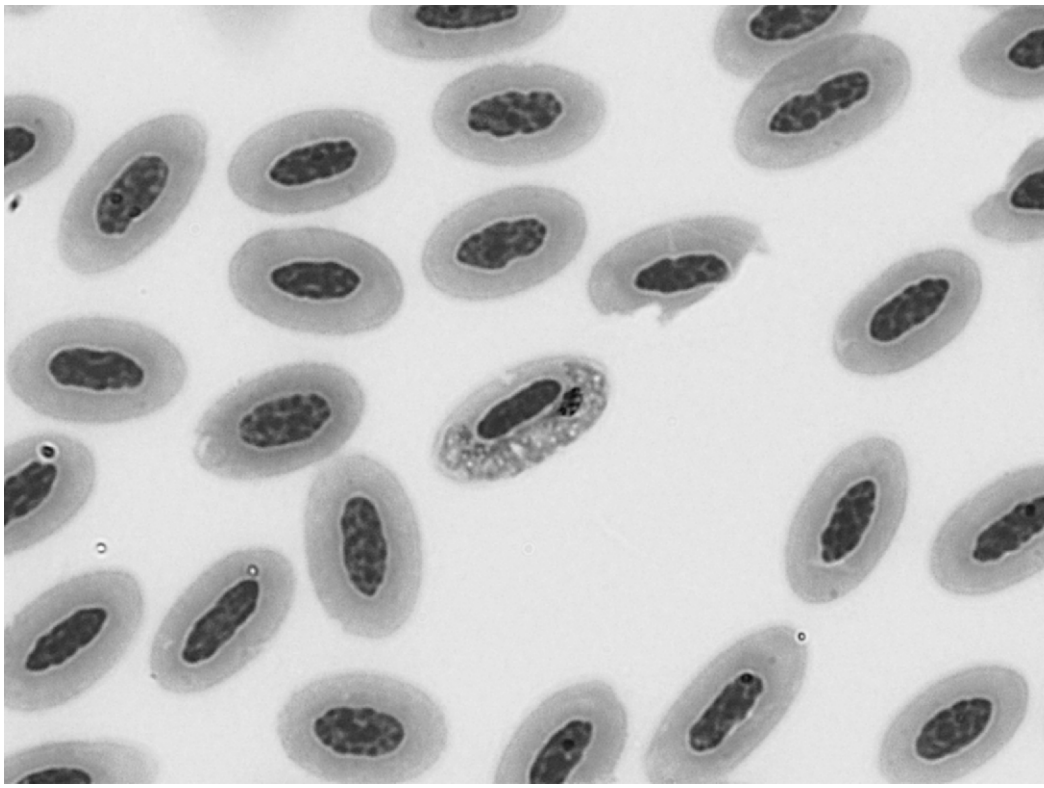

FIGURE 2. Gamete of *Plasmodium fallax* in erythrocyte of *Falco tinnunculus neglectus* on Santo Antão, Cape Verde.

cause vast distribution of hemosporidian infections, and migratory birds are likely to be important in dispersing parasites throughout the southwestern Pacific (Laird, 1960). Kestrels from the neighboring African mainland, for example, exhibit higher levels of infection (Earlé et al., 1991) than do more-isolated Cape Verde kestrels. Furthermore, the Cape Verde archipelago is situated in the dry belt of the sub-Sahara northeastern trade wind zone. Rainfalls are unpredictable and precipitation below 200 m altitude approximates 200 mm/yr (Slezak and Mandorfer, 1999); above 700 m altitude, clouds represent the source for precipitation. However, all infected kestrels had their territories near sea level. On Santo Antão only one temporary small river and no lakes exist. These conditions are not favorable for the development of possible vectors including both mosquitoes and ceratopogonid midges. The low prevalence of hematozoa probably reflects low transmission rates, which can, in part, may be due to low vector numbers. Further, the possibility that infection may occur more efficiently in large and dense kestrel populations may explain the higher observed prevalence on Santo Antão and Santiago as opposed to the Sal, Boavista, and Maio populations, which are smaller and widely distributed. The occurrence of *P. fallax* in Falconiformes is rare and Kučera (1981), Wiehn and Korpimäki (1998), and Krone et al. (2001) did not detect the parasite in *F. tinnunculus*. The presence of this parasite in the isolated kestrel population on Cape Verde is an interesting finding.

