INCIDENCE OF TRICHOMONAS GALLINAe IN MOURNING DOVE, ZENAIDURA MACROURA, POPULATIONS OF ARIZONA

by

Milo A. Straus

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1966
STATEMENT BY AUTHOR

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This thesis has been approved on the date shown below:

[Signature] 

ZYLE K. SOWLS 
Associate Professor of Wildlife Management 

May 17, 1966
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ABSTRACT

The reports of death occurring in mourning dove populations in Alabama and California due to *Trichomonas gallinae* emphasize the potential hazard of this disease to the mourning dove. This study attempted to determine the prevalence of the disease in Arizona. A total of 822 mourning doves were tested for the presence of *T. gallinae* over a one year period. Doves were live-trapped in modified Stoddard quail traps, a throat swab was taken of the captured dove and the swab was placed in a test tube containing five percent normal horse serum thioglycollate medium. The test tube containing the swab was incubated at 36.5°C for 72 hours then a sample was examined under the microscope using low power to determine the presence or absence of trichomonads.

The average incidence was 20 percent with a variation from a low of four percent to a high of 41 percent. Incidence decreased from September through the winter months, with an increase beginning in February and March. The disease incidence among immatures was significantly higher than among adults and disease incidence among males was significantly higher than among females.
INTRODUCTION

The proper management of a wild game species demands a knowledge of the basic principles of wildlife management. One of these principles concerns the role of population control. In such a category falls disease and the necessity for an understanding of the cause and effect of disease in a population. Many times a disease in wild populations is not immediately recognized unless it occurs in epizootic proportions. This is particularly true in bird populations. The relatively small size and protective plumage of a bird and the habit of seeking a sheltered, secluded place to die, make it difficult to discover sick or dead individuals before a large die-off occurs.

One disease known to occur in bird populations is trichomoniasis. In mourning doves, (Zenaidura macroura), the causative agent is Trichomonas gallinae. The reports of death occurring in mourning dove populations in Alabama and California due to T. gallinae emphasize the potential hazard of this disease to the mourning dove. In Arizona, the mourning dove is listed first among game birds bagged (Gallizioli 1961). The kill for 1959 was the highest of any upland game species and hunter interest, as shown by the percent of licensed hunters who shoot doves, was 40 percent.
Since the mourning dove is an important game species, the present study attempted to determine the effect of trichomoniasis on the population. The objectives were to determine the incidence among both adult and immature mourning doves in various areas of the state and determine whether mortality due to trichomoniasis had an appreciable effect on the population.
NOMENCLATURE

Trichomoniasis is a disease of the upper digestive tract of various birds, particularly columbids. It is caused by a flagellated protozoan which lives in head sinuses, mouth, throat, esophagus and other organs of birds. The correct designation for the pathogenic protozoan flagellate occurring in the upper digestive tracts of birds is *Trichomonas gallinae* (Stabler 1938). Rivolta first described *T. gallinae* in 1878 and referred to the organism as *Cercomonas gallinae* (Stabler 1938). Eventually it was placed in the genus *Trichomonas* since it was not a cercomonad. (Stabler 1938, 1954a, Jacquette 1948). The flagellate was commonly referred to as *T. columbae*. Stabler resurrected Rivolta's name *gallinae* and explained that it had priority over the other specific names. (Stabler 1938, Jacquette 1948). Stabler (1947) lists *T. hepatica* and *T. columbae* as described from the pigeon, *T. diversa* from the upper digestive tract of turkeys, and *T. halli* from the mouth of the chicken as synonyms for *T. gallinae*. 
Phylum - Protozoa

Subphylum - Mastigophora

Order - Trichomonaidida

Family - Trichomonadidae

Genus - *Trichomonas*

Species - *gallinae* (Noble and Noble 1961)
HISTORY

Trichomoniasis is perhaps the oldest disease to be recognized in birds and for several centuries was known as "canker" or "roup" in various breeds of pigeons. Years before the causative protozoan was known, falconers described the disease in their trained hawks, which were fed on domestic pigeons, and referred to the disease as "frounch" (Stabler 1954).

Although the causative organism was first reported and described by Rivolta in 1878 (Stabler 1938), there was no further record of the disease until Jowett reported it in South Africa in 1907. Jowett referred to the organism as Cercomonas hepaticum but his description was inadequate (Waller 1934). Von Rutz of Hungary gave a detailed description in 1913 and named the organism Trichomonas columbae. Waterman reported the infection in the West Indies in 1919 and Heelsbergen and Bos reported the disease in Holland in 1925 and 1932 respectively (Waller 1934).

