Trichomonas gallinae
(protist: flagellate)

Overview

Protists are single-celled organisms with membrane-bound nuclei (eukaryotes). Flagellates are protists that swim using one or more flagella (undulipodia); each arising from a small centriole (basal body, kinetosome) and having a microtubular axoneme core (2+9 configuration). Rather than forming a monophyletic group, flagellates are divided into several disparate groups: metamonads (amitochondriate flagellates), heteroloboseans (amoeboflagellates), euglenozoans (euglenids and kinetoplastids), stramenopiles (heterokonts), alveolates (dinoflagellates) and cercozoans (biflagellates). The metamonads comprise fornicates (diplomonads), parabasalians (trichomonads, hypermastigids, retortamonads) and preaxostylans (oxymonads). Parabasalid flagellates are anaerobic amitochondriate protists which have distinctive parabasal bodies (dictyosomes) adjacent to flagellar basal bodies (kinetosomes) and an axostyle-pelta complex providing structural support. Trichomonads are a major constituent group and most have 4-6 apical flagella, one being recurrent and often forming an undulating membrane supported by a costa. Most species have simple life cycles with longitudinal binary fission of motile-flagellated or rounded tissue-phase cells (only a few species form cysts). Many *Trichomonas* spp. have been reassigned to different genera based on the number of free anterior flagella e.g. *Tritrichomonas*, *Tetratrichomonas* and *Pentatrichomonas* having 3, 4 and 5 anterior flagella respectively. Confusingly, the name *Trichomonas* has been retained for some species with 4 anterior flagella (mainly those in man, some rodents and birds). Most trichomonad species are endocommensals in mammals, birds, reptiles and insects but several species are parasitic in the alimentary or urogenital tracts of man and domestic animals.

Classification:

Domain:	Eukaryota (membrane-bound nucleus)
Supergroup:	Excavata (with conspicuous ventral feeding groove)
Group:	Metamonad (amitochondriate flagellates with karyomastigonts)
Phylum:	Parabasalia (anaerobic flagellates with parabasal body supporting Golgi cisternae or dictyosome, trichomonads, hypermastigids, retortamonads)
Class:	Trichomonadea (single mastigont, comb-like structure absent, infrakinetosomal body absent)
Order:	Trichomonadida (lamelliform undulating membrane, B-type costa)
Family:	Trichomonadidae (5-6 flagella, cone-like axostyle)
Genus:	*Trichomonas* (parasites/commensals in tubular organs of vertebrates)
Species:	*T. gallinae* (causes canker in birds)

Parasite biodiversity and host range: Protists are unicellular eukaryotes that move using undulipodia (flagella or cilia), pseudopodia (false-feet) or a unique gliding motion. Flagellated species have one or more flagella with an internal microtubular core (in a characteristic 2+9 configuration comprising 2 single central microtubules and 9 peripheral doublets) anchored to a submembranous protein structure (known variously as a centriole, basal body, kinetosome or blepharoplast). Many types of flagellated cells have been described and recent phylogenetic studies have classified them into several disparate groups: including the metamonads (amitochondriate flagellates), heteroloboseans (amoeboflagellates), euglenozoans (euglenids and kinetoplastids), stramenopiles (heterokonts), alveolates (dinoflagellates) and cercozoans (biflagellates). While most flagellated protists are free-living organisms swimming and feeding in aquatic environments, representatives of several groups have developed symbiotic relationships with various hosts; some being endoparasitic in vertebrates (notably anaerobic metamonads in tubular organs, and heterotrophic euglenozoans occurring in blood or tissues), and some being parasitic in invertebrates (alveolates in crustacean tissues) (representatives tabulated below).