We are grateful to G. Valkiūnas, M. Peirce, and N. Robert for their help in identifying the parasites and R. Dennis, F. Furigo, S. Krätschmer, A. Kunz, U. Nau,

A. Nemitz, U. Nienaber, and G. Segelbacher for technical assistance in the field. We thank C. Beiglböck, M. East, and M. Visser for help with manuscript preparation and R. Adlard at Queensland Museum, South Brisbane, Australia for being in charge of reference samples of our specimens. This project was funded by the European Commission through a Marie Curie Fellowship to S. M. Hille.

LITERATURE CITED

ATKINSON, C. T. 1988. Epizootiology of *Haemoproteus meleagridis* (Protozoa: Haemosporina) in Florida: Potential vectors and prevalence in naturally infected *Culicoides* (Diptera: Ceratopogonidae). Journal of Medical Entomology 25: 39–44.

———, K. L. WOODS, R. J. DUSEK, L. S. SILEO, AND W. M. IKO. 1995. Wildlife disease and conservation in Hawaii: Pathogenicity of avian malaria (*Plasmodium relictum*) in experimentally infected Iiwi (*Vestiaria coccinea*). Parasitology 111: 59–69.

———, R. J. DUSEK, K. L. WOODS, AND W. M. IKO. 2000. Pathogenicity of avian malaria in experimentally-infected Hawaii Amakihi. Journal of Wildlife Diseases 36: 197–204.

———, R. C. UTZURRUM, J. O. SEAMON, A. F. SAVAGE, AND D. A. LAPOINTE. 2006. Hematozoa of forest birds in American Samoa—Evidence for a diverse, indigenous parasite fauna from the South Pacific. Pacific Conservation Biology 12: 229–237.

BENNETT, G. F., R. A. EARLÉ, H. DUTOIT, AND F. W. HUCHZERMEYER. 1992. A host–parasite catalogue of the haematozoa of the sub-Saharan birds. Onderstepoort Journal of Veterinary Research 59: 1–73.

BOURNE, W. R. P. 1955. A new race of kestrel *Falco tinnunculus* Linnaeus from the Cape Verde Islands. Bulletin of the British Ornithologists' Club 75: 35–36.

BUB, H. 1986. Vogelfang und Vogelberingung Teil III. Fang mit Schlagnetzen, Kätscher und Hand, Greifvogel- und Wasservogelfang, Abend- und Nachtfang, Fang an der Tränke Neue Brehm Bücherei, Ziemsen Verlag, Wittenberg.

DESSER, S. S., AND G. F. BENNETT. 1993. The genera *Leucocytozoon*, *Haemoproteus* and *Hepatocystis*. *In* Parasitic Protozoa, J. P. Kreier (ed.). Academic Press, San Diego, California, pp. 273–305.

DOGIEL, V. A. 1949. Biological peculiarities of parasite fauna of migratory birds. Izvestiya Akademii Nauk SSSR. Seriya Biologiya 1: 99–107. [In Russian.]

EARLÉ, R. A., G. F. BENNETT, H. DUTOIT, D. H. DESWARDT, AND J. J. HERHOLDT. 1991. Regional and seasonal distribution of avian parasites from northern South Africa. South African Journal of Wildlife Research 21: 47–53.

GABALDON, A., AND G. ULLOA. 1980. Holoendemicity of malaria: An avian model. Transactions of the Royal Society of Tropical Medicine and Hygiene 74: 501–507.

HAIBA, M. H. 1949. *Haemoproteus* sp. investigated in falcons (*Falco tinnunculus ruficoliformis* and *Falco naumanni pekinensis*) of Egypt. Journal of the Royal Microscopical Society 69: 239–242.

