

## Bovine Trichomoniasis

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### INTRODUCTION

Reproductive diseases are a significant cause of reduced productivity in cattle breeding systems. Infectious diseases are usually endemic and result in less efficient reproduction, infertility, miscarriage, and reduced productivity. These diseases are typically asymptomatic or subclinical, complicating their identification (Campero et al. 2003). Trichomoniasis is one of the livestock's most common protozoal diseases, and the most widely known trichomonad in veterinary medicine is *Tritrichomonas (T.) foetus*, the etiologic agent of bovine trichomoniasis.

Bovine trichomoniasis is a venereal protozoan disease that occurs in many geographic areas worldwide, with most cases occurring in intensively managed cattle farms (Florin-Christensen and Schnittger 2018). This causative agent is *T. foetus*, a flagellated protozoan that occurs solely in cattle genitalia (Yao 2013). In infected cattle, there is vaginitis, endometritis, infertility, miscarriage, and early embryonic death (Martin et al. 2021). Mazzanti first discovered it in 1900, and since then, much work has been done on its incidence, especially in the United States and Britain. Emmerson (1932) reported the first case of bovine trichomoniasis in Pa McNutt in the U.S.A., and Walsh and Murray reported the disease in Iowa in 1930 (Danan and Teschke 2015). Several protozoan species occur in the bovine reproductive system, like the preputial cavity in bulls. These protozoa include *T. foetus*, which may be zoonotic, and cause opportunistic infections in humans (Yao 2012).

The trophozoites of *T. foetus* are transmitted among bulls and cows during coitus, causing metritis and early embryonic death in cows, but infected bulls typically are without clinical signs (Parthiban et al. 2015). Infected cattle with

trichomoniasis might experience mild "vaginitis" or "endometritis," or the infection can be as serious as causing severe inflammation throughout the whole reproductive tract. Other complications may include pyometra in pregnant cattle, inability to be pregnant, and decreased calving ratio (Alobaidii et al. 2021). Sexual intercourse is the primary transmission mode of *T. foetus* from infected to healthy animals, most commonly via natural mating (BonDurant 2005). The bulls get infected while breeding infected cows and stay symptomless carriers of the infection (Fig. 1). However, the protozoan can subsist in the raw and processed semen of breeder bulls and be transmitted via artificial insemination (AI) (Eaglesome et al. 1995). Also, the protozoa endures freezing in liquid nitrogen, where the protozoa-contaminated semen is preserved (Yao et al. 2011). Hence, artificial insemination cannot eliminate the disease but can reduce the prevalence rate, as reports indicate that AI substantially reduced the incidence of trichomonosis and other venereal infections (Van Bergen et al. 2006). Other means of transmission are also possible. For example, Goodger and Skirrow (1986) reported that unsanitary estrus detection through vaginal examinations led to the transfer of *T. foetus*, carried via contaminated gloves, from infected to non-infected cows.

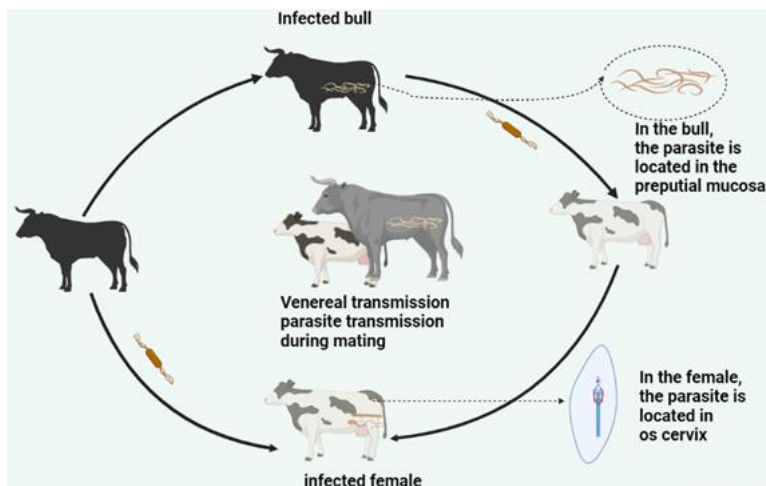
The transmission of *T. foetus* by insects, such as flies, was reported by Clark et al. (1977), as insects can transmit infection among cows. Also, infection is possible through direct contact between a healthy cow's vulva and that of an infected cow and passive transmission through a healthy bull's penis. Some females maintained infection up to 9 weeks postpartum through a normal pregnancy (Skirrow et al. 1985). *T. foetus* decreases cattle productivity by increasing reproductive losses and reducing conception rates. Bovine trichomoniasis causes a sustained breeding season (Adeyeye et al. 2012). The protozoa were also documented to cause human infections in immunocompromised and immunosuppressed individuals, including meningoencephalitis and peritonitis (Yao 2012), as mentioned in Fig. 1.