Higher taxonomy	Class or order	Family	Genera	Hosts (tissues)	Trans-mission*
Supergroup: Excavata (with conspicuous ventral feeding groove)					
Group: Metamonad (amitochondriate flagellates with karyomastigonts)					
Phylum: Fornicata (diplomonads)	Order: Diplomonadida (1-2 karyomastigonts)	Hexamitidae (2 karyomastigonts with binary axial symmetry)	*Giardia*	vertebrates (gut)	direct (f-o)
			Hexamita *Spironucleus*	vertebrates (tissues)	direct (f-o, w)
Phylum: Parabasalia (with parabasal body)	Order: Trichomonadida (3-5 anterior flagella plus recurrent flagellum)	Monocercomonadidae (costa absent, most without undulating membrane)	*Histomonas*	birds (gut, liver)	direct (f-o)
			Dientamoeba	vertebrates (gut)	direct (f-o)
		Trichomonadidae (stout axostyle, costa, undulating membrane)	*Trichomonas*	vertebrates (urogenital tract, gut)	direct (f-o, v)
		Cochlosomatidae (anterior adhesive disc)	*Cochlosoma*	birds (gut)	direct (f-o)
Group: Discoba (diverse group supported robustly by molecular studies)					
Phylum: Euglenozoa (flagella inserted in anterior pocket, heterotrophs, autotrophs)	Class: Kinetoplastea (heterotrophs, with extranuclear DNA (= kinetoplast) associated with mitochondrion)	Ichthyobodonidae (flagellar pocket continues as groove)	*Ichthyobodo* (= *Costia*)	fish (gills, skin)	direct (w)
		Parabodonidae (epizoic or endozoic)	*Cryptobia*	fish (gills, skin)	direct (w)
			Trypanoplasma	fish (blood)	indirect (v-b)
		Trypanosomatidae (monogenetic forms in insects/plants, digenetic forms in vertebrates & arthropods)	*Trypanosoma*	vertebrates (blood, tissues)	indirect (v-b)
			Leishmania	vertebrates (blood, tissues)	indirect (v-b)
Supergroup: SAR (Stramenopiles + Alveolata + Rhizaria) (3 groups unified by molecular studies)					
Group: Alveolata (with cortical alveoli)					
Phylum: Dinoflagellata (with unique mesokaryotic nuclei)	Order: Blastodiniales (uninucleate trophonts with chloroplasts)	Oodiniaceae (trophont with rhizoid-like invasive organelle)	*Amyloodinium* *Crepidoodinium* *Piscinoodinium*	fish (skin)	direct (w)
	Order: Syndiniales (multinucleate plasmodial trophonts)	Syndiniaceae (without chloroplasts)	*Haematodinium* *Ichthyodinium*	crustaceans, fish (tissues)	direct (w)
Phylum: Perkinsozoa (parasitic)	Order: Perkinsorida (released trophonts form biflagellated zoospores)	Perkinsidae (incomplete conoid)	*Perkinsus*	gastropods, bivalves (tissues)	direct (w)

*f-o = faecal-oral transmission; v-b = vector-borne transmission, w = water-borne transmission; v = venereal transmission

Metamonads are a group of excavates (with ventral feeding groove) that have several subcellular elements associated with their flagella forming a unique mastigont (an ultrastructural complex of organelles and cytoskeletal fibrils (incl. dictyosomes (Golgi bodies), centrioles (basal bodies) and a microtubular axostyle)). The metamonads comprise fornicates (diplomonads), parabasalians (trichomonads, hypermastigids, retortamonads) and preaxostylans (oxymonads). Most metamonads are amitochondriate but have retained reduced organelles of mitochondrial origin (fornicates containing mitosomes while parabasalians possess hydrogenosomes). Members of the phylum Parabasalia typically possess parabasal bodies adjacent to Golgi bodies (dictyosomes), and have microtubular arrays forming a conspicuous pelta-axostyle complex (cap-like pelta and a cone- or tube-like longitudinal axostyle). Six parabasalid classes are currently recognized on the basis of morphological, biological and molecular phylogenetic studies. Cells in three classes (Trichomonadea, Tritrichomonadea, Hypotrichomonadea) bear single mastigonts (set of kinetosomes (basal bodies) and associated appendages – ancestral unit comprising 4 kinetosomes) with flagella arranged in an anterior tuft, but many have one recurrent flagellum forming an undulating membrane (lamelliform or rail-type) supported by a costa (A- or B-type) and sometimes a basal comb-like structure and/or infrakinetosomal body. Many species are symbiotic (mutualists, commensals or parasites) in animals, although some are free-living in moist habitats. Most species have simple life cycles with longitudinal binary fission of motile-flagellated or rounded tissue-phase cells (only a few species form cysts). Cells in another three classes (Cristamonadea, Trichonymphea, Spirotrichonymphea) have more complex structures, often with multiple mastigonts bearing hundreds to thousands of flagella. Most were previously assigned to the now-defunct group Hypermastigida and they are primarily found as symbionts (mutualists) in insects (mostly termites).

