

## A BRIEF REVIEW OF AVIAN TRICHOMONASIS

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### INTRODUCTION

*Trichomonas gallinae* is a common inhabitant of the upper digestive tract of birds, particularly pigeons and doves. It may spread from the upper digestive tract to the cranium, thorax, and abdomen. In these sites, it commonly produces a yellowish, firm, caseous response on the part of the host, which condition is referred to as canker.

The organism was discovered in the upper digestive tract of a pigeon squab by Rivolta in 1878. He named the organism *Cercomonas gallinae*. The correct designation for this pathogenic flagellate of bird is *Trichomonas gallinae* (Rivolta, 1878) Stables, 1938. Stables (1938) pointed out that the first name for this organism was *Cercomonas gallinae*, as proposed by Rivolta. Since it is not a cercomonad, it has been eventually placed in the correct genus *Trichomonas*.

Stabler (1954) indicated that with one possible exception (*Trichomonas gallinarum*), any trichomonad found in the digestive tract anterior to the gizzard, in the tissue of the head, thorax, or abdomen of any bird is *Trichomonas gallinae*.

*Trichomonas gallinae* has been reported from various parts of the world. It was found in pigeons from South Africa, Hungary, Germany, Japan, and the United States. Cankerous turkeys are observed in Connecticut and the middle west, cankerous chickens in California.

### MORPHOLOGY OF *TRICHOMONAS GALLINAE*

The following description (Figure 1) of *Trichomonas* is based on Stabler (1941, 1954). The body is roughly pear-shaped. Measurement of this fixed and stained flagellate from six species of host (pigeon, gold eagle, sparrow, red-shouldered, red-tailed, and Cooper's hawks) averages  $10.5 \times 5.2$  microns ranging in length from 6.2 to 18.9 microns and in width from 2.3 to 8.5 microns.

The anterior flagella, marginal filament, costa, axostyle, and parabasal apparatus arise from the blephaloplast which is located at the extreme anterior end of the animal. There are four flagella in the nondividing organism. These flagella range from 7.7 to 13.1 microns in length, averaging 9.9 microns.

The parabasal apparatus is composed of a parabasal body and a parabasal fibril. From its origin, the parabasal fibril passes dorsal to the nucleus and sweeps to the posterior region of the organism. The parabasal body lies along the dorsal side of

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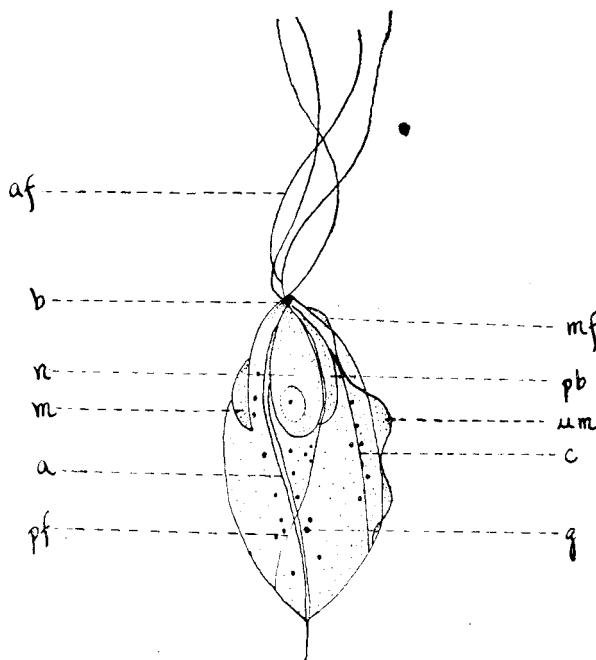


Figure 1. Semidiagrammatic representation of *Trichomonas gallinae*.  
 a=axostyle; af=anterior flagellum; b=blepharoplast;  
 c=costa; g=cytoplasmic granules; m=mouth;  
 mf=marginal filament; n=nucleus; pb=parbasal body;  
 pf=parbasal fibril; um=undulating membrane.  
 (After Stabler, J. of Morphology, 1941).

the parbasal fibril in the region of the nucleus. It averages 3.6 microns in length and is sausage shaped.

