HAEMOSPORIDIOSIS AS A FATAL DISEASE IN MUSCOVY DUCKS (CAIRINA MOSCHATA) IN SOUTH BOHEMIA

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Abstract. A description of the disease afflicting muscovy ducklings (Cairina moschata) which repeatedly occurred in South Bohemia during the last decade. The symptoms and causative agent seem to be similar to the earlier described case of illness in muscovy ducks reported from the Rhine region (Comínchau and Jonas 1977). However, the disease was not caused by Le. va. von simondii, as the German authors erroneously supposed, because the schizogonic stages from the internal organs of the infected ducks considerably differ from the developmental stages of Le. von simondii. The causative agent is probably some species of haemosporisidae acquired from the local populations of wild birds, a species able to attain partial development in the internal organs of muscovy ducklings. In this aberrant host, however, only schizogonic division takes place, the parasite is unable to form the body stages and consequently complete its life cycle. As the morphology of blood stages is unknown, the causative agent cannot be identified. In the morphology of schizogonic stages the causative agent resembles the parasites of the genus Haemoproteus or Plasmodium, but it is less similar to Leucocytozoon. It is evidently not directly the species Haemoproteus nettionis which seems to have caused a similar disease in muscovy ducks in Canada (Julian and Galt 1980), because this species is missing in Central Europe.

In 1972—1979 an outbreak of a disease occurred in muscovy ducks raised in several localities in the South Bohemian region which showed high morbidity and mortality mainly in ducklings. The examination of dead and ill birds revealed that this disease was probably similar to that described by Comínchau and Jonas (1977) in muscovy ducks in the Federal Republic of Germany. The mentioned authors consider Leucocytozoon simondii to be the causative agent of the disease in question. At the beginning we also thought it to be an infection with this parasite, but at closer comparison it became evident that the infection was caused by a haemosporidal agent which considerably differs morphologically from Le. simondii. Julian and Galt (1980) described cases of a very similar disease in muscovy ducks from Canada and indicated Haemoproteus nettionis as the agent. Owing to the fact that this species does not occur in anatids in Central Europe (Kučera 1981, 6) it was very likely another species of blood parasite. The present paper therefore describes the parasite found and discusses which species of blood protozoan it might be.

MATERIAL AND METHODS

Cases of an infection in muscovy ducks (Cairina moschata L. forma domestica) were reported by individual breeders from the following localities in the South Bohemian region:

1. Locality in the district of Blatná. The infection occurred in July and August 1972. A total of 24 ducklings, produced by two ducks, died. The first duck used to lead her young to the local pond once their hatching and at age 3 weeks all 11 ducklings died. The second duck which had produced 17 their hatching and at age 2 weeks occasionally ducklings was kept away from the pond, but in its immediate vicinity and a few ducklings occasionally escaped to the pond. From this brood 13 ducklings died at age 4 weeks and the remaining full 2 ducklings survived. In mid-September the first duck produced nine ducklings which were prevented from going to the pond at all and no one of them became infected.

2. Locality of Trebení. The infection occurred in July 1973 among the ducks of a breeder and a few ducklings died at age 4—5 weeks.
3. Locality in the district of Prachatice. The infection occurred repeatedly in the summer of 1977 and in August 1978. The owner kept the ducks few feet away from the water since hatching. In the former year a few ducks died, in the latter year 12 out of 17 ducklings died at age 14 days. The illness was observed in late April in 1978 when one young, yet one-year-old duck died.

4. Locality in the district of České Budějovice. In August 1980 about 15 ducklings aged 2-6 weeks died. From this locality the illness was already reported on 17 December 1979 when one young, not yet one-year-old duck died.

5. Locality in the district of Tábor. In October 1978 one two-month-old duck died.

Well was seeking in retrospect the data referring to the above mentioned cases we failed to find any more details.