Trichomoniasis was first reported in North America by Waller in 1934 who found the infection in some domestic pigeons in Minnesota (Waller 1934) (Stabler and Herman 1951). The same disease was first reported in the native mourning dove by Cauthen in a captive colony of pigeons (Columba livia), ring doves (Streptopelia risoria), and eastern
mourning doves (*Zenaithura macroura carolinensis*), on Long Island, New York. Most of the birds in the colony were infected (Cauthen 1934, Stabler and Herman 1951). Cauthen also reported the presence of the protozoan in a pigeon colony in Baltimore, Maryland and in sick pigeons from New Jersey (Cauthen 1936).

Stabler (1941b) inspected a total of 412 individual birds representing forty-four species, twenty-two families and eleven orders. The total number of birds positive for *Trichomonas gallinae* was 168, of which twelve were raptors and 156 were pigeons. Reports of the occurrence of the disease in mourning doves have been common in the last few years. McCullock (1950) reported the parasite from an eye infection in a dove in North Carolina. A study made by Stabler in Colorado showed that of 202 columbids examined, 77.5 percent of common pigeons (*Columba livia*), 17.3 percent of mourning doves (*Zenaithura macroura carolinensis*), and 19.1 percent of band-tailed pigeons (*Columba fasciata*) were infected (Stabler 1950). A study of 109 band-tailed pigeons, 100 common pigeons, and 100 western mourning doves in Colorado revealed that 19.3 percent of the band-tailed pigeons, 23.0 percent of the mourning doves, and 69.0 of the common pigeons tested were positive for *T. gallinae* (Stabler 1951a). The first published report of *T. gallinae* in Kentucky was in 1950 (Russel 1951).
Locke and Herman (1961), reported that during a ten year period (1950-1960), in Maryland, the incidence among series of mourning doves shot or trapped varied from 0 to 12.5 percent. During this period, 44 sick doves were sent to the laboratory and 25 tested positive for *T. gallinaceae*.

All 51 eastern white-winged doves (*Zenaida asiatica*), tested by Stabler in 1961 in Texas, were positive for *T. gallinaceae* (Stabler 1961). Thirty-two of 42 white winged doves tested in Arizona in the summer of 1963 were positive. Twenty-three white-winged dove nestlings were examined at this time and 21 were infected. (Toepfer, Locke and Blankenship 1966).
MORPHOLOGY

The morphology of *Trichomonas gallinae* has been described by several investigators with Stabler (1941a, 1942, 1954a), contributing some of the most significant work regarding its structure and biology. The organism is in the family Trichomonadidae which is characterized by having several anterior flagella, a pelta lying at the anterior margin of the body, an undulating membrane, a costa extending along the base of the membrane, an axostyle extending through the center of the body often projecting from the posterior end, and one nucleus (Noble and Noble 1961). This flagellate belongs to the genus *Trichomonas*, a name used when it was believed that the members of the genus possessed three anterior flagella. Today, this group is known to possess four anterior flagella, but the generic name *Trichomonas* still remains (Noble and Noble 1961).

*Trichomonas gallinae* has the shape of an elongate ellipsoid, being narrowed at both ends, the widest point at or slightly posterior to the middle. Stabler (1941a) gives the average size of the animal as $10.5\mu \times 5.24\mu$. The mean length to width ratio is about $1.84\mu$, with the body being quite plastic and able to squeeze between two particles separated by a distance not exceeding the diameter of the axostyle (Abraham and Honigberg 1964).
The anterior flagella, typically four in number, the axostyle, the parabasal complex, the costa, the marginal filament, and the undulating membrane arise at the basal granule at the anterior end of the animal (Stabler 1954a). The flagella, in many permanent preparations, appear to be arranged in two pairs with one pair being somewhat longer than the other. All the flagella are uniform in diameter and end in little knobs or rods (Abraham and Honigberg 1964). Stabler (1954a) gives the average length of the flagella as 9.9μ while Abraham and Honigberg (1964) list the average length as 10.1μ (S-0.78), 10.0μ (S=1.30), and 11.0μ (S=0.98) for the three strains examined.