Trichomonad taxonomy can be very confusing as many *Trichomonas* spp. have now been reassigned to sister genera based on the number of anterior flagella e.g. *Tritrichomonas*, *Tetratrichomonas* and *Pentatrichomonas* having 3, 4 and 5 anterior flagella respectively. Confusingly, the name *Trichomonas* has been retained for some species with 4 anterior flagella (mainly those in man, some rodents and birds). Recent ultrastructural and molecular biological studies have also led to the placement of the genus *Tritrichomonas* into a separate class (Tritrichomonadea) as the cells contain unique comb-like structures and infrakinetosomal bodies at the bases of their recurrent flagella (both lacking in members of the class Trichomonadea). Several genera (*Hypotrichomonas* and *Trichomitus*) were assigned to another class (Hypotrichomonadea) as their cells contained comb-like structures but lacked infrakinetosomal bodies.

Family	**Key characters to 'trichomonad' families in vertebrates***						**Representative genera**
	Number of flagella	**Undulating membrane**	**Costa**	**Axostyle**	**Comb-like structure**	**Infra-kinetosomal body**	
Class Tritrichomonadea (uninucleate to binucleate)							
Order Tritrichomonadida (endobiotic in vertebrates (mammals, birds, reptiles, amphibia, fish))							
Tritrichomonadidae	4-5	rail-type	A-type	tube-like	present	present	*Tritrichomonas*
Simplicimonidae	4	absent	absent	tube-like	present	present	*Simplicimonas*
Monocercomonidae	4	absent	absent	cone-like	present	present	*Monocercomonas*
Dientamoebidae	0-4	absent	absent	cone-like	absent	absent	*Dientamoeba, Histomonas*
Class Trichomonadea (single karyomastigont)							
Order Trichomonadida (with costa) (endobiotic in vertebrates (mammals, birds, reptiles, amphibia) and invertebrates)							
Trichomonadidae	5-6	lamelliform	B-type	cone-like	absent	absent	*Cochlosoma, Trichomonas, Trichomitopsis, Tetratrichomonas, Pentatrichomonas*
Order Honigbergiellida (without costa) (endobiotic in vertebrates (mammals, reptiles, amphibia))							
Hexamastigidae	5-6	absent	absent	cone-like	absent	absent	*Hexamastix*
Class Hypotrichomonadea (single karyomastigont)							
Order Hypotrichomonadida (endobiotic in vertebrates (reptiles, amphibia, mammals) and invertebrates)							
Hypotrichomonidae	4	lamelliform	A-type	cone-like	present	absent	*Trichomitus, Hypotrichomonas*

*Taxa found exclusively in invertebrate hosts (such as termites and cockroaches) are not listed.

