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Psittacosis in Domestic Pigeons.

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It may perhaps be taken for granted that all members of the order, Psittaciformes, are susceptible to psittacosis. Within the order are parrots, macaws, conures, corellas, quarrions, cockatoos, parrotlets, cockateels, paroquets and parrakeets. The budgerigar or lovebird (*Melopsittacus undulatus*) is the shell parrakeet and, of course, is often the source of human infections.

Most public health regulations of the present day are designed to counter the dangers associated with the Psittaciformes, and it is not widely recognised that avian species without this order can, on occasion, be a menace to man.

In 1933, Meyer and Eddie isolated a relatively weak strain of the virus from apparently healthy canaries (*Serinus canarius*), that were associated with two human cases of psittacosis in one household. About the same time these authors inspected an aviary and found a listless butterfly finch (*Cyanospiza ciris*), which had a distended abdomen, its plumage ruffled and its tail soiled with faeces. The virus was not detected by microscopic examination of material from the finch, but the intraperitoneal injection of a liver and spleen emulsion killed test mice in ten days with typical psittacosis.

Meyer afterwards demonstrated that the Java rice bird (*Padda oryzivora*) could become infected naturally. He also showed the Pekin robin (*Liothrix luteus*) and the bullfinch (*Pyorrhula vulgaris*) to be susceptible.

In 1938, Haagen and Mauer in Germany recorded the presence of psittacosis virus in imported finches and in indigenous siskins (*Spinus spinus?*) and coaltits (*Parus ater?*); these were all natural infections. In the same year the same authors proved that the fulmar petrel (*Fulmarus glacialis*) was responsible for the disease in man in the Faroe Islands.

Gradually it is being realised that no avian species can be regarded as harmless, unless exhaustive experiments have proved it to be insusceptible. Even the domestic chicken can be infected artificially.

So far as we are aware, psittacosis has never been diagnosed in the domestic pigeon (*Columba livia* var. *domestica*) and a recent outbreak forms the subject of this paper.

In March 1939 a big pigeon breeder in Johannesburg, Transvaal, sent two young fancy domestic pigeons for examination. Both birds were dead on arrival. The owner stated he had about 200 pigeons and that a few of them were ailing. One of the birds, that died before it was sent, had been ill only four days; the symptoms were listlessness and lack of appetite. The other had been off colour two or three weeks, and had moped and shown no interest in food.

We examined the carcases superficially, and noted that the vent feathers were soiled with diarrhoeic faeces. Veterinary students were then told to perform the autopsies, and they were warned to be careful and not infect their eyes and mouths, as it was possible that the deaths were due to salmonellosis. The first pigeon had a liver twice the normal size and the organ was diffusely yellowish in parts; there was pronounced intestinal catarrh; no other obvious lesions were detected. The second pigeon was rather decomposed; the liver was slightly swollen; there was marked catarrh of the intestines; a moderate degree of aerocystitis characterised the left abdominal air sac. Heart blood from each bird was seeded in brilliant green bouillon and on brilliant green agar slants—no bacterial growth occurred.

Smears of the heart blood, lungs and spleen were stained with Giemsa. The students were dismissed. An hour later, the lung and spleen smears of the first pigeon were found to contain rather numerous colonies of psittacosis virus; the organisms were relatively infrequent in the spleen and liver of the second. As was to be expected, the virus particles showed a marked predilection for macrophages.

The viscera of the birds were retrieved and an emulsion of the lungs of both pigeons was inoculated intraperitoneally into 6 white mice. Five of the mice died of bacterial septicaemia within 2 days. On the fifth day the remaining mouse died of typical psittacosis and the L.C.L. bodies were seen in fairly large numbers in the macrophages of the peritoneal exudate.

A spleen and liver emulsion of this mouse was injected intraperitoneally into 6 more mice. Owing to the danger of spreading the infection in unsuitable rooms, these mice were sacrificed 5 to 7 days afterwards, and the parasites were demonstrated in the spleens and peritoneal exudates of all.

A visit was paid to the infected premises. The aviaries were beyond reproach and no sick pigeons were observed. The only other bird present was an apparently healthy budgerigar, and it was in contact with the pigeons. No legal authority existed for dealing with the outbreak, and the owner was not disposed in any way to worry about it. As a result, we were unable to examine even the lovebird to see if it was a carrier.

Although four students, two assistants and the author had been exposed to considerable risk of infection, none of them subsequently developed any signs of illness.