The nucleus is located in the anterior portion of the flagellate, is oval in shape and usually contains a single nucleolus. Fifty nuclei of three different strains of T. gallinae were measured by Abraham and Honigberg (1964), and the three strains averaged 2.4μ (S-0.29) by 1.3μ (S-0.22), 2.4μ (S=0.32) by 1.2μ (S=0.24), and 2.4μ (S=0.35) by 1.3μ (S=0.26). The axostyle passes ventral to the nucleus and continues to the posterior end of the body, where it protrudes for a short distance. The anterior segment is flattened into a spatulate capitulum which is closely applied to the left ventral surface of the nucleus (Abraham and Honigberg 1964).
The parabasal complex consists of a parabasal body and a parabasal fiber, and is located dorsally and somewhat to the right of the nucleus. The parabasal fiber passes dorsally to the nucleus and towards the posterior region of the animal. Stabler (1941a, 1954a) gives 3.6μ as the length of the parabasal body which lies along the dorsal side of the parabasal fibril. The undulating membrane and the costa originate posterior and dorsal to the anterior flagella. The costa has a length two-thirds to three-quarters that of the organism and passes along the dorsal margin of the undulating membrane (Stabler 1954a). The outer edge of the undulating membrane is the marginal filament which extends down the dorsal side of the body and ends at the posterior tip of the costa (Stabler 1941a, 1954a). The granular material in the cytoplasm is mainly concentrated in the regions of the axostyle, parabasal fibril and costa. Abraham and Honigberg (1964) observed two distinct rows of paracostal granules extending nearly the entire length of the supporting rod, and two rows of granules accompanying the intracytoplasmic segment of the axostyle, being somewhat smaller than the paracostal granules and less constant in their distribution.
Figure 1. Line drawings of *Trichomonas gallinae*.

A. Anatomical features; B. Left view;
C. Right view.
PATHOGENESIS

*Trichomonas gallinae* is found in the upper digestive tract and associated tissues of the bird. Normally the protozoan is not found posterior to the gizzard in the digestive tract, but secondary involvement of the tissues and organs of the body cavity may result (Stabler 1947). *T. gallinae* is capable by itself of causing the symptoms and disease, while associated bacteria may or may not influence the course of the disease (Stabler 1947). Stabler (1947) has demonstrated that *T. gallinae* is capable by itself of causing the symptoms and disease, while associated bacteria do not influence the course of the disease. It has been further shown that in some cases a single trichomonad can cause trichomoniasis (Stabler and Kihura 1954).

A bird suffering from trichomoniasis may exhibit one or more of a variety of conditions. Typically, the bird will show some necrosis somewhere in the body (Stabler 1947). Mortality and/or the symptoms involved depend to a large extent on the virulence of the strain of trichomonad involved and on the degree of immunity developed by a previous non-lethal infection (Stabler 1951). Research involving *T. gallinae* infection has demonstrated that a marked variation in strain does exist. Stabler (1948a, 1951) has isolated one strain which produces severe lesions in the viscera, particularly the liver, and causes only moderate necrosis in the mouth. Other strains
never invade the body cavity, but, produce severe lesions in
the mouth and head sinuses, many times invading the brain and
orbital regions. Depending on the organism, strain, dose, and
tissue involved, an affected bird may recover or die from the
disease. In a chronic infection, the bird will appear healthy,
the visceras will be free of involvement and the trichomonads
will be present mainly in the mouth, pharynx, esophagus and
crop (Stabler and Herman 1951). Such a bird can probably be
considered a "carrier" of the disease.

The lesions of *T. gallinae* typically appear as firm,
yellow, areas of caseous necrosis, ("yellow buttons"), on the
oral mucosa near the nasal cleft or to the rear of the palatal
flaps. The lesions grow rapidly in size and become firm,
cheesy lumps, often blocking the passage of food and hinder­
ing breathing or swallowing. The bird begins to lose weight
rapidly, becomes weak and listless, then dies in about ten
days. Haugen and Keeler (1952), observed that in some birds
there is severe diarrhea. If the infection invades the body
cavity, the liver may be completely covered with necrosis or
may exhibit only a few focal necrotic areas. The pancreas,
small intestine, air sacs, lungs, and heart all may be involved
(Stabler 1947). Herman (1953) found that in some instances
the lesions may extend up into the skull and periorbital
tissue, causing blindness.

Stabler (1947) experimentally infected 30 pigeon
squabs (*Columba livia*) and his work presents one of the best
clinical examples of trichomoniasis:
"Invariably, the first sign of infection was a small, yellowish area (or areas) which appeared somewhere in the oral mucosa. Usually, there was one initial lesion which in most cases appeared in the mid-line between the pair of soft palatal flaps located in the pharyngeal region of the mouth. These first lesions appeared three to 14 days (average 7.4 days) after infection. They generally increased rapidly in size and number, frequently reaching such proportions that the esophagus or trachea was blocked completely. The tissues of the roof of the mouth were extensively invaded, involving the production of large, caseous masses which sometimes even included the bones of the floor of the skull. In one bird these lesions extended to the tissues about the eyes, and trichomonads were recovered in fluid emerging therefrom."

