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IMMUNOLOGIC STATUS OF MOURNING DOVES FOLLOWING AN EPIZOOTIC OF TRICHOMONIASIS

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Abstract: An epizootic of trichomoniasis in mourning doves at the Carolina Sandhills National Wildlife Refuge began in 1969 and continued into 1970. The disease was seen in 16% of the adults and 2% of the immatures in 1970, but only one immature bird out of 37 surveyed (3%) carried *Trichomonas gallinae*. Challenge infection of 33 doves from the epizootic area showed 85 percent to be resistant to trichomoniasis, compared to 69 percent resistance in doves from Maryland, where no epizootic has occurred for at least 3 years.

Plasma protein changes which occurred following challenge infection were identical in CSNWR and Maryland doves which showed evidence of disease. Of the doves which showed no signs of disease, those from the CSNWR exhibited no change in beta globulins, identical to the response in pigeons which survive an infection by *T. gallinae*, but they had some tissue invasion by the parasite.

INTRODUCTION

Reports over the past 20 years have implicated trichomoniasis as the cause of heavy mortality among mourning doves (*Zenaidura macroura*). Losses are not confined to immature doves which are highly susceptible, but also include many adults, and occasionally, mostly adults.[2] Reports of epizootics consist primarily of estimations of total mortality and descriptions of the environmental conditions existing during the period of heaviest losses. Little is known regarding adult-to-adult transmission of *Trichomonas gallinae* under natural conditions,[4,11] or of the physiologic condition of doves prior to and following the epizootics.

Outbreaks of trichomoniasis occurred during the summers of 1969 and 1970, at the Carolina Sandhills National Wildlife Refuge (CSNWR) near McBee, South Carolina. The disease was seen in 16% of 56 adults and 2% of 252 immatures trapped between June 1 and July 12, 1970. Prior to and after these dates no diseased doves were seen in approximately 400 doves examined. No epizootics occurred at other nearby banding stations, some within 16 km.

Two studies were undertaken to determien the effect of the epizootic on remaining doves. The first study was an on-location survey to determine the percentage of parasite carriers in the population, and the second study was to determine the resistance of the immature doves to trichomoniasis in the laboratory.

MATERIALS AND METHODS

Doves were examined in the field on July -7, 1970 by swabbing their throats and crops with cotton-tipped applicators. The adherent material was cultured in 5 ml Diamond's medium,[1] containing 0.5 ml deactivated pigeon plasma and 5000 units of penicillin-streptomycin, and then incubated at 37 C for 48 hours. Tubes inoculated in the field were transported under refrigeration to the Patuxent Wildlife Research Center (PWRC), Laurel, Maryland, for incubation.

Doves trapped at the CSNWR on September 7 were shipped by air to the PWRC where they were examined for *T. gallinea*. Blood sampels were taken for red cell counts and electrophoretic determinations of immunoglobulin levels. Uninfected immature doves were inoculated orally with a strain of *T. gallinae* known to be highly pathogenic for doves. A control group of doves trapped on the PWRC in September, where no outbreaks of trichomoniasis have been observed for several years, was similarly treated and served for comparison with the CSNWR doves. Inoculated doves of both groups were observed for 5 weeks. Any birds becoming moribund or exhibiting severe canker during this period were killed and blood samples were taken for comparison with their original red cell counts and immunoglobulin levels. Cultures were also prepared from liver and lung tissue to determine the extent of tissue invasion by the parasite. At the end of 5 weeks the remaining doves were killed and sampled for comparison with the doves that had shown signs of disease during the experimental period.

Plasma proteins were electrophoretically separated on Sepraphore III® strips for one hour at 400 v in 0.05M vernal buffer. Relative amounts of each protein band were determined on a Photovolt® densitometer and compared to the original samples.

RESULTS

One of the 37 immature mourning doves (3%) examined during the epizootic at the CSNWR proved positive by culture for *T. gallinae*. No infections were detected among 56 doves sent to the PWRC in September, 2 months following the first sampling. Of 18 adults and 24 immatures trapped at the PWRC in September, only one immature bird was infected. Red blood cell counts of doves trapped on the CSNWR and PWRC were essentially identical prior to experimental infection (Table 1).

Challenge infections with a highly pathogenic strain of *T. gallinae* were administered to 33 uninfected, immature, CSNWR doves and 23 uninfected, immature, PWRC doves. This procedure allowed us to estimate the percentage of birds susceptible to trichomoniasis.[7]

Five CSNWR doves (15%) and seven PWRC doves (31%) showed signs of trichomoniasis within 3 weeks after infection. Tubes inoculated with liver and lung tissue from these birds were all positive for *T. gallinae*, even when the lesions occurred only in the head sinuses or throat. Most diseased doves had lesions in their lungs which is consistent with previous findings.[5]

When doves survived 5 weeks without overt signs of disease they were killed and examined. None had lesions when necropsied and none of their organ cultures were positive for *T. gallinae*. However, all still carried the organism in their upper digestive tracts. Red blood cell counts were slightly depressed in doves exhibiting cankers.

In general, birds with trichomoniasis underwent increases in globulins and reductions in pre-albumin and albumin plasma protein fractions between the times of capture and death (Table 2). A slight rise in alpha and beta globulins was observed in the PWRC doves that showed no signs of trichomoniasis.