The costa passes along the dorsal margin of the flagellate at the base of the undulating membrane. Its length is two-thirds to three-quarters of the body. The marginal filament may occasionally be seen as a double structure, serving as the outer edge of the undulating membrane, and ending at the posterior tip of the costa. It does not continue as a free posterior flagellum.

Passing the ventral aspect of the nucleus, the narrow axostyle continues to the posterior end of the body where it protrudes for a short distance.

The nucleus, usually located in the anterior portion of the animal, averages 4.6 microns in length. It is oval in shape and possesses one, sometimes two, endosomes, and numerous small scattered granules.

The mouth, though present, is usually closed and therefore quite difficult to demonstrate. It lies in the anterior end and extends along the ventral side of the axostyle opposite the nucleus.

The multiplication of this organism is by means of binary fission. Autotomy and somatella formation are also found in *Trichomonas gallinae* (Figure 2).

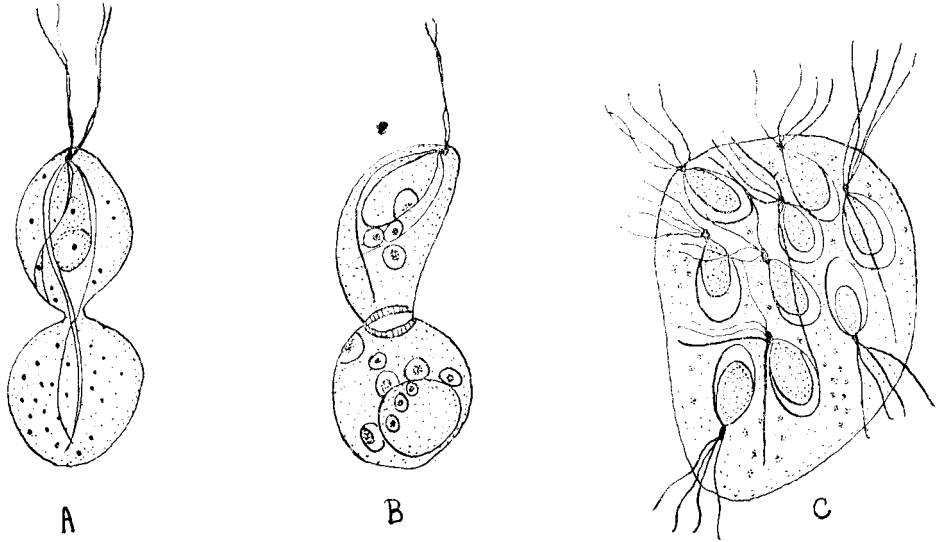


Figure 2. Autotomy and somatella of *Trichomonas gallinae*.

A. Red-tailed hawk: autotomy nearly complete. B. Red-tailed hawk: note more deeply staining areas of the differentiation in the region of cleft. C. Domestic pigeon: somatella with nine karyomastigonts. (after Stabler, J. of Morphology, 1941).

### NATURAL HOSTS OF *TRICHOMONAS GALLINAE*

The bird is the only natural host of this parasite. Mammals, as well as various birds, have been experimentally infected. Stabler (1954) mentioned that the primary host of *Trichomonas gallinae* was the domestic or common pigeon (*Columba livia*). The organism also occurs in many other species of birds including hawks and falcons which feed on pigeons.

Its natural hosts, besides the pigeon, include the mourning dove, India dove, wood pigeon, band-tailed pigeon, ring dove, white winged dove, turkey, chicken, Copper's hawk, golden eagle, duck hawk, Java sparrow, zebra finch and orange-cheeked waxbill.

A number of other birds have been experimentally infected. These include bobwhite quail, canary, English sparrow (Levine, Boley and Hester, 1941), barn swallow, goldenfinch, song sparrow (Stabler, 1953). Parenteral infections have also been produced experimentally in rats and kittens by Rakoff (1934) and in mice by Honiberg (1959).