"Of all the alimentary tract, the pharyngeal region received the heaviest attack. Here the soft palate, including the flaps to the rear, was frequently a huge mass of caseation necrosis which extended forward and to the back in great lumps of yellow necrotic material completely covering all surfaces. The odor from a mouth of this kind was most fetid."

"Whereas the esophagus was occasionally simply lined with caseation, this structure was not so generally involved as the mouth and pharynx. The lesions here frequently presented the appearance of small mounds, from the centers of which extended small spurs. Sometimes there was
no gross evidence of disturbance. The crop, too, was commonly free of caseation, but when present the lesions were usually at the base, just anterior to the proventriculus. This latter structure was attacked only on rare occasions, but at least once it was very heavily involved. The gizzard and the small and large intestines were never seen to bear lesions internally."

"Twenty-one birds died of trichomoniasis. At autopsy, the most striking visceral changes were noted in the liver. Only one dying pigeon was seen to have a perfectly clean liver, and cultures from it were negative for Trichomonas. Another bird, killed early for class purposes, likewise had no gross liver pathology, through T. gallinae grew out in culture. The lesions were solid, caseous affairs, as were those of the oral cavity, and ranged from a few large areas which extended deep into the organ, to livers in which the caseation actually represented ninety per cent of the bulk. In some infections the areas were small but extremely numerous. In others both large and small ones were intermingled. The extent to which both lobes of the liver were involved was quite marked."

Stabler (1947) further notes that the other viscera were involved mainly as contact extensions of the hepatic lesions. The lungs were involved in only one case, and in four cases the ventricles of the heart showed focal necrosis. At death the birds appeared severely wasted and weak. They stood huddled, with feathers ruffled and frequently toppled
over when forced to walk. There was an extensive amount of greenish fluid accumulated in the crop. The average bird died on the tenth day following the introduction of the parasite. Unlike Stabler's results, the birds examined during the present study showed no lesions on the viscera. Undoubtedly this was due to strain variation from the type studied by Stabler.
MATERIALS AND METHODS

Culture Techniques

Data were collected almost exclusively from mourning doves live trapped in modified Stoddard quail traps (Stoddard 1931). A throat swab was taken of the captured dove and the swab placed in a test tube containing culture medium.

Stabler (1951a) examined birds by removing material from the pharyngeal portion of the mouth with forceps, being sure to obtain material from behind the palatal flaps. He immediately examined this material in saline with a microscope, and found the method to be superior to the culture method. Diamond (1954) found that positive results were obtained frequently by culture when there were no trichomonads observed by direct smear examination. I found the culture method to be the most practical and better results were obtained than with the direct smear examination method advocated by Stabler (1951a).

The throat swabs were obtained by the following procedure:

1. The bird was placed on it's back in one hand.

2. The thumb and index finger were used to hold the bird's mouth open while the bird was examined for caseous lesions.
3. A cotton swab was moistened in sterile saline solution, rotated against the back of the bird's mouth, and then removed.

4. The cap was removed from a test tube containing culture medium, the entire swab was dropped into the medium, and the cap was replaced.

Fluid thioglycollate medium containing five percent normal horse serum was used as the culture media (Abraham and Honigberg 1964). Solid thioglycollate medium (29.8 grams) was added to 1000 ml. of cold distilled water which was then heated to boiling to dissolve the thioglycollate. The media was then autoclaved at 15 pounds pressure, 121°C for 15 minutes, then allowed to cool to room temperature. When the media had cooled, sterile horse serum was added to make the five percent solution. One ampoule (1,000,000 units) of penicillin G and one ampoule of streptomycin sulfate (1 gm.) were added to the media to control the bacterial growth.

The media was kept in an aspirator jar fitted with a length of rubber tubing containing a hollow glass rod at the tip. A clasp was used to clamp off the rubber tubing. This container facilitated an easy transfer of the medium to sterile test tubes with minimum danger of contamination. Ten ml. of media were transferred to each test tube. The sterile transfer of media from container to test tube was accomplished by flaming both the tube and the glass rod attached to the aspirator jar. The medium was stored in
the dark at room temperature until used. Thioglycollate medium is useable as long as no more than the top one-third of the media turns pink.