In February 1940, another young pigeon was received from the same breeder. It died just after it arrived and the vent feathers

were soiled with liquid faeces. Adequate precautions were taken to prevent human infection. There was a yellow diphtheritic pseudomembrane on the back of the tongue, due to *Trichomonas hepatica*. The wall of the left abdominal air sac was thickened and turbid. The liver was slightly swollen and there was a marked pseudomembranous perihepatitis, but no trichomonads or bacteria could be found in smears made from the surface of the liver. The spleen was a little enlarged and light pink in colour, and a prolonged search revealed four macrophages containing psittacosis granules. A blood smear showed a few *Haemoproteus columbae*. Six white mice were inoculated intraperitoneally with an emulsion of the heart, lungs, spleen and liver; unfortunately, all died within a day as a result of a *Salmonella typhimurium* infection, the presence of which had not been suspected when the bird was autopsied. Brilliant green agar slants seeded with the heart blood of the pigeon, showed colonies of *S. typhimurium* in 24 hours. This pigeon undoubtedly died of salmonellosis. It was infected also with *Trichomonas hepatica*, *Haemoproteus columbae* and the virus of psittacosis Unfortunately, the presence of the last named could not be confirmed by mouse inoculation.

The owner was requested to send two more pigeons. These soon arrived, and were young. One was dead and slightly decomposed. The vent feathers were soiled because of diarrhoea; the lungs were oedematous, and the liver and spleen swollen; spleen and liver smears were full of pigment, due to the breaking down of numerous *H. columbae*. *S. typhimurium* was isolated from the heart blood. Six white mice were inoculated intraperitoneally with an emulsion of liver, spleen and lung, but all died within 5 days of a bacterial peritonitis and septicaemia.

The living pigeon was rather weak and emaciated and had diarrhoea. It was killed. Nothing special was to be observed at autopsy, except a few cheesy lesions in the mouth due to *T. hepatica*. *H. columbae* appeared infrequently in the red cells, and a blood culture yielded a few colonies of *S. typhimurium*. An emulsion of liver, spleen and lung was injected intraperitoneally into 6 white mice and two died of peritonitis within 3 days. The other four mice lived and were sacrificed after 12 days, as they looked healthy. All four had rather swollen spleens and slight ascites. One animal had leucaemia. The virus of psittacosis was easily found in macrophages in a smear of the liver surface of one mouse; in a second mouse the parasites were rare; in the remaining two mice no organisms could be seen.

Thus, these last two pigeons again showed *T. hepatica*, *H. columbae* and *S. typhimurium* infections. In one of them, the presence of psittacosis was confirmed by mouse inoculation, although the organisms were not shown microscopically in the bird itself. The deaths were undoubtedly due, mainly if not entirely, to paratyphoid.

These experiences are reported at length, because they illustrate most clearly the difficulties that may be encountered when an attempt is made to diagnose psittacosis, particularly the latent form. One

should not be deterred or defeated by the presence of salmonellosis; in such cases mice should be injected with material from birds that have only just sickened for, in them, the Salmonellas will probably be too few to kill a mouse. Theoretically, it is possible to separate the Salmonellas from the L.C.L. bodies by filtration or centrifugation, but this is hardly likely to be successful when the latter are rare. Another complicating factor when we consider filtration and centrifugation is the relative weakness of many psittacosis strains for mice; we cannot afford to lose any of the little virus present by these processes. Strains from Australian birds are usually of comparatively low pathogenicity, and the new Johannesburg pigeon strain seems to be the same.

It is not unusual for paratyphoid fever to complicate psittacosis infections in avians. Nocard in 1893 isolated a Gram-negative bacterium from a bird with parrot-fever, and called it *Bacillus psittacosis*. It now seems certain that this organism was really *S. typhimurium*. Meyer and Eddie reported that salmonellosis is not infrequently found in shipments of South American birds, and they diagnosed dual infections of paratyphoid and psittacosis in some paroquets. The great majority of these avian Salmonellas are IV variants of *S. typhimurium*, i.e. *S. typhimurium* var. *Copenhagen*.

Summary.

Psittacosis has been found in fancy domestic pigeons in South Africa. This is the first record of the disease here, and probably the first of its presence in pigeons. The diagnosis was confirmed by mouse inoculation. The virus seems to resemble most Australian strains, in that it is not highly virulent for mice.

Some of the pigeons were also suffering from *S. typhimurium*, *H. columbae* and *T. hepatica* infections.

Psittacosis apparently has not occurred in the owner of the pigeons, or in his family and servants. Seven students and laboratory workers remained healthy, although exposed for about two hours to the infection while the first birds were being autopsied.

Acknowledgment.

The writer wishes to thank Mr. T. Meyer of this institute for preparing the photomicrographs of the organisms in the pigeons.

ADDENDUM.

At the end of 1940 the owner of the pigeons consented to their destruction and 282 were killed. Only two looked sick—due to sporadic diseases. Spleens were collected in 50 per cent. glycerine from a third of the birds, ten being put in each bottle. Next morning six white mice were injected intraperitoneally with a mixture of the mushed spleens in each bottle. A few died of intercurrent diseases during the following three weeks, but in no instance was psittacosis diagnosed. The survivors were then killed for the inoculation of a second generation of mice. Again it was impossible to set up

psittacosis. Besides the pigeons and a few bantams kept well away from them, no avians were on the premises. These results suggest that psittacosis does not easily spread from one pigeon to another.