### HOST-PARASITE RELATIONSHIPS

In columbid birds, *Trichomonas gallinae* is transferred with the pigeon milk from the crop of an infected parent to the newly hatched offspring within minutes after the latter's emergence from the egg. It is thus impossible for the young bird to escape contamination by an infected parent.

The normal location for *Trichomonas gallinae* is the upper digestive tract of its avian host. It includes the mouth pharyngeal region, esophagus, and crop. From the mouth it may enter the head sinuses and invade the brain and eye region, often appearing in the tears. The tissues of head and neck are regularly invaded by some strains, where their destructive activities become apparent. It has been reported that this organism may invade the proventriculus in chickens (Levine and Bandle, 1939) and pigeon (Stabler and Engley, 1946).

Its presence in the gizzard and intestine is a matter of dispute. A number of workers (Bushnell and Twiehaus, 1940; Wehr, 1943) did careful morphological studies on the trichomonads from the avian mouth and crop, as compared with those from the intestine and caeca. They have shown that the upper digestive tract forms are quite distinctive from those of the gut.

Stabler and Engley (1946) reported that *Trichomonas gallinae* was found in the organs of the thorax and abdomen, especially from the lungs, liver, heart and pancreas. It has been found also on the surfaces of thoracic and abdominal air sac, pericardium, and substernal membranes. The abdominal fluid may teem with the organisms. It has also been observed in the avian blood. Jaquette's experiment (1950) indicates that *Trichomonas gallinae* reaches the abdominal viscera via either the blood or lymphatic tracts, but not via the gut and bile ducts. Jaquette esophagotomized a series of 18 pigeons and placed virulent *Trichomonas gallinae* in their mouths. Although their crops remained negative, several of the birds showed typical liver trichomoniasis, ten of them showing hepatic lesions characteristic of trichomoniasis.

## PATHOLOGY

In the pigeon, trichomoniasis is essentially a disease of young birds. Eighty to 90 per cent of the adults are infected but show no signs of disease. The severity is due in part to differences in virulence of different strain of the trichomonad (Stabler, 1948). Severely affected birds lose weight, stand huddled with ruffled feathers, and may fall over when forced to move. A greenish fluid containing large numbers of trichomonads may be found in the mouth.

Lesions are found in the mouth, sinuses, orbital region, pharynx, esophagus, crop and even proventriculus. The early lesions in the mouth are small, yellowish, circumscribed areas in the mucosa. They increase in number and develop into large caseous masses which may extend through the base of the skull to the brain. The early lesions in the pharynx, esophagus, and crop are small, whitish to yellowish, caseous nodules which may also grow. A large amount of fluid may accumulate in the crop. The lesions in the liver, lungs, and other organs are solid, yellowish, caseous nodules ranging up to a centimeter or more in diameter.

Honiberg (1961) demonstrated that environmental conditions played an important role in modifying the pathogenicity of *Trichomonas gallinae*. The presence of agar

and methyl cellulose in the culture enhanced the development of subcutaneous lesions.

### IMMUNOLOGY

Different strains of *Trichomonas gallinae* differ greatly in virulence (Stabler, 1948a). Previous infection bestowed more or less immunity against virulent strains (Stabler, 1948b). Stabler (1953) found that immunity did not increase with age of uninfected birds. Certain breeds of strains of birds may be more sensitive than others. Levine and Brandley (1940) were able to infect chickens from one source readily while chickens from other source were very resistant.

### EPIDEMIOLOGY

In considering the epidemiology of trichomoniasis due to *Trichomonas gallinae*, it must be remembered that the organism is extremely delicate, incapable of encystment, and thus unable to survive unfavorable conditions for more than the briefest period. It is killed instantly upon drying. Since it inhabits the alimentary canal anterior to the muscular stomach, it cannot leave the host via the droppings (Stabler, 1954). This means that the nares, eyes, and mouth are the only portals by which *Trichomonas gallinae* may ordinarily leave the host. With the mouth, then, as the main point of infection, mouth to mouth contamination is the rule in certain bird types (columbids). Thus drinking water becomes virtually the sole avenue of infection in the epizootics such as occur in gallinaceous birds.