Practically in all cases the ducks were kept in small flocks, could move freely since hatching and were allowed access to ponds or streams around the breeder's house. In some cases the ill ducklings were treated with Sulafamidin and Furazolidin. Some of the ill ducklings from the České Budějovice locality were hospitalized at the State Veterinary Institute where they were treated with Pusranol (phlorrhizin + flucitidin) in a dose of 0.25 g/kg of feed.

The ill or dead birds were histologically investigated and some of them were tested microbio-
logically. However, only histological investigations yielded positive results. If possible, also blood smears and bone marrow impressions were made at autopsy; after fixation in methylalcohol the impressions were stained with Giemsa. The lungs, liver, gut, spleen, brain, heart, kidneys and samples of skeletal muscle of the dissected birds were fixed in 10% formal and the histological preparations were stained with hematoxylin-eosin. The examinations were performed under microscope by means of ul immersion objective (10-fold magnification). A few healthy adult ducks from contaminated localities were also examined by means of blood smears. These birds, however, were found to be negative.

Histological preparations with hepatic schizonts and merozoïdes of Leucocytozoon simoni were loaned material from the collections of the Department of parasitology, Natural Sciences Faculty at the Charles University in Prague by courtesy of Dr. KNDZ. Černá. The histological preparations with merozoïdes of parrot aberrant leucocytozoonosis were obtained by courtesy of M. Lavička, M.V.D., of the Central State Veterinary Institute in Prague.

RESULTS

In the infected muscovy ducklings torpidity, even lethargy and reluctance to movement were noted. The birds breathed with difficulty (had laboured breathing) especially when they were forced to move and refused to drink and feed. The symptoms lasted about a week and the majority of the infected ducklings died during this period. The surviving ducks were in a very bad condition and were lagging behind their development. The drugs administered to some ducks (see Material and Methods) had no essential effect on the course of the illness. The drugs, however, were administered only after the appearance of clinical symptoms, when the pathological changes in the organs of the ill ducklings were more or less irreversible character, so that the treatment was of no avail.

The dead and ill birds were dissected and most organs revealed important pathological changes. The congested epicardium was with numerous petechiae and in some cases hydropic edema was observed. The lungs were heavily filled with blood, with oedema and filaments and firm to touch. The liver was enlarged and congested, sometimes with petechiae under the liver sheath. The kidneys were palid, anemic, only locally congested and penetrated by petechial haemorrhages. Sometimes, evidently due to the thirst of ducklings, the kidneys were swollen, enlarged, containing stigmata urates in the distended renal tubules. Likewise the spleen was sometimes enlarged, with dilated blood vessels under the capsule. In some cases congestion in the mesentery and serosity of gut were observed.

In all cases the histological sections of organs showed, under microscope, schizogonic formations located partly in the endothelial cells of capillaries, blood vessels and sinusoids and partly in the poorly defined macrophages. As a rule, masses of schizonts were found in the lungs, deposited in blood extravasates and in the blood-filled blood vessels with a marked admixture of eosinophils. Likewise the spleen, and propria mucosa of intestines were infected, showing identical inflammatory infiltration. The schizonts were less frequently found in the endothelium of the blood vessels of brain, kidneys, heart and the skeletal muscle as well as sinuodoids. In addition, dystrophic even necrotic cellular changes and mixed inflammatory infiltrates in portal fields with marked admixture of eosinophils were noted in the liver parenchyma. The schizogonic formations in less damaged tissue parts currently occurred in the endothelial cells along the blood vessels. The schizonts in the inflammatory infiltrates were detected in undetermined macrophages. The size of the host cell was somewhat enlarged, the nucleus however did not as a rule, differ from the nuclei of infected cells. The schizonts were not observed in the blood smears.

DURATION OF INFECTION

In detecting the schizonts in the blood smears the process was based mainly on the size of the intracellular forms of the merozoïdes, their distribution in the blood blood vessels, and the ratio of normal blood cells to infected cells.

In the blood smears infected cells were found in the following proportions: 1:0:17, 1:0:20, 1:0:25, 1:0:30, 1:0:35, 1:0:40, 1:0:45, 1:0:50, 1:0:55, 1:0:60, 1:0:65, 1:0:70, 1:0:75, 1:0:80, 1:0:85, 1:0:90, 1:0:95, 1:1:00. The ratio of infected cells to normal cells was determined with great accuracy.