The test tube containing the swab was then incubated at 36.5 C for 72 hours. After incubation a sample was removed from the top and bottom of the tube through a pipette, and placed on a microscope slide. The slide was then examined under the microscope, using low power, and the presence or absence of trichomonads was determined. I learned that trichomonads, if present, are usually found at the bottom, however, at times they may be present near the top of the media and not at the bottom. For this reason it is best to take a sample from both areas.

The best growth occurs, and the trichomonads are most numerous, after cultures have been incubated 48 to 72 hours. After 72 hours the number of trichomonads able to live in the media begins to decrease. For this reason it is desirable to subculture to keep the cultures viable and maintain a stock culture. Subculturing is accomplished by using a pipette to remove 0.2 c.c. of material from the tube containing the culture and placing the material in a tube containing fresh media. Both the pipette and the edge of the fresh media tube should be flamed to avoid contamination. Subculturing should be repeated every 72 hours to maintain the stock culture. A method used by the Animal Pathology
Department of the University of Arizona for partially decontaminating cultures has worked successfully. An 8 mm. I.D. or larger glass tube six to eight inches long is bent into a "V" and stood upright. The tube is filled with media and the culture material is placed in one arm of the "V" tube. The culture is incubated and when it is necessary to subculture the material is drawn from the opposite arm of the "V" tube. The pipette does not have to pass through the layer of contaminants and the material can be subcultured relatively free of bacteria and mold.

Sampling Areas

Four areas were selected for the most intensive sampling. These were (1) University of Arizona Campbell Avenue Farm in Tucson, Pima County, (2) Continental feed lot, Pima County, (3) Arlington Feed lot, Maricopa County, and (4) Dobson feed lot, Maricopa County. Although not tested extensively, a sample was taken from Nogales, Santa Cruz county in the winter of 1966. These areas were selected as they had some of the largest concentrations of doves in the state and afforded a good cross section of both resident birds and breeding migrants. The areas are also indicative of the dove populations throughout the state as there is a flow of migrants through the areas during the year. The Santa Cruz Valley, one of the major dove areas of the state is encompassed within the boundaries formed by the four locations. All birds tested were banded.
In addition to the live-trapped birds used several throat swab samples also were collected from hunter-killed birds brought into the hunter check station operated by the Arizona Game and Fish Department at Arlington during the opening days of the dove hunt in September.

The research sampling centered on mourning doves, but a number of white-winged doves were also tested.
RESULTS

Research was conducted from October 1964 to March 1966. A total of 763 live trapped birds, and fifty-nine hunter killed birds were tested. The data shows that the average incidence of Trichomoniasis over this period was 20 percent. (Table 1). Fig. 2 indicates that the incidence varied considerably throughout the year, from a low of four percent in January, 1965, to a high of 41 percent in September of 1965. The highest incidence occurred during the summer months of June to September with a sharp decrease from September through the winter months, and an increase beginning again in February and March.

After September it was difficult to distinguish juveniles from adults so data for immatures were restricted to the summer months. The incidence of the disease among immatures was higher than among the adult population from June to September (Fig. 3). Analysis by chi-square method showed a significant difference at the 5 percent level between incidence in immatures and adults ($X^2=4.99$) (Table 3). Although the incidence among adults was much lower during June and July, it increased sharply to a point comparable to the incidence among immatures at the end of the summer. The slight decrease in incidence in the adult population between June and July was apparently die to sample size rather than a fluctuation in incidence.
Table 1. Incidence of Trichomoniasis in total sample, Oct. 1964-Mar. 1966

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Table 2. Incidence of Trichomoniasis in samples of adults and immatures June-Sept.

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<td>257</td>
<td>63</td>
<td>24</td>
</tr>
<tr>
<td>August</td>
<td>60</td>
<td>16</td>
<td>26</td>
<td>41</td>
<td>10</td>
<td>24</td>
</tr>
<tr>
<td>September</td>
<td>25</td>
<td>10</td>
<td>40</td>
<td>45</td>
<td>19</td>
<td>42</td>
</tr>
</tbody>
</table>
Table 3. $X^2$ Values of population samples, at .05 significance level.