Differential diagnoses of bovine trichomoniasis include anaplasmosis, bovine viral diarrhea, brucellosis, campylobacteriosis, chlamydiosis, infectious bovine rhinotracheitis, leptospirosis, and neosporosis.

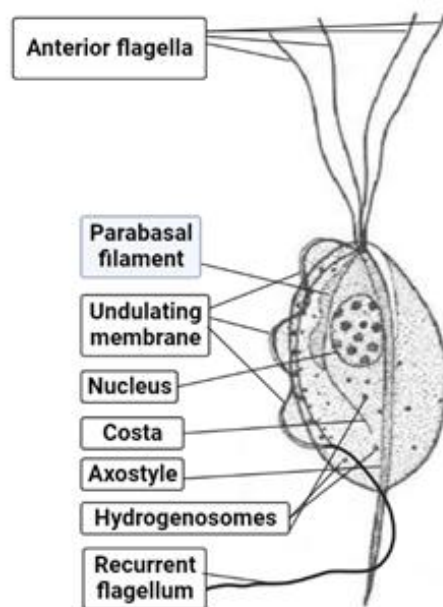
These diseases may cause clinical signs, including infertility, vaginitis, pyometra, abortions, and vaginal discharge, which should be excluded (Florin-Christensen and Schnittger 2018).

### Morphology of the Agent

*T. foetus* has a pyriform or ovoid trophozoite stage about 8–18 µm long and 4–9 µm wide (Issa 2014). The locomotive activity of the trophozoite occurs via several structures,



**Fig. 1:** The life cycle of bovine *T. foetus*.



**Fig 2:** A *Trichomonas foetus* trophozoite.

like the undulating membrane and four flagella. The flagella are located in the cell's apical pole and originate from the basal bodies or kinetosomes. Three similar-length flagella are directed forward, while the fourth flagellum (the recurrent flagellum) is directed toward the cell's posterior part, is associated with the undulating membrane, and stretches beyond the undulating membrane's posterior end (Benchimol 2004). Cattle (*Bos indicus* and *B. taurus*) are the usual hosts of *T. foetus*. The number of flagella after examination under a phase contrast microscope or after staining is an essential morphological feature that can assist in differentiating *T. foetus* from other flagellated bovine parasites.

Nevertheless, non-*T. foetus* trichomonads are invariably challenging to distinguish from *T. foetus*, depending on morphology (Pereira-Neves et al. 2003). Trichomonads are highly motile and are about the size of leukocytes.

*Trichomonas vaginalis* has four flagella on the anterior side, while *T. foetus* has three anterior flagella and one recurrent flagellum (Benchimol et al. 2006) (Fig. 2).

### Prevalence

Bovine trichomoniasis is a significant problem worldwide. In Iraq, *T. foetus* infection was first reported in cows in Nineveh province, with a higher infection rate in >2–4-year-old cows and early embryonic death (Alobaidii et al. 2021). The protozoan disease is widespread, affecting many cattle herds in North and South America, parts of Europe, Africa, Asia, and Australia (Güven et al. 2013; Yao 2013; de Oliveira et al. 2015). Trichomoniasis is prevalent in Argentina, reducing pregnancy rates by 15%–25% (Campero et al. 2003). The within-herd prevalence rates of trichomoniasis in bulls are 26.4% in South Africa (Pefanis et al. 1988), 30.6–50.0% in Australia, and 5.8–38.5% in California (Skirrow et al. 1985). Many studies have reported infected bulls with *T. foetus* in the United States of America (Szonyi et al. 2012), Argentina (Mardones et al. 2008), Spain (Mendoza-Ibarra et al. 2012), Austria (McCool et al. 1988), the Republic of Transkei (Pefanis et al. 1988), Colombia (Griffiths et al. 1984), Tanzania (Swai et al. 2005), Nigeria (Bawa et al. 1991), Canada (Waldner et al. 2013), and Argentina (Molina et al. 2013). Australian surveys have shown infection rates of about 8.4%. About 10.7% of cows were infected with *T. foetus* in a sizeable Californian dairy farm (Goodger and Skirrow 1986). Northern Spain was considered a hotspot of infection since natural breeding is still implemented (Mendoza-Ibarra et al. 2012). Compared to other livestock diseases, The rate of *T. foetus* infection is expected to be low in the United States. Hence, control of the disease is not unified at the federal level, leading to the enactment of different regulations among states (Martin et al. 2021). Twenty-six states had trichomoniasis control/management program regulations in place to curtail the spread of this disease as of 1 April 2014 (Yao 2015). The herd size and bull:cow ratio are vital for infection prevalence (Mardones et al. 2008). Factors