The size of the schizonts varied between 3.7 and 8.1 pm depending on the thickness of the capillary. When more cells in a row were infected, the length of schizonts seemed to be much greater (Plate II, Fig. 9). Schizogonic formations of the second type in macrophages (Plate I, Figs. 2, 5, 8, Plate II, Figs. 11, 13) were 11.1 x 7.3 pm large on the average, the smallest measuring, 4.7 x 3.9 pm and the biggest 24.7 x 9.5 pm. In the clusters of infected cells the separate schizonts were difficult to distinguish, so that the schizogonic formations seemed to have even larger dimensions (Plate I, Figs. 8, Plate II, Fig. 13). In histological preparations the schizonts inside the schizonts appeared to be spherical, oval or even crescent-like corpuscles of dark colour, frequently with a lighter ring around. Sometimes they were very minute, measuring 0.5 x 0.5 pm, the biggest reaching the size up to 1.9 x 1.4 pm. Judging from the size of the merozoïdes two types of schizogonic stages seemed to be present here: schizonts with tiny merozoïdes and schizonts with big merozoïdes. Moreover, there were also various transient forms of merozoïdes and their average size was 1.4 x 1.1 pm. The schizogonic formations were also found on the bone marrow impressions stained by Giemsa. Likewise, they occurred here in macrophages and the merozoïdes were almost always of ring-like form with dark red nuclei and a band of pink-blue cytoplasm around a small vacuole (Plate I, Figs. 6, 7, Plate II, Fig. 12). A band of the dimensions of these schizonts as well as merozoïdes agreed with the formations observed in histological preparations.

Merozoïdes were far less frequently found in the blood smear from the infected birds. They occurred either in the regressive changed or partly desintegrated. The leucocyte type and their morphology was of the same ring-like type as that observed in the bone marrow impressions, or they occurred freely in the blood plasma. These free merozoïdes were most frequently of oval shape, measuring 0.5 x 1 pm on the average, with a red-stained nucleus and a thin band of blue cytoplasm. Most frequently they occurred in clusters (Plate II, Fig. 14). Apart from these merozoïdes no other developmental stages of haemoporiodesia were present in the peripheral blood of the ill or dead birds.

DISCUSSION

The disease observed in muscovy ducks, which was caused by the above described parasite, may be designated as haemoporiodesis. It is evidently haemoporiodesis despite the fact that we have not found in the blood stages of these parasites. The schizonts found in the internal organs of the infected ducks are similar to typical developmental stages of a number of haemoporiodesis (see e.g. Garnham 1966) not only in their morphology, but in the localization in the endothelial cells and in the macrophages and in the distinct spread in the blood stream to all internal organs. The infected ducklings were given access to free nature since their hatching and apparently became infected.
there by the blood-sucking insects. The disease occurred most frequently in June to August, namely in the period when an intensive transmission of avian haemosporidian by insect vectors takes place (Kučera 1981b, c). The sporadic case reported from December 1979 may be regarded as a relapse of the previously acquired infection or explained by the fact that November and December of that year were extraordinary warm, so that the transmission by blood-sucking insects cannot be ruled out even in this period.

The incidence of the disease is supposed to be far higher than that revealed by the number of cases related by us. The muscovy ducks are raised in South Bohemia exclusively by individual breeders and their flocks are not under regular veterinary control. Only a few breeders submitted their dead or ill birds to veterinary examination, so that many cases went unnoticed. The first findings of the parasites discussed were made by one of the authors (K. M.) shortly after her arrival at her working place in České Budějovice in 1972 and it may be surmised that the disease in question probably occurred in South Bohemia much earlier.