### TREATMENT

In drug therapy, some compounds have been administered in the drinking water, others have been applied topically to the birds' mouths and throats. Certain degrees of success have been claimed for the following compounds: weak hydrochloric acid (Brunthaler, 1945), acriflavine (Rosenwald, 1944) and iodine with or without glycerine (Brunthaler, 1945).

A number of workers have recommended the use of copper sulfate to eliminate *Trichomonas gallinae*. Jaquette (1948) administered the copper sulfate in drinking water and conducted the most extensive survey of this compound. He used pigeon (white kings) with subclinical infections. The most effective concentration for non-breeding pigeons was 100 mg of the drug per 100 cc of solution. The optimal concentration for breeding birds, without producing evidence of toxicity, was 35 mg of the drug per 100 cc of solution. Lower concentrations were less effective, and higher concentrations became markedly toxic. The toxic manifestations resulting from administration of copper sulfate were depression, e.g. loss of weight and possible liver damage. A high percentage of treated birds did become free of *Trichomonas gallinae*, although Jaquette felt that possibly all exposed birds suffered liver damage.

Schnitzer, Kelly, Soo-Hoo, Grunberg, and Unger (1951), Using a calcium salt of a stibonic acid (2-car-boxymethyl-mercaptobenzene stibonic acid), treated several protozoan infections in mice, including *Trichomonas gallinae*. The lesions were localized abscesses in the ventral subcutaneous tissues. The topical treatment was begun four hours after infection and repeated once after 24 hours. In less severe infections, the stibonic acid at 2 mg/cc, eliminated living trichomonad by 4 days, but in severe infections it was ineffective.

Remarkable anti-*Trichomonas gallinae* properties for 2-amino-5 nitrothiazole (enheptin) in pigeons have been reported by Stabler and Mellentin (1953). This compound removed the flagellate from all carriers and was completely effective against even the most virulent strains. The investigators recommended 7 daily doses of 28 mg/kg for homing pigeons and 45 mg/kg for commercial birds. Stabler, Schnitzer and Harmen (1958) used 6.3 gm enheptin soluble in a gallon of drinking water for 7-14 days in non-breeding pigeons. The birds consumed 9-27 mg of the drug per day and fifty-three of sixty-one infected birds were freed of their infection. Zwart (1959) obtained promising results with 1.25 per cent enheptin in the drinking water of a Dutch aviary where the infection had been found in zebra finches and in an orange-cheeked waxbill.

Several compounds, reported to be non-effective, are potassium permanganate, Lugol's solution, iron-sulfate (Florent, 1938), penicillin, the sulfa drugs (Sanders, 1945), metachloridine (Stabler, 1947), and a combination of crystalline potassium penicilline plus sulfadiazine, sulfamerazine, and sulfathazine (PSSS) (Stabler and Mellentin, 1953).

Hamilton and Stable (1953) have stressed the need for caution in administering the broad spectrum antibiotics in parasitic diseases. They cited the case of a gyrfalcon which had severe trichomoniasis and was given several treatments with aureomycin. Upon autopsy, it showed, in addition to the cankerous lesions, numerous sites of active infection with *Aspergillus fumigatus*. It is felt antibiotics may cause the exacerbation of latent fungal infections and even, perhaps, of latent trichomoniasis.

## 關於鳥類渦鞭蟲病 (Avian Trichomoniasis)

### 研究之檢討

#### 中文摘要

諸 亞 儂

鳥類渦鞭蟲病 (Avian trichomoniasis) 爲鳩、鴿、鷄及火鷄等之重要疾病，由原生動物渦鞭蟲 (*Trichomonas gallinae*) 寄生後所引起。渦鞭蟲寄生此等鳥類之消化管，自口腔傳入，經食

道至嗉囊，由此蔓延至肝、肺等器官，使這些部位發生癌腫，其感染程度與鳥類種別有關。蟲體由口腔、鼻及眼排出，再由口腔傳入另一寄主，故飲水，鴿乳為主要之傳染途徑。硫酸銅（Copper sulphate）和 2 Carboxymethyl-mercaptobenzene 為治療本病之有效藥物，尤以後者為最，抗生素則反引起菌類感染（Fungal infection）。

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