<table>
<thead>
<tr>
<th></th>
<th>+</th>
<th>-</th>
<th>Total</th>
<th>Percent</th>
</tr>
</thead>
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<tr>
<td>Immature</td>
<td>82</td>
<td>275</td>
<td>362</td>
<td>24</td>
</tr>
<tr>
<td>Adult</td>
<td>41</td>
<td>207</td>
<td>248</td>
<td>16</td>
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<td></td>
<td>$x^2 = 4.99$ (significant)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>34</td>
<td>187</td>
<td>221</td>
<td>15</td>
</tr>
<tr>
<td>Female</td>
<td>15</td>
<td>164</td>
<td>179</td>
<td>8</td>
</tr>
<tr>
<td></td>
<td>$x^2 = 5.70$ (significant)</td>
<td></td>
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<td></td>
</tr>
<tr>
<td>Campbell</td>
<td>48</td>
<td>138</td>
<td>186</td>
<td>26</td>
</tr>
<tr>
<td>Dobson</td>
<td>13</td>
<td>74</td>
<td>87</td>
<td>15</td>
</tr>
<tr>
<td></td>
<td>$x^2 = 4.031$ (significant)</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Campbell</td>
<td>48</td>
<td>138</td>
<td>186</td>
<td>26</td>
</tr>
<tr>
<td>Arlington</td>
<td>14</td>
<td>67</td>
<td>81</td>
<td>17</td>
</tr>
<tr>
<td></td>
<td>$x^2 = 2.299$ (not significant)</td>
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<td></td>
</tr>
<tr>
<td>Campbell</td>
<td>48</td>
<td>138</td>
<td>186</td>
<td>26</td>
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<tr>
<td>Continental</td>
<td>34</td>
<td>148</td>
<td>182</td>
<td>19</td>
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<td></td>
<td>$x^2 = 0.176$ (not significant)</td>
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<tr>
<td>Immature</td>
<td>48</td>
<td>137</td>
<td>185</td>
<td>26</td>
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<tr>
<td>Campbell</td>
<td>39</td>
<td>138</td>
<td>177</td>
<td>22</td>
</tr>
<tr>
<td></td>
<td>$x^2 = 0.758$ (not significant)</td>
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</table>
Figure 2. Percent incidence of trichomoniasis in mourning dove population sample (adult and immature) from October 1964 to March 1966.
Figure 3. Percent incidence of trichomoniasis in sample of adult and immature mourning doves during the summer months.
More males than females were infected and a chi-square analysis (Table 3) showed there was a significant difference at the 5 percent level between males and females in the degree of infection by T. gallinae \((X^2=5.70)\). The four locations where the majority of the sampling was done did not show considerable variation in incidence (Fig. 4). Although the incidence at the Campbell Avenue location was higher (Table 4), there was a significant difference only between Campbell Avenue and Dobson feed lot (Table 3). There was no significant difference in the incidence of T. gallinae between immatures at the four locations (Table 3).

Fifteen of the 763 live-trapped doves examined exhibited lesions. Ten of the birds were adults. Several of these birds were kept under observation, and all succumbed to the disease. The fulminating case appeared more prevalent in adults than in immatures.

Twenty-five white-winged doves, ten live trapped and 15 hunter-killed birds, were examined. All of the live trapped birds and 14 of the hunter-killed birds were positive for Trichomonas gallinae. None of the 25 white-wings exhibited lesions or showed any symptoms or signs of disease.
Figure 4. Incidence of trichomoniasis at four sampling areas June to July.
Table 4. Incidence of Trichomoniasis in sample at four locations Oct. 1964-Mar. 1966

<table>
<thead>
<tr>
<th>Location</th>
<th>Total</th>
<th>Number Positive</th>
<th>Percent Positive</th>
</tr>
</thead>
<tbody>
<tr>
<td>Continental</td>
<td>202</td>
<td>35</td>
<td>17</td>
</tr>
<tr>
<td>Arlington</td>
<td>223</td>
<td>36</td>
<td>16</td>
</tr>
<tr>
<td>Dobson</td>
<td>87</td>
<td>13</td>
<td>15</td>
</tr>
<tr>
<td>Campbell</td>
<td>189</td>
<td>49</td>
<td>26</td>
</tr>
</tbody>
</table>
DISCUSSION

Establishing incidence of a disease in a wild population is usually extremely difficult. A sample representative of the occurrence of the disease in the population must be obtained. It may be very likely that diseased birds are less susceptible to being trapped because of their restricted activity. Obviously, sick individuals may be observed, however, in all probability, this is only a small portion of the diseased population. Sampling does not take into consideration birds that have recovered and no longer harbor the organism, or the fact that incidence rates may vary from time to time. For these reasons, incidence figures based on population samples give only the minimum incidence or occurrence of the disease.