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associated with a high bovine trichomoniasis rate in a herd include the herd size. So, the infection hazard is higher in large herds that share grazing, have a significant number of bulls with a high ratio of 4 years or older bulls, and a high ratio of bulls to cows (Szonyi et al. 2012).

### Pathogenesis and Pathology

The underlying factors affecting the loss of the embryo or fetus are not accurately identified. However, some of these mechanisms include the adverse effects of enzymes released by the protozoan, the effect of antiparasitic inflammatory reactions in the uterus, and the parasite's direct mechanical activity (Campero and Cobo 2006). Cyto-adherence and cytotoxicity are thought to be the principal mechanisms (Petropolis et al. 2008).

The concentration of *T. foetus* in the cervicovaginal mucus changes during the estrus cycle, and the highest concentration is observed a few days prior to the estrus phase (Schuster and Schaub 2001). The uterus was believed to be the primary infection site, but several studies of naturally infected cows indicate that the os cervix is the preferred site. Placentitis and a uniform pattern of placental and fetal lesions are also seen. The fusional stage of abortion is associated with variation in the pathogenicity of *T. foetus* strains. The infective threshold number of organisms or the host's immune condition is unknown and should be further studied. Bovine trichomoniasis causes abortion, usually during early gestation (BonDurant 2005). A scant purulent preputial discharge may be observed within the first two weeks of infection. Older bulls seem to become permanent *T. foetus* carriers, possibly due to the growth of epithelial crypts in the preputial cavity (Walker et al. 2003). It is rare for abortions due to *T. foetus* to occur after six months of gestation. The cow or heifer usually recovers spontaneously when the placenta and fetal and placental membranes are eliminated following abortion. However, chronic catarrhal or purulent endometritis, which may cause permanent sterility, may occur if a part of the placenta or membrane remains. Sometimes, the abortion fails to occur following fetal death, and maceration results in the uterus (Schlafer and Foster 2016). There is a lack of research on how *T. foetus* affects the conceptus and causes abortion. However, there is a possible role of tumor necrosis factor (TNF) in malaria-induced abortion, and lymphokine-mediated cytotoxicity is perhaps essential in bovine trichomoniasis (Yule et al. 1989).

Microscopic lesions in aborted fetuses consist of pyogranulomatous bronchopneumonia and necrotizing enteritis with trichomonads invading the tissues. Specifically, pulmonary air passages contain many neutrophils, macrophages, multinucleated giant cells, meconium, and trichomonads located extracellularly and phagocytized. Small focal collections of lymphocytes and plasma cells are observed in the interstitium. Multiple trichomonads are dispersed in the aborted fetuses' interlobular septal connective tissue and aggregated in the fetuses' interlobular

septal and subpleural vessels. Additionally, fetuses may have pronounced focal hemorrhage in interlobular septa and airways of some pulmonary lobules. Mild focal epithelial degeneration to diffuse necrosis and loss of epithelium might occur in the gastroenteric tract. Fetuses may have marked mucosal, submucosal, and subserosal hemorrhage. Also, the forestomach, abomasum, and small and large intestines may contain thrombotic lesions. Multiple large intraepithelial vesicles comprising fibrin strands and erythrocytes occur in the mucosa of the rumen and omasum overlying hemorrhagic foci (Schlafer and Foster 2016).

### *Tritrichomonas foetus* in Bulls

Infection with *T. foetus* is limited to the reproductive system and, in bulls, the preputial cavity and urethral orifice (Michi et al. 2016). Bulls are the natural carriers of the parasite (Higgins 2006). Young bulls are either more tolerant to *T. foetus* or can eliminate the infection more efficiently. Bulls 1–2 years old are refractory to infection (Michi et al. 2016). The parasite survives in fresh, pure, or diluted semen that has been refrigerated and can resist cryopreservation, and transmission through AI with contaminated semen is probable (BonDurant 2005). Feces are commonly found in the preputial cavity of bulls since they tend to mount each other. The feces may comprise trichomonad species other than *T. foetus*, such as *Pentatrichomonas hominins* and nonpathogenic species of *Tetratrichomonas* (Campero et al. 2003). The possibility of *T. foetus* contagion between males is considered very low.

Chronically infected bulls are considered asymptomatic carriers for years since the clinical signs of the disease are not apparent, but bulls infected with the acute form have lesions and discharge in the genital organs for a short time (González-Carmona et al. 2012). Unlike female cattle, histopathological changes in bulls are absent, and unlike female cattle, bulls do not self-cure without prior vaccination (Higgins 2006). Previous studies have been unable to detect lesions associated with *T. foetus* infection. Tests such as the mucus agglutination test and the ELISA test have limited use in diagnosing the parasite since they are not adequately sensitive and specific, and infected bulls do not develop enough immune responses for serological diagnoses (Voyich et al. 2001).

Rhyan et al. (1999) detected *T. foetus* in the superficial layers of the penile and preputial epithelium in histological sections of the reproductive tracts of bulls infected with *T. foetus*. However, they failed to detect the parasite's invasion of these structures' basement membrane or dermis. The absence of the parasite's invasion of these tissues may explain the limited immunologic reaction in *T. foetus*-infected bulls. Significantly higher amounts of specific antibodies in the preputial secretions of infected bulls than non-infected bulls resulted from local antigen uptake, processing, and antibody deposition. The absence of pathologic changes and the immune response's inability to eliminate the parasite from

the preputial cavity led to chronic infection, particularly in older bulls.

Several studies have tried to determine the correlation between the age of bulls and infection risk and concluded that as the bull ages, the chance of *T. foetus* infection increases (Rae et al. 2004). Investigators of *T. foetus* have likewise argued that the growth of crypts in old bulls is a cause of age-related vulnerability to *T. foetus* (BonDurant and Honigberg 1994). Several studies have proposed different susceptibility levels of cattle breeds to *T. foetus* infection (Rae et al. 2004).

### **Trichomonas foetus in Cows**

Cows are more susceptible to *T. foetus* infection as only 103 trichomonads are required to establish infection in female bulls (Higgins 2006). It was shown that an infected bull could infect previously uninfected susceptible nulliparous cows by a single service with a 95% infection rate. Transmission from infected cows/heifers to bulls appears less efficient (Yao 2015). The late-gestation abortion by trichomonads supports the observed occurrence of "carrier cows." The cows can deliver normal calves and maintain infection throughout pregnancy and six to nine weeks postnatal, becoming an infection source for bulls (Yule et al. 1989).

Infection can be self-limiting in cows, and the parasites can be cleared from the reproductive tract after about three months (Yule et al. 1989). Most gestations are lost approximately 2.5 weeks postconception when maternal recognition has taken place, but embryonic death might happen at any time until five months of gestation (BonDurant 1985). However, later in gestation, embryonic or fetal loss results in abnormally long interservice intervals (2–5 months). Fetal deaths at approximately 50 to 70 days postcoitus have been reported, and deaths as late as eight months' gestation may occur (BonDurant 1985). After a variable period of infertility after the initial exposure, cows regain their fertility, even though infected bulls breed them. This suggests that infected cows develop an immune response to the parasite that reduces their susceptibility to subsequent infection for some time, possibly as long as six months, the fetal membranes are retained, and a chronic catarrhal or purulent endometritis usually results (Anderson et al. 1994). After the parasite has initially multiplied in the vagina, it remains in the uterus, and the cells' number in the vagina may change during the estrous cycle. This fluctuation may be influenced by the cycle type, regular or prolonged (Mancebo et al. 1995). Chronically infected cows with *Trichomonas foetus* were carriers of the infection for as long as ten months (Mancebo et al. 1995). Also, chronic infections were observed throughout normal pregnancies, with the ability to isolate *T. foetus* for as long as nine weeks.

### **Diagnosis**

Due to the insidious nature of *T. foetus* infection, the parasite occurrence on cattle farms often goes undetected until a

substantial loss has already occurred. Infection in females often goes undetected due to early abortion resulting in re-exposure of females to males, increased calving to conception intervals (BonDurant 2005), and smaller, less developed calves due to the shortened weaning season. The most common practice for detecting infection within a herd is the demonstration of a live *T. foetus* by culture scrapings from the preputial smegma in sexually rested bulls (Higgins 2006).