The class Trichomonadea contains a diverse range of cells with single karyomastigonts giving rise to 2-6 flagella, with one being recurrent but lacking a comb-like structure and infrakinetosomal body. Two orders are recognized: Honigbergiella (undulating membrane absent or lamelliform without supporting costa); and Trichomonadida (most with lamelliform undulating membrane supported by B-type costa and stout cone-like axostyles). The family Trichomonadida contains some 10 genera (*Cochlosoma, Lacusteria, Pentatrichomonas, Pentatrichomonoides, Pseudotrichomonas, Pseudotrypanosoma, Tetratrichomonas, Trichomitopsis, Trichomonas* and *Trichomonoides*) which vary in their cellular, organellar and flagellar configurations, zoogeography (host ranges), and biological characteristics (heterotrophs ranging from mutualists to commensals to parasites). Trichomonads (*sensu latu* = in the broadest sense) usually exhibit strong site specificity (tissue tropism) and occur in the alimentary, urogenital or respiratory tracts of their hosts, where they may cause very different types of disease. Several specialized species living in the urogenital tracts of vertebrates may cause severe inflammatory diseases, with *Trichomonas vaginalis* causing vaginitis in humans, and *Tritrichomonas foetus* causing bovine infertility. A few species living in the upper respiratory and alimentary tracts of birds may cause life-threatening diseases, including *Trichomonas gallinae* causing canker in birds. In contrast, those inhabiting the intestinal tracts of vertebrate and invertebrate hosts are often considered to be symbiotes or commensals (rather than parasites) as most infections appear benign. Rather than try to cover trichomonad biodiversity and their disparate clinical significance in one comprehensive section, it has been elected to showcase representatives in 4 separate sections, targeting:

- urogenital infections by *Tritrichomas foetus* in cattle;
- urogenital infections by *Trichomonas vaginalis* in humans;
- oral infections by *Trichomonas gallinae* in birds; and
- enteric infections by *Trichomonas, Tetratrichomonas* and *Pentatrichomonas* spp. in a wide range of hosts.

While numerous *Trichomonas* spp. have been detected in the alimentary tracts of birds, most occur below the level of the stomach (in the intestines) and only a few infect organs above the stomach (oral cavity, throat). One species in particular, *Trichomonas gallinae*, has been associated with severe necrotizing caseous lesions in the upper digestive tract causing a condition commonly known as canker. Infections have been reported in various bird species around the world, notably in juvenile pigeons (squabs) but also including domestic poultry (turkey poults, chickens) as well as aviary birds (budgerigars, parrots).

Parasite species	Size (µm)	Vertebrate Hosts	Location	Clinical signs	Distribution
Class: **Trichomonadea** (single karyomastigont) [4-6F-CLS-IKB(+CA in most genera, TA or reduced in others)]†					
Order: Trichomonadida [5-6F+LUM+BC]					
Family: Trichomonadidae [+CA]					
Trichomonas [5F(=4A+R)+LUM-RF]					
Trichomonas gallinae (syn. *Cercomonas gallinae, C. hepaticum, Trichomonas columbae*) [+AF]	5-20 x 2-9	Columbiformes (rock pigeon, wood pigeon, white-crowned pigeon, band-tailed pigeon, pink pigeon, Seychelles blue-pigeon, Eurasian collared dove, ring turtle-dove, Madagascar turtle-dove, Indian dove, spotted dove, red-eyed dove, laughing dove, zebra dove, mourning dove, Galapagos dove, white-winged dove, white-bellied dove, ground dove, white-tipped dove), Accipitriformes (bald eagle, black kite, Egyptian vulture, shikra, African goshawk, rufous-chested sparrowhawk, Cooper's hawk, western marsh-harrier, northern goshawk, gray hawk, red-shouldered hawk, red-tailed hawk, Eurasian buzzard, golden eagle, Bonelli's eagle, booted eagle, secretary-bird), Falconiformes (Cooper's hawk, red-tailed hawk, red-shouldered hawk, golden eagle, lesser kestrel, Eurasian kestrel, American kestrel, red-necked falcon, merlin, lanner falcon, saker falcon, gyrfalcon, peregrine falcon, sparrow hawk, duck hawk), Strigiformes (barn owl, scops owl, screech-owl, horned-owl, eagle-owls, tawny owl, barred owl, little owl, boobook), Gruiformes (rails); Passeriformes (finches, canaries, sparrows, jays, crows, currawong, magpie, ravens, mockingbirds), Psittaciformes (lorikeets, budgerigars, cockateil, tovi parakeets), Cuculiformes (channel bill cuckoo), Caprimulgiformes (frogmouth), Anseriformes (ducks, geese), rarely Galliformes (chickens, turkeys, bobwhite quail), Charadriiformes (gull); plus experimental subcutaneous assays in mice	upper digestive tract (mouth, pharynx, oesophagus, proventriculus, crop), liver, lungs	canker (roup, frounce), necrotizing lesion	worldwide