The results of the present survey show that trichomoniasis is present in the Arizona dove population. These results are consistent with data obtained by other research workers. Haugen (1952) in studying the trichomoniasis outbreak in Alabama, found losses occurred mainly through June, July, and August, were few during the winter months, but started again in March. I found that the incidence followed the same pattern. The peak occurs at a time when the population is highest. Population figures have been swollen by migrants, and juveniles are being added to the population as the hatching season is at its peak.
Haugen (1952) dealt mainly with dead or dying birds that showed lesions and found that the majority of the birds were adults. Although I found only a small number of birds that exhibited lesions, the largest number were adults. Haugen (1952) presents three possibilities for the low percentage of young birds dying from trichomoniasis:

1. The juveniles may not have been as susceptible as adults.

2. It was likely that the immatures died in the nest and were never found.

3. Possibly breeding was curtailed in birds that recovered from an attack of trichomoniasis.

I discovered that the incidence among immatures in Arizona is higher than among adults although more adults succumb to the disease.

It has been established that races of *Trichomonas gallinace* vary in virulence (Stabler 1948a, 1948b, Abraham and Honigberg 1964). Stabler (1948b) has also shown that a nonlethal infection with *T. gallinace* produces an immunity which completely protects the host against death from one of the virulent strains. Birds that harbor a virulent strain of *T. gallinace* superimposed on a protecting, non-lethal strain, may pass the disease on to clean birds with variable results (Stabler and Herman 1951). The recipient may die from the disease, or show no symptoms and become a carrier. The recipient in turn may pass the disease to another bird with the same results, the other bird will die or become a
carrier. It is obvious the effect this disease passage would have on the population. Until the squab is able to forage for itself, it is fed entirely on "pigeon milk" produced by the adult. Therefore, it is impossible for the young bird to escape exposure to whatever is in the parent's mouth. If the parent bird has the disease, invariably the squab will become infected. This behavior would explain the higher incidence in immatures. An adult bird could have the disease, pass it to the young, then recover and no longer harbor the organism. If the adult was captured and tested after this, the test would show negative although the adult had infected the juvenile, thus increasing the incidence in immatures. The higher incidence of death among adults could occur if a bird receiving a virulent strain as its first infection either died from the disease or was so weakened that it became easy prey for predators. If this were the case the bird probably would not get a chance to breed and pass the disease to the young. Haugen (1952) found that a number of females that died of trichomoniasis during the nesting season showed no signs of egg development, and the testes of several males were not in breeding size. These facts although inconclusive indicate that the chances are less, although not eliminated, for a juvenile bird to get a lethal infection. It seems improbable that the young are less susceptible to the disease initially. However, if the adult bird passes a non-lethal strain to the juvenile, subsequent
immunity to a lethal strain could result. Thus, although there would be a higher incidence, there would be a lower mortality rate among immature birds.

The incidence among males was significantly higher than among females, especially during the summer. This might be due to the different social behavior and nesting habits of the sexes. The female is on the nest during the evening, through the night, and during the early morning, while the male is on the nest during the middle of the day. When the female is off the nest to feed and water, birds normally are not concentrated, hence there is less contact and less chance to contract the disease. The male however, is more inclined to flocking, and is off the nest during the morning and evening, a time when the birds tend to flock together for feeding. Since the male is in close contact with other birds, it has more chance to contract the infection, thus allowing a higher incidence. This higher incidence, then is possibly due to the male's behavior rather than his susceptibility to the disease.

The incidence at the various locations of the study did not vary considerably (Table 3). The incidence was highest at the Campbell Avenue site because of the large percentage of immatures captured. Sample size, and the large number of adult birds at Dobson feed lot account for the significant difference between the two areas.
The incidence was highest near the end of the summer after the peak of the breeding season, when the population was highest because of the addition of immatures. The months of June and July are the peak hatching period and are the warmest and driest months of the year. Because of the increased water need due to the heat, birds become more concentrated around watering sites. Increased concentration means increased possibility of infection. Since the majority of the birds will be nesting, increased infection in adults will mean increased infection in nestlings, thus causing high incidence figures through August and September. Rosen (1959, 1960) in studying outbreaks of trichomoniasis in California, felt that artificial feeding of birds with backyard feeders contributed to outbreaks of the disease because feeding in this manner encouraged dove concentrations. The same effect will occur when doves are concentrated at watering sites. Stabler (1947) states that shallow water pools, highly charged with organic matter, could easily prove satisfactory for the survival of *T. gallinae* until ingested by a susceptible host. Haugen and Keeler (1952) also felt that shortage of drinking areas had an effect on incidence. Towards late summer there will be birds in the flocks, immatures and adults, that have acquired immunity and survived. These birds serve as carriers and can infect other individuals at the watering sites.
The reason for the sharp decline in observed incidence throughout the winter is not clear. After September many of the birds have migrated. Gallizioli (1961) estimated the winter residents at only 20 percent of the summer population. It is possible that birds north of Arizona are less apt to have trichomoniasis and once many of the Arizona birds have gone south the incidence decreases. Since the weather is cooler, especially at night, it would be impossible for T. gallinace to live in the water pools. Certain carrier birds are still present and test positive, but they are only a minority of the population.