Practical identical infection in muscovy ducks with similar symptoms and morphologically very similiar parasite was described previously by Commichau and Jonas (1977) from the Rheinland in the Federal Republic of Germany. Although these authors did not detect gametocytes in the blood of the surviving ducks either, they determined the causative agent only on the basis of schizogonous stages in the internal organs of L. simondi causes high losses primarily in North America (e.g. Herman 1963 etc.), but also occurs in Europe, though it is quite rare in Anseriformes in Central Europe and seems to be rather a northern species (Kučera 1981b, c, Herman 1963). The comparison between the morphology of schizonts observed in the internal organs of muscovy ducks and the morphology of schizonts observed in the internal organs of muscovy ducks and the morphology of internal stages of L. simondi shows that the parasites found in muscovy ducks cannot be considered as developmental stages of L. simondi. While making this comparison we not only used published data on the development of L. simondi (Garham 1968, Fallis and Eide 1970, Eide and Fallis 1972, Dessier 1973, Fallis and Dessier 1974, 1977, Dessier and Ryckman 1976, Herman et al 1977 etc.), but also the comparative material of haemosporidian and megaloschizonts of L. simondi in histological preparations (see Material and Methods). In their size the hepatic schizonts of L. simondi resemble the stages found by us in the internal organs of the muscovy ducks. However, in the L. simondi infection these stages are encountered only in parenchymous cells of liver, while the schizonts observed by us in muscovy ducks occurred in macrophages and endothelial cells of all organs examined and never in the parenchymous cells of liver. Likewise megaloschizonts, which are so typical of the infection caused by L. simondi, were never found in our material. Although Commichau and Jonas (1977) consider some stages from internal organs of muscovy ducks to be megaloschizonts, they did not give their description in detail. The photographs published by them, however, show that these stages are probably identical with the stages found by us and are not the true megaloschizonts. After Fallis and Dessier (1974) the megaloschizonts of L. simondi are characterized primarily by the fact that: 1. They originate from the hepatic schizonts and develop in reticuloendothelial cells primarily of the vascular endothelium, 2. they cause extreme hypertrophy of the host cell nucleus and 3. grow to considerable size (100 or more than 400 μm), producing over one million of merozoites. The schizonts observed by us in the organs of muscovy ducks were present in the reticuloendothelial cells (endothelium, macrophages), but they never caused hypertrophy of the host cell nucleus; their average size was 11.1 ± 7.3 μm and the maximum length of elongated forms was 40 μm. They also contained only a few scores of mezoocytes and never had the typical habitus of megaloschizonts, as could be verified directly from the comparative material.

Commichau and Jonas (1977) probably confused the two organisms due to the fact that in heavily infected organs of muscovy ducks are sometimes closely packed one on another that they appear to be bigger than the individual schizonts (see e.g. Plate I, Fig. 8 and Plate II, Fig. 13).

Another proof that the infection in question was not caused by L. simondi is the fact, that no gametocytes typical of this species were ever encountered in the blood of muscovy ducks by Commichau and Jonas (1977). It is true that haemosporidian gametocytes appear in the blood of their hosts only after a certain prepatent period, which lasts about 4 to 5 days in L. simondi. If it were an infection by L. simondi, we should have found gametocytes in ducks which survived the infection. Briggs (1960) also compared the course of infection caused by L. simondi in domestic ducks and in muscovy ducks and detected gametocytes in the blood of both duck species, though in muscovy ducks gametocytaemia was lower. Unfortunately this author paid no heed to the morphology of the internal stages of L. simondi in the ducks in question.

The muscovy duck is also known to be the host of L. simondi in other regions (Lapage 1961, Garnham 1966, Hau et al. 1973), so that the L. simondi infection in our case is to be atypical owing to unusual host is improbable.

Identical infection in muscovy ducks was also observed in Canada (Julian nad Galt 1980). Neither these authors found any gametocytes of haemosporidian in the blood of the infected birds, and described schizonts of identical morphology in their internal organs as in our case. Moreover, their paper shows that the causative agent is Haemoproteus, most likely a species occurring in Anseriformes — H. nettonius (Johnston et Clendan, 1909) Coster, 1936. This parasite has sofar not been considered to be non- pathogenic to the muscovy ducks by us (1973). After Julian and Galt (1980) its increased pathogenicity to muscovy ducks and inability of forming gametocytes in this host is due to the fact that muscovy ducks (genus Cainta) are unusual hosts for this parasite. Muscovy ducks originally come from South America and in the North America Haemoproteus nettonius occurs in ducks of other genera (primarily Anas) systemically related to the genus Cainta.