TABLE 1. Results of blood examination immediately after capture of mourning doves and after challenge infection with **T. gallinae.**

	Initial Mean Red Blood Cell Count x 10^6/mm^3	Mean Red Blood Cell Count After Challenge with *T. gallinae*
South Carolina Adults (31)	3.27	with lesions 3.06 (5)
Maryland Adults (18)	3.34	without lesions 3.31 (28)
South Carolina Immatures (33)	3.22	with lesions 2.86 (7)
Maryland Immatures (23)	3.22	without lesions 3.15 (16)

TABLE 2. Mean plasma protein levels of adult and immature mourning doves at time of capture, and of immatures following experimental infection with a canker-producing strain of **T. gallinae.**

State and Age	Pre-albumin	Albumin	Globulins		
			Alpha 1 & 2	Beta	Gamma
South Carolina					
Adults	23.5*	33.1	7.0	32.3	3.6
Immatures					
Preinfection	30.8	27.3	8.4	27.9	3.6
Lesions (died)	13.3	5.5	30.0	46.0	4.9
No lesions (killed)	36.3	30.6	8.8	20.0	4.9
Maryland					
Adults	32.3	37.7	7.7	19.2	2.8
Immatures					
Preinfection	29.7	32.9	8.9	23.9	4.1
Lesions (died)	16.4	9.2	17.6	48.3	8.2
No lesions (killed)	33.7	22.1	10.3	31.6	2.9

* Numbers represent percent of total protein.

DISCUSSION

Considering the high observed incidence of disease in the adult doves, it was surprising that so few immature birds were infected with *T. gallinae*. There are two possible explanations: (1) The immature doves examined may have been produced by uninfected parents and therefore never had been exposed to the parasite. Squabs produced by infected parents probably died in the nest or very shortly thereafter, leaving only uninfected squabs to fledge. (2) The immature doves may have been infected and spontaneously lost the organism, a phenomenon described in pigeons by Jacquette[3] and observed by the senior author in experimentally infected morning doves. The immature: adult ratio of 4.12:1 for doves trapped at the CSNWR in 1970, was substantially lower than in preceding years, supporting the former possibility. However, in 1969, when there were 12.61:1 immatures per adult in CSNWR-trapped doves, there was an epizootic of trichomoniasis that lasted longer than did the 1970 outbreak. On the basis of available data, we cannot implicate disease in nestling mortality in 1969 or 1970, although the possibility does exist.

If the doves were never infected, as suggested by the first possibility, then why were 85% resistant to trichomoniasis when challenged? Ordinarily, resistance develops only after recovery from an infection. Stabler[10] and Kocan[6] proved unequivocally that pigeons become immune to trichomoniasis following recovery from infection by any strain of *T. gallinae;* however, Sileo[9] questioned whether this occurs in mourning doves. If acquired immunity is not the explanation for the observed high level of resistance, an inherited or physiological resistance must be considered. The only extensive work on natural and acquired resistance to trichomoniasis has been done with pigeons *(Columba livia),* and although this information may not hold true for all species of columbids, a study of resistance to trichomoniasis in wild mourning doves did reveal that 82% were refractive to disease when experimentally infected.[7]

The lack of parasites in the doves does not preclude the acquired immunity hypothesis. Kocan (unpublished) found that pigeons retained their acquired immunity to trichomoniasis for as long as 18 months after being freed of *T. gallinae*, and also that a long-lasting immunity results in mourning doves which spontaneously lose their trichomonads. Work needs to be done with laboratory-reared, nonimmune mourning doves before the question of resistance can be resolved.

It is difficult to assess the effect of the epizootic on remaining individuals without knowing which type of resistance is present in the population. The greater resistance of the CSNWR doves (85%) as compared to PWRC doves (69%) may be due to a higher rate of exposure to the parasite as a direct result of the epizootic. If this occurred the loss due to trichomoniasis might tend to be offset by a higher percentage of reistant individuals remaining in the population. Assessment of the long-term effect of trichomoniasis on the population is complicated by the total lack of information concerning conditions which precipitate an epizootic. The two most likely possibilities are: (1) a highly virulent strain of the parasite is introduced into the population, and through adult-to-adult transmission reaches all nonimmune individuals; or (2) the birds undergo some physiological change that lowers their resistance and allows organisms that they are carrying, or to which they are subsequently exposed, to invade and produce lesions.

Changes of beta and gamma globulin levels in the doves that survived the challenge infection indicated that CSNWR doves were more resistant to the parasite than were PWRC doves (Table 2). Birds that succumbed to experimental infection had plasma protein changes similar to those described in nonimmune pigeons dying from visceral trichomoniasis.[4] Survivors from the CSNWR differed from survivors from the PWRC in showing no change in beta or gamma globulin fractions — the same response that occurs in immune pigeons having no tissue invasion by the parasite. The PWRC survivors showed plasma fraction changes similar to experimental pigeons in which the parasite invaded some organ but did not produce a fatal infection. Such changes are characterized by temporary increases in beta globulin followed by increases in gamma globulin. Increases in gamma globulin would have been expected to occur if the doves had been allowed to recover, but since they were killed while lesions were present, only large increases in beta globulin were evident.[4] Presumably the level of immunity was lower in the PWRC group, resulting in a greater response to the parasite.

In conclusion, immature doves trapped at the Carolina Sandhills NWR following the 1970 epizootic of trichomoniasis were highly resistant to trichomoniasis even though most of them were not carrying the parasite. Lack of parasite carriers prevented determination of the nature of the resistance. Based on previous work with pigeons, resistance was assumed to be the result of exposure to the parasite and acquisition of protective antibodies followed by spontaneous loss of the parasite. However, the possibility of inherited immunity cannot be disregarded.

The presence or absence of the parasite in surviving doves does not allow prediction concerning future epizootics. Surveys of infected doves identify individuals that are capable of transmitting *T. gallinae* at the time of examination, but give no indication of susceptibility to trichomoniasis.

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