Only a minority (15) of the birds tested exhibited lesions or soars due to T. gallinace infection. Therefore the strain of parasite prevalent in Arizona's mourning dove population may not be extremely virulent. I could not tell that the population was appreciably hurt by the presence of the disease. However, there is no reason to assume that a lethal strain is not present in the population, although it may not be particularly prevalent now.
We know too little about trichomoniasis to propose definite measures for controlling the disease in wild birds. The easiest method of controlling game species is the regulation of the population by hunting, but this is inadequate in the control of disease.

Various methods of control have proven successful in caged populations. Cu SO\textsubscript{4} in the drinking water is a fairly successful treatment (Biester and Schwarte 1954). Enheptin (2-amino - 5-nitrothiazole), was tested by Stabler and Mellentin (1953). The drug was placed in gelatin capsules and given orally to infected pigeons. The drug was effective in this form but not in a soluble type. Furazolidone (N - 5-nitro - 2 furfurylidene - 3 amino - 2 orazolidone) was also tested by Stabler (1957). It was given in gelatin capsules; however, effective levels of the drug were accompanied by loss of weight, convulsions, and sometimes death.

Until a method of administering drugs to large numbers of wild doves is found, no treatment will be highly successful in wild populations. The only effective method would be to irradiate the carriers, for treating sick birds will not prevent reinfection. At the present time there is
no successful method for treating trichomoniasis in Arizona's mourning dove population. The drugs tested were successful in destroying the *T. gallinace* organisms to such an extent that infected birds no longer harbored the parasite.
CONCLUSION

The figures on the incidence of trichomoniasis in Arizona's dove populations indicate the importance *Trichomonas gallinae* could play in dove management. Although only a minority of birds exhibited caseous lesions or were seriously affected by the parasite this does not imply that the disease could not develop in epizootic proportions as occurred in Alabama. Stabler (Stabler and Herman 1951) holds the opinion, although it can never be proven, that *T. gallinae* played an important role in the extermination of the passenger pigeon (*Eotopistes migratorius*). This idea is supported by Haugen (1952) who states that the Alabama trichomoniasis outbreak occurred in the same area where a large flock of passenger pigeons disappeared in 1881.

The bird, particularly columbid birds, is the only natural host for *T. gallinae*. Stabler (1941b) strongly feels that the common pigeon is an important source in the spread of *T. gallinae*. I do not doubt that the pigeon can and does serve as a reservoir, however, it is probably not as important in its effect on Arizona's mourning dove population as it is on dove populations in other areas of the country. The high incidence of *T. gallinae* present in the white-winged dove population would seem to have more far reaching effects on mourning doves in Arizona. The mourning doves in the wild
come in contact with, and mingle with, the white-winged doves far more frequently than with common pigeons. Therefore, the white-winged doves constitute an important source of infection, although admittedly, not the only one.
SUMMARY

(1) A total of 822 mourning doves, adults and immatures, were tested for the presence of Trichomonas gallinace over a one year period. The average incidence was 20 percent with a variation from a low of four percent to a high of 41 percent. This highest incidence occurred during the summer months of June and September. Incidence decreased from September through the winter months, with an increase beginning in February and March.

(2) The disease incidence among immatures was significantly higher than among adults ($X^2=4.99$). Incidence among adults reached a point comparable to that in immatures towards the end of the summer.

(3) Incidence among males was significantly higher than among females ($X^2=5.70$).

(4) There was not a significant variation in the incidence of trichomoniasis among the four locations involved in the majority of the testing.

(5) Fifteen of the birds examined showed lesions; ten of these were adults.

(6) T. gallinace was present in 24 of 25 white-winged doves examined.
LITERATURE CITED


Rosen, Mert N. 1960. Trichomoniasis or canker of doves. California Department of Fish and Game. Game Management leaflet No. 2.


Stabler, Robert M. 1938. Trichomonas gallinae (Rivolta 1878), the correct name for the flagellate in the mouth, crop, and liver of the pigeon. J. Parasit. 24:553-554.


