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THE WHITE-CROWNED PIGEON A FRUIT-EATING PIGEON AS A HOST FOR *Trichomonas gallinae*

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Abstract: Trichomoniasis resulting from infection by *Trichomonas gallinae* was observed in 12 laboratory reared white-crowned pigeons (*Columba leucocephala*). A field survey of nestlings in the Florida Keys revealed a prevalence of 88% *T. gallinae* carriers but no evidence of trichomoniasis could be found among the wild birds.

INTRODUCTION

Members of the family Columbidae constitute the majority of the hosts for *Trichomonas gallinae*. Stabler¹ gives a list of hosts, and more recent records have been reported by others.^{2,3,4,5} Most of these records are of ground-feeding or primarily grain-eating species, which through their natural behavior patterns come into contact with other such species at some time during their lives, and thereby have occasion for cross transmission of *T. gallinae*.

MATERIALS AND METHODS

Twenty-four white-crowned pigeon squabs were collected in the Florida Keys during the 1969 nesting season and were hand reared on a pabulum-condensed milk-water diet until they fledged. Twelve of the fledglings were shipped to the Patuxent Wildlife Research Center in Laurel, Maryland, where they were housed indoors and gradually converted to a commercial pigeon grain diet.

In August 1970, squabs from 41 nests on three offshore islands in the Florida Keys were swabbed and checked for the presence of *T. gallinae*.

RESULTS

All 12 of the pigeons collected in 1969 were harboring *T. gallinae* at the time of their arrival at the Center and during the following 2 months 10 of the squabs died with visceral trichomoniasis, which had severely damaged their livers. The remaining two birds survived the winter in apparent good health but died the following spring from trichomoniasis. None of the 12 pigeons which remained in captivity in Florida until the following spring showed signs of trichomoniasis. These pigeons were fed a commercial pigeon pellet.

Trichomonads isolated from liver lesions in the laboratory reared white-crowned pigeons were placed into the mouths of non-immune *T. gallinae*-free homing pigeons. These died between 6 and 9 days following exposure. Since all of the laboratory reared white-crowned pigeons were fed with the same tube when younger, it was assumed that all the birds had the same strain(s) of *T. gallinae* in their crops.

Cultures in Diamond's medium¹ revealed that 36 (88%) of the nests sampled in 1970 contained squabs positive for *T. gallinae*. These squabs ranged in age from several hours to 18 days. No

oral lesions could be seen in any of the squabs, but trichomonads were present in their crops in sufficient numbers to be visible on direct microscopic examination; this was true even for squabs that were several hours old and had been fed only once.

Dead squabs (apparently drowned after falling from the nest) were observed in the mangrove swamps only occasionally, even though no scavengers or predators were present.

DISCUSSION

The death of the white-crowned pigeons reared at Patuxent was unexpected, considering that mortality or canker due to *T. gallinae* had never been reported from this species. The hand rearing, shipment, and conversion to a grain diet, in combination or singly, may have been responsible for their demise; but it is impossible at this time to make any conclusive statement as to why these birds succumbed and those in Florida in captivity survived. We reared two white-crowned pigeons with homing pigeon foster parents that were carriers of nonvirulent *T. gallinae*, and the two white-crowns appeared normal when they fledged.

Apparently, the commercial pigeon grain diet did not precipitate the observed condition.

The high prevalence (88% positive nests) of *T. gallinae* in the wild white-crowned pigeons was also surprising. Considering their arboreal fruit eating habits and that they acquire most of their water from the fruits they eat, it is highly unlikely that adult to adult transmission occurs except between mated individuals. This leaves only adult to young transmission to insure the parasite's survival.

It was concluded that a high percentage of wild white-crowned pigeons harbor *T. gallinae*, but that white-crowns are probably resistant to trichomoniasis under natural conditions even though the parasite is highly virulent for homing pigeons. This is borne out by the absence of canker in wild squabs and by the very low level of mortality observed among wild birds even when no predators or scavengers were present to remove dead birds.

A study of various columbid species that are isolated from other species by their behavioral patterns or by geography, might give some clue as to when their peculiar parasites originated and how they developed with their host species.

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LITERATURE CITED

1. DIAMOND, L. S. 1957. The establishment of various trichomonads of animals and man in axenic cultures. *J. Parasitol.* 43: 488-90.
2. HAYSE, F. A., and P. JAMES. 1964. *Trichomonas gallinae* isolated from the white-fronted dove (*Leptotila verreauxi*). *J. Parasitol.* 50: 89.
3. LOCKE, L. N., and W. H. KIEL. 1960. Isolation of *Trichomonas gallinae* from the white-winged dove *Zenaida a. asiatica*. *Proc. Helminthol. Soc. Wash. D.C.* 27: 128.
4. LOCKE, L. N., FRANCIS S. LOCKE, and D. H. REESE. 1961. Occurrence of *Trichomonas gallinae*, in the ground dove, *Columbigallina passerina* (L.). *J. Parasitol.* 47 (Sec. 1): 532.
5. LOCKE, L. N., and P. JAMES. 1962. Trichomonad canker in the Inca dove, *Scardafella inca* (Lesson). *J. Parasitol.* 48: 497.
6. STABLER, R. M. 1954. *Trichomonas gallinae*: A Review. *Exptl. Parasitol.* 3: 368-402.

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