HILLE, S. M., M. NESJE, AND G. SEGELBACHER. 2003. Genetic structure of kestrel populations and colonization of the Cape Verde archipelago. Molecular Ecology 12: 2145–2151.

JARVI, S. I., J. J. SCHULZ, AND C. I. ATKINSON. 2002. PCR diagnostics underestimate the prevalence of avian malaria (*Plasmodium relictum*) in experimentally-infected passerines. Journal of Parasitology 88: 153–158.

———, M. E. M. FARIAS, H. BAKER, H. B. FREIFELD, P. E. BAKER, E. VAN GELDER, J. G. MASSEY, AND C. I. ATKINSON. 2003. Detection of avian malaria (*Plasmodium* spp.) in native land birds of American Samoa. Conservation Genetics 4: 629–637.

KORPIMÄKI, E., P. TOLONEN, AND G. F. BENNETT. 1995. Blood parasites, sexual selection and reproductive success of European kestrels. Ecoscience 2: 335–343.

KRONE, O., J. PRIEMER, P. SÖMMER, T. LANGGEMACH, AND O. LESSOW. 2001. Haemosporida of birds of prey and owls from Germany. Acta Protozoology 40: 281–289.

KUČERA, J. 1981. Blood parasites of birds in central Europe. 3. *Plasmodium* and *Haemoproteus*. Folia Parasitologica 28: 303–312.

LAIRD, M. 1960. Migratory birds and the dispersal of avian malaria parasites in the South Pacific. Canadian Journal of Zoology 38: 153–155.

MANWELL, R. D. 1935. How many species of avian malaria parasites are there? American Journal of Tropical Medicine 15: 265–282.

MARKOV, G. S. 1939. Dynamics of the parasite fauna of the s Uchenze Zapiski LGU. Seriya Biologiya LGU 11: 172–222. [in Russian.].

MELLOR, P. S., J. BOORMAN, AND M. BAYLIS. 2000. *Culicoides* biting midge: Their role as arbovirus vectors. Annual Review of Entomology 45: 307–340.

PEIRCE, M. A., AND J. E. COOPER. 1977. Haematozoa of East African birds. East African Wildlife Journal 15: 213–216.

———, A. G. GREENWOOD, AND J. E. COOPER. 1983. Haematozoa of raptors and their birds from Britain, Spain and the United Arab Emirates. Avian Pathology 12: 447–59..

———, G. F. BENNETT, AND M. BISHOP. 1990. The

haemoproteids of the avian order Falconiformes. Journal of Natural History 24: 1091–1100.

SLEZAK, G., AND P. MANDORFER. 1999. Länderprofil/Österreichische Forschungsstiftung für Entwicklungshilfe Kap Verde. Südwind, Vienna, Austria, 52 pp.

STEADMAN, D. W., E. C. GREINER, AND C. S. WOOD. 1990. Absence of blood parasites in indigenous and introduced birds from the Cook Islands, South Pacific. Conservation Biology 4: 398–404.

VALKIUNAS, G. 2005. Avian malaria parasites and other haemosporidia. CRC Press, Boca Raton, Florida.

———, AND T. IEZHOVA. 1993. *Haemoproteus kairullaevi* sp. (Haemosporidia: Haemoproteidae) from common mynah *Acridotheres tristis* (Passeriformes). Zoologiya Zhurnal (Moscow) 72: 141–146. [in Russian.]

WARNER, R. E. 1968. The role of introduced diseases in the extinction of the endemic Hawaiian avifauna. Condor 70: 101–120.

WASIELEWSKI, T., AND G. WÜLKER. 1918. Die Haemoproteus—Infektion des Turmfalken. Archiv für Schiffs- und Tropenhygiene 22: 1–100.

WEATHERHEAD, P. J., AND G. F. BENNETT. 1992. Ecology of parasitism of brown headed cowbirds by haematozoa. Canadian Journal of Zoology 70: 1–7.

Received for publication 2 October 2006.