Trichomonas gypaetinii		Accipitriformes (bearded vulture, Egyptian vulture, black vulture)	upper digestive tract	non-pathogenic	Europe
Trichomonas stableri	slender 13 x 6, rounded 8 x 6	Columbiformes: columbid (pigeons)	oral cavity, oesophagus	caseo-necrotic lesions	North America

†Coding: + = present; - = absent; #F = total number of flagella; #A = number of anterior flagella; R = recurrent flagellum; RF = recurrent flagellum extending posteriorly as free flagellum; LUM = lamelliform undulating membrane; BC = B-type costa; AF = axostyle protrudes posteriorly; CA = cone-like axostyle (*Trichomonas*-type); TA = tube-like axostyle (*Tritrichomonas*-type); CLS = comb-like structure; IKB = infrakinetosomal body.

Parasite morphology: *Trichomonas gallinae* forms motile trophic stages known as trophozoites which possess 5 flagella. Trophozoites have ovoid to pyriform bodies ranging from 5-12 µm in length, with well-fed forms becoming robust and extending up to 20 µm in length. Cells possess a single prominent nucleus located anteriorly adjacent to a distinctive parabasal body formed by dictyosomes (Golgi complexes). The nucleus and parabasal body are associated with small dense basal bodies (kinetosomes) forming a single karyomastigont unit (ancestral unit with 4 kinetosomes). The kinetosomes give rise to 4 flagella which project forwards forming an anterior tuft around 8-10 µm in length. In *T. gallinae*, a separate kinetosome gives rise to a fifth flagellum which is recurrent (directed posteriad) and attached longitudinally to the cell body forming a short undulating membrane that does not reach the end of the body. The undulating membrane is lamelliform in appearance (rather than rail-like) and is underpinned by a slender elongate rod-like structure in the cell cytoplasm known as the costa, which is striated with a periodicity known as B-type (rather than A-type). Trophozoites swim with a characteristic jerky forward motion, and the undulating membrane imparts a quivering/shimmering appearance to the cell body. Trophozoites also possess a slender longitudinal hyaline rod-like structure known as an axostyle which is composed of concentric rows of microtubules forming a cone (rather than a tube). The axostyle begins near the nucleus and runs posteriorly through the cell body, protruding through the posterior end and terminating in a sharp point. Trichomonads are anaerobic and do not have mitochondria, but rather possess membrane-bound organelles known as hydrogenosomes (formerly called siderophil granules) often located in rows along the axostyle. *T. gallinae* does not form true encapsulated cysts, but cells may sometimes round up and lose their flagella and undulating membranes. This generally occurs under unfavourable conditions, and has been observed to be reversible. These stages have been called ‘pseudocysts’ and they are thought to behave as resistant forms under stressful environmental conditions.

Site of infection: Trophozoites are usually found in the upper alimentary tract (mouth cavity, pharynx, oesophagus, proventriculus, crop) and nasal cavities of birds, sometimes penetrating to underlying organs (liver, lungs, heart, pancreas). In juvenile pigeons (squabs), infection may be seen at the umbilicus and can track up into the liver via the falciform ligament connection. Infections have been recorded mainly Columbiformes (> 20 species of pigeons/doves), birds of prey (> 40 species of eagles, hawks, falcons, vultures and owls), as well as in several Psittaciformes (lorikeets, budgerigars, parakeets), Passeriformes (finches, canaries, sparrows, corvids, mockingbirds), Gruiformes (rails), Cuculiformes (cuckoos), and Anseriformes (ducks, geese), but rarely in Galliformes (chickens, turkeys, bobwhite quail) and Charadriiformes (gulls).