In Central Europe, however, Haemoproteus does not occur in Anseriformes (Kučera 1981b) and from other parts of Europe only two sporadic cases of infection by these parasites were reported from ducks in the Paris and London zoos (Kovatsi et al. 1957, Hammerton 1931). After Williams and Bennett (1980) the Haemoproteus species in anatids are distributed throughout the world except the Palaearctic region. Consequently, in our case the infection was not caused by Haemoproteus nettonius directly, but most likely by another species of haemosporidian.

Having their site in endothelial cells of capillaries and sinuosids of internal organs of muscovy ducks and due to their morphology as well, the described schizonts very much resemble not only the schizogonous stages of some known Haemoproteus species (see Nusskern 1980, Bakker 1966, Garnham 1966, Kohn and Fiala 1973, Peirs 1976), but similar stages of Plasmodium, primarily the species of the subgenus Haemamoeba (see e.g. Garnham 1966 etc.). However, Plasmodium does not occur in free-living Anseriformes in Central Europe either (Kučera 1981a, c) and even from other parts of Europe not a single finding of these parasites in Anseriformes is known.

On the other hand, different species of Haemoproteus and Plasmodium as well as Leucocytozoon were observed in other wild birds in Central Europe (Kučera 1981b, c).

It may be gathered from the above said that muscovy ducks in our case must have been infected by some unspcific parasite species acquired from local populations of wild birds, apparently other than Anseriformes. Consequently, in our case the infection
may be considered an aberrant infection in muscovy ducks, because it was caused by an unusual parasite species in an exotic host. The causative agent is virulent to such an extent that it is able to develop in the internal organs of muscovy ducks and to cause death of these hosts. It is, however, unable to complete its life cycle in this unusual host and to form blood stages (gametocytes) of further transmission.

A similar aberrant haemoparasitism in Europe occurred in the last two decades as the so-called aberrant leucocytozoan stages of parrots kept in open-air cages (see e.g. Frank 1965, Walker and Garnham 1972, Minnik and Dym 1972). This disease affecting the young of Australian parrots shows similar symptoms as those described by us in the case of infection of muscovy ducks. In the internal organs of the infected parrots, however, typical megaloschizonts were present which are an evidence that in this case the infection was caused by some undetermined haemoparasite species (Frank 1965, Walker and Garnham 1972). However, these megaloschizonts are absolutely different from the stages present in the internal organs of muscovy ducks. We have verified this fact directly in the comparative material from the parrots.

The morphology of schizonts present in the internal organs of muscovy ducks shows that in our case the causative agent of the disease is most probably some species of *Haemoproteus* or *Plasmodium*.

In view of our insufficient knowledge of the morphology of similar stages in the majority of known species of haemoparasida in Central Europe also the related *Leucocytozoon* may be involved. This genus namely contains species known for the absence of megaloschizonts in their development (Khan and Fallis 1970, Clark 1965). The differentiation of genera and species of avian haemoparasida, however, is mostly based only on the morphology of blood stages (Garnham 1960). In our case this morphology is unknown and therefore it is very difficult to identify the parasites described in muscovy ducks.

The above discussion shows that the infection in muscovy ducks was not caused by *Leucocytozoon simpsoni* as had been supposed earlier (Commichau and Jonas 1977). It was apparently an aberrant infection caused by an undetermined haemoparasite, whose reservoir are probably the populations of wild and domestic birds. In agreement with Khan and Galt (1960) the most probable causative agent seems to have been *Haemoproteus*, but some other species of *H. nitidus* typical of ducks, which does not occur in Anseriformes in Central Europe. The morphology of the parasite stages found indicates that *Plasmodium* might be involved and *Leucocytozoon* for the time being, cannot be ruled out, either.