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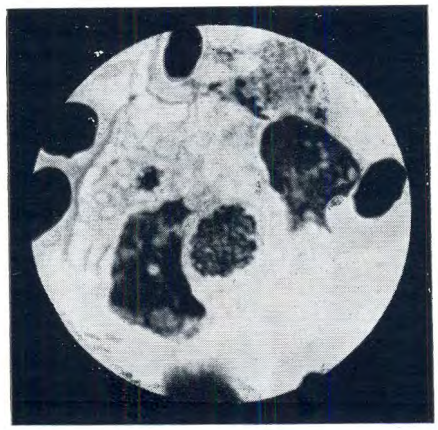

Fig. 1.—1400 x. Pigeon. Lung smear. Vacuolated macrophage containing a large colony of Levinthal-Coles-Lillie bodies to the right of the nucleus, and a smaller colony above the nucleus. To the right of and above this cell, is another cell parasitized mainly with initial bodies.

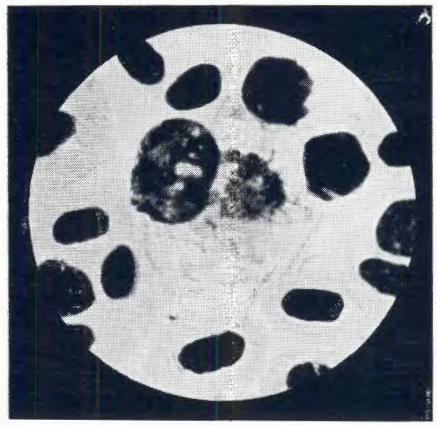

Fig. 2.—1400 x. Pigeon. Lung smear. Macrophage containing a colony of elementary bodies to the right of the nucleus.

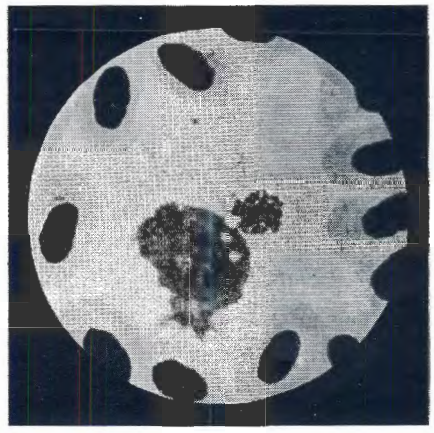

Fig. 3.—1400 x. Pigeon. Lung smear. Macrophage harbouring a colony of *Rickettsia psittaci*.

Fig. 4.—1400 x. Pigeon. Lung smear. A group of initial and elementary bodies lying scattered between erythrocytes.

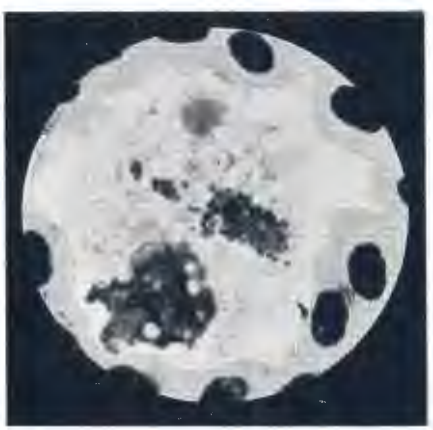

Fig. 5.—1400 x. Pigeon. Lung smear. Macrophage with elementary bodies in the cytoplasm.

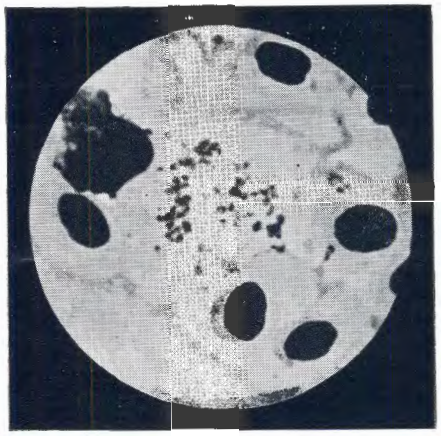

Fig. 6.—1400 x. Pigeon. Lung smear. A free-lying group of initial and elementary bodies.

All smears were strained with Giemsa.

Lillie called the parasites, *Rickettsia psittaci*, in 1930. In the same year Levinthal proposed the name, *Microbacterium mutiforme psittacosis*. Most authors usually refer to the organisms as L.C.L. bodies, or Levinthal-Coles-Lillie bodies. The elementary bodies are the small particles of the virus, that stain like chromatin. Initial bodies are the larger virus particles, that stain light blue with Giemsa.