Pathogenesis: Infections may cause clinical disease (trichomoniasis) in the upper digestive and respiratory tracts of birds, varying in severity from mild mucosal inflammation to caseous areas that block the oesophageal lumen and prove fatal. The disease is often canker, but is also known as frounce in raptors, or roup in columbiforms and psittaciforms. Trophozoites occur free in the lumina of the tubular organs as well as browsing and attaching to mucosal surfaces where they cause diffuse cellulitis and inflammation with increasing numbers of heterophils. The parasites may then invade underlying tissues causing rounded lesions with spur-like projections that coalesce to form ulcerative and exudative lesions. The lesions may become necrotic with thick caseous masses (composed of fibrin, inflammatory cells, bacteria and trichomonads) that can block passages (mouth, pharynx, oesophagus, crop, sometimes proventriculus and conjunctival sac). Some parasite strains also create diphtheritic membranes associated with fibrinous lesions in internal organs such as pharynx, lungs, liver and peritoneum. Affected birds exhibit depression, drowsiness, listlessness, weakness, ruffled and matted feathers, and have greenish exudates from the beak and/or yellow cheesy caseous masses (canker) around the beak, eyes or nasal passages. Nasal and oral exudation may impair breathing (with dyspnoea) and feeding (with dysphagia, excessive salivation, swallowing difficulties, sometimes regurgitation) leading to anorexia, weight loss, emaciation, inability to stand or maintain balance, increased thirst, swollen faces, pendulous crops, sometimes diarrhoea, and even death (as early as 4 days after infection, but usually around 3 weeks). The severity of infection depends on differences in host susceptibility, the intensity of infection, the stage of infection (acute/chronic) and strain differences in parasite virulence, some strains notably causing systemic disease by invading internal organs (liver, lungs, pericardium, air sacs, pancreas) occasionally forming abscesses (liver, lungs). Clinical infections occur mainly in young birds, especially pigeon squabs, although many birds recover from disease but may remain asymptomatic carriers. Birds harbouring light infections or infections by avirulent strains appear to develop some protective (premunitive) immunity against disease which wanes after infections are lost. Humoral and cellular immune responses

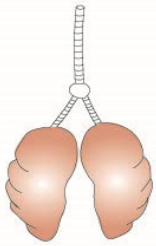
appear to be involved following the phagocytosis of parasites by leucocytes, leading to the production of serum and secretory antibodies, and lymphocytic infiltrates. The worldwide distribution of disease closely mirrors that of rock pigeons which are considered to be principal hosts responsible for most dissemination. Clinical disease occurs more frequently in spring and summer months and is thought to be associated with peak periods of bird reproduction (including courtship, nesting and feeding nestlings).

Developmental cycle and mode of transmission: Trichomonads have simple direct life-cycles involving the multiplication of trophozoites by longitudinal binary fission and their transmission to new hosts by contact or contaminated food or water. The parasites do not form true cysts, but rounded nonflagellated pseudocysts may also be involved in transmission cycles. Parasites are very sensitive to desiccation and survive poorly in the external environment, but trophozoites have been found to remain viable for up to 5 days in moist grains and for several hours in water sources (depending on salinity). Studies have indicated that the minimal infective dose may be as small as a single trophozoite. Birds come into direct contact with each other when billing during courtship, when cross-feeding, and when feeding nestlings with maternal crop milk or regurgitated food. Birds with oral or throat lesions often have difficulty swallowing large pieces of grain and may drop them where they are taken up by other birds. Birds of prey have been found to become infected by feeding on infected prey where trophozoites may survive for up to 48 hours after host death. Risk factors for clinical infections include high bird population densities (especially overcrowding in caged birds), poor hygiene (particularly of food and water sources), and concomitant infections (notably pigeon circovirus). Epidemiological studies have also shown that the prevalence of infections in wild birds is greatest during hot dry weather, possibly due to more birds gathering at fewer water holes.