**БИДОСОПИДИОЗ КА МРИМЕРЕННОЕ ЗАБОЛЕВАНИЕ У ТОК ВИДА CARRINA MOSCHATA В ЮЖНОЙ ЧЕХИИ**

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Резюме. Данное описание заболевания утка видов *Cairina moschata*, повторяющегося в южной Чехии в последние десятилетия. Признаки и возбудители заболевания описываются сходными с описанным заболеванием уток видов *Cairina moschata* (Khan et al. 1977). Однако, это заболевание не вызвано организмом, идентичным описанному заболеванием уток видов *Cairina moschata* (Khan et al. 1977). Объяснение возникновения заболевания ведется появившейся вирусной инфекции данному виду *Cairina moschata*, который собирался частично развивается в внутренних органах уток. Этот вирус необычайно опасен, но, в то же время, не окажет влияния на развитие уток.

**Список литературы**


Minnik P., Dym B., *Leucocytozoon* as
A CONTRIBUTION TO THE KNOWLEDGE OF LOUSE FLIES (DIPTERA, HIPPOBOSCIDAE) FROM AFGHANISTAN

The research workers of the Institute of Parasitology, Czechoslovak Academy of Sciences, studied the parasites of domestic animals in Afghanistan during an expedition organised in the autumn (September—October) of 1976. The collections and studies were carried out at the Kabul abattoir and in some other localities. The results follow up the line of the previous expedition in 1974 (Minář J. et al., Folia parasit. (Praga) 24: 92—93, 1977). The authors thank Dr. Dr. K. Blažek and Dr. A. Amin for their collaboration.

Hippoboscus longipennis Fabricius, 1805


Lipoptena capreoli Rondani, 1878

Material: 6♂♂, 18 ♀♀, Kabul 27. 9. 1976, Host: domestic goat (Capra hircus L.). This species was found in Afghanistan by the previous expedition of the Czechoslovak Academy of Sciences (Minář et al. 1977). The hitherto found specimens were wingless and collected in the autumn period.

Pseudolynchia canariensis (Macquart, 1840)

Material: 3♂♂, 5 ♀♀, Kárenzmir, 6. 10. 1976, Host: domestic pigeon (Columba livia L.). This species is distributed in the south of Europe, in subtropics and tropics of Asia and Africa. It was reported from Afghanistan by Maa (Studies in Hippoboscidae (Diptera), Panific Insects Monograph 10, Ent. Dept. B. P. Bishop Mus., Honolulu, 148 pp., 1966).

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Fig. 1. Elongated schizont in the endothelium of capillary in the heart muscle. Fig. 2. Schizont in the liver sinusoid. Fig. 3. Schizont in the endothelium of capillary in proprion mucosa of intestine. Fig. 4. Cross section of capillary in kidney almost blocked by schizont located in the endothelium. Fig. 5. Two schizogenic stages in the inflammatory infiltrate in the lungs. Fig. 6. Schizonts with ring-like formations in the bone marrow impression. Fig. 7. Distorted ring-like forms in the macrophage from bone marrow. Fig. 8. A cluster of schizogenic formations in the inflammatory infiltrate in proprion mucosa of intestine. Figs. 1—8 have been drawn half schematically from the preparation by means of Abbe's drawing apparatus in the uniform scale of magnification (see the abscissa 10 μm in Fig. 2).

Fig. 9. Photomicrograph of two schizogenic formations located in a row in the endothelium of capillary in muscle layer of intestine. Fig. 10. Schizont in kidney capillary (cross section). Fig. 11. Schizont in liver sinusoid. Fig. 12. Schizogenic stage with ring-like forms from the bone marrow impression. Fig. 13. Inflammatory infiltrate in proprion mucosa of intestine with numerous schizonts. Fig. 14. A cluster of merontes in the blood plasma on blood smear. All photomicrographs are in the same scale of magnification, corresponding with the scale of Figs. 1—8 in Plate I (see abscissa 10 μm in Fig. 2).