Differential diagnosis: Clinical infections are strongly suggested by the occurrence of characteristic caseous (canker) lesions in the oral/nasal cavities of birds, but there may be some confusion with other pathological conditions (such as nonspecific pharyngoesophagitis, vitamin A deficiency, sialoliths (salivary stones)) or other infectious diseases (poxviruses, fungi, *Capillaria* nematodes), particularly those involving granuloma formation (e.g. tuberculosis, mycoplasmosis, salmonellosis, coligranuloma). Definitive diagnosis is made by the direct microscopic detection of motile trophozoites in exudates or scrapings collected from birds. Wet mounts are generally prepared from swabs collected from the oral cavity and examined unstained by high contrast bright-field microscopy or by dark-field, phase-contrast or interference-contrast microscopy. Smears may also be prepared and stained with Giemsa, silver, iron haematoxylin, malachite green, methylene blue, or acradine orange. Host tissues may also be collected at autopsy and histological sections examined after staining with haematoxylin and eosin, Giemsa or periodic acid-Schiff stains. Several studies have also developed immunohistochemical stains using polyclonal or monoclonal antibodies labelled with fluorochromes or enzymes. Parasites may be readily cultured *in vitro* using a variety of liquid/semiliquid media at 37-40°C, but essential macromolecules, vitamins and minerals need to be included as trophozoites lack the ability to synthesize many materials *de novo*. Suitable media include Diamond's TYI media, Trichosel medium, medium 199 and Hollander fluid (HF) medium, most with serum supplements to provide lipids, fatty acids, iron and trace metals. A range of tissue cultures have also been used to support trichomonad growth, including chick liver cells, monkey kidney cells, chicken hepatocellular carcinoma cell (LMH) monolayers, and Japanese quail fibrosarcoma cell (QT-35) monolayers. Experimental studies have been used to examine parasite pathogenicity following the subcutaneous inoculation of known numbers of axenically cultured trophozoites and then measuring lesion size 6 days later. An enzyme immunoassay has been developed to help diagnose infections in poultry by the detection of specific host antibodies against parasite antigens. More recently, a range of molecular biological techniques have been used to detect and characterize trichomonads in clinical samples by isoenzyme electrophoresis, Western blot analyses, restriction enzyme analyses and the polymerase chain reaction (PCR) amplification of nuclear gene sequences (large and small subunit ribosomal RNA, internal transcribed spacers, alpha-tubulin, iron-hydrogenase) and fluorochrome- or chromogenic-based *in situ* hybridization (ISH) assays.

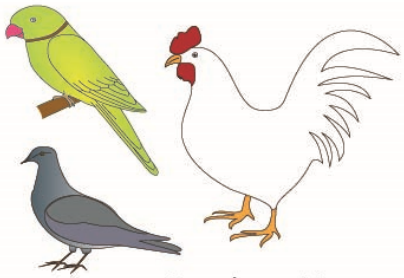
Treatment and control: A variety of chemotherapeutic agents have been used to treat clinical infections in birds, initially involving adding stringents, disinfectants or antibiotics to drinking water or topically to mouth and throat of affected birds, and subsequently using antiparasitic drugs as water or food additives or as systemic formulations. In particular, good results were achieved using nitroimidazole drugs (metronidazole, carnidazole, ronidazole, dimetridazole) which exhibit selective toxicity for anaerobic bacteria and protozoa. These drugs are usually well tolerated but they may cause some adverse side-effects and toxicity problems (e.g. dimetridazole). There are also growing instances of treatment failures due to the emergence of drug-resistant parasite strains due to subtherapeutic dosing and sustained prophylactic use. Recent studies have indicated that some synthetic polyphenolic/flavonoid compounds (chalcones) may be used to treat infections. In all instances, however, large caseous masses do not respond to treatment and recourse is often made to their surgical removal. A variety of preventive measures have been employed to reduce sources of infection and limit transmission. Regular health surveillance and treatment can help prevent the introduction of infections in commercial flocks and aviaries, especially when new or translocated birds are isolated in quarantine. Barrier systems and screens should be used around bird holding facilities to deny access to potential carriers (wild birds), and birds of prey should be prevented from hunting urban pigeons. Strict hygiene and sanitation may be used to limit environmental contamination by avoiding communal food and water sources, regularly cleaning feeders and water containers using bleach disinfectants, and thoroughly drying washed fomites, cages and buildings. Good husbandry practices should be used to keep birds in optimal nutrition in non-crowded stress-free conditions to stop them from becoming more susceptible to infection and disease.

Trichomonas gallinae

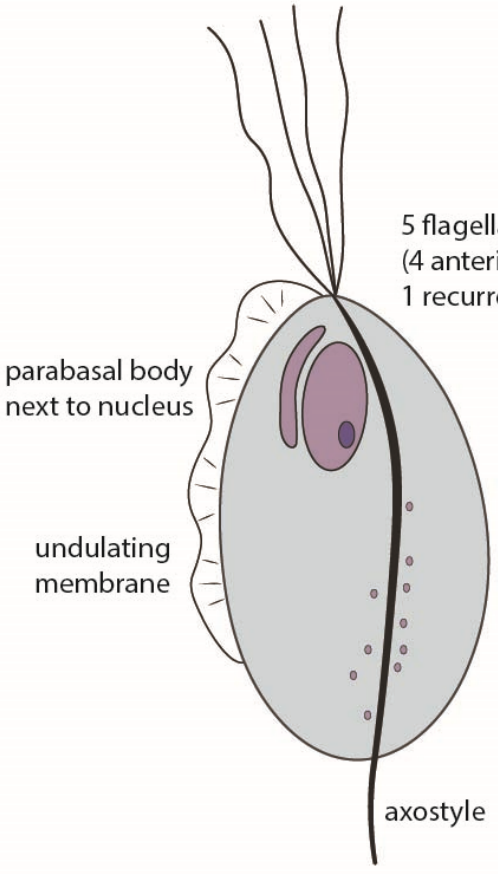


upper alimentary tract
(necrotizing caseous
lesions (canker))

division by
longitudinal
binary fission



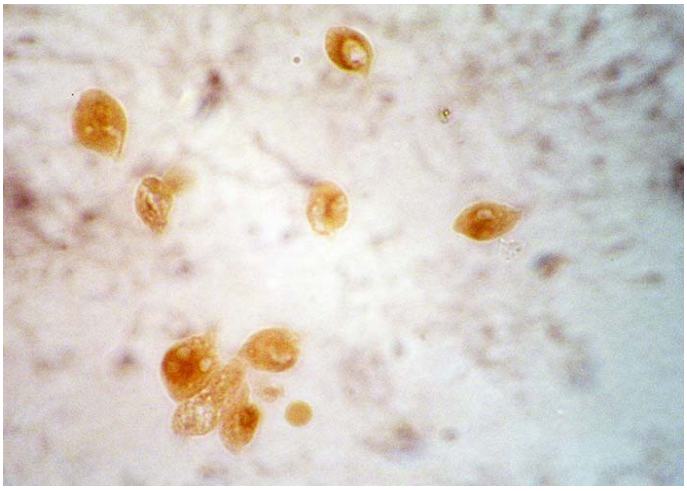
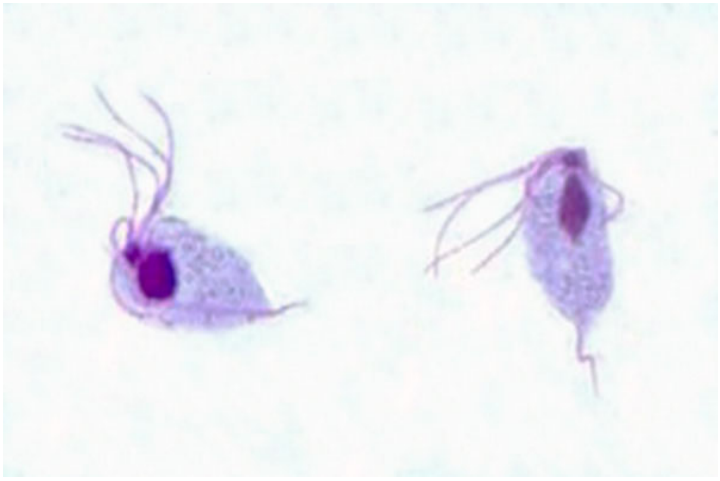
Vertebrate Hosts
(birds)



trophozoites
(5-20 µm)

no cyst formation

direct transmission by transfer of trophozoites
during close contact or via contaminated fomites

	
Trichomonas gallinae trophozoites	*Trichomonas gallinae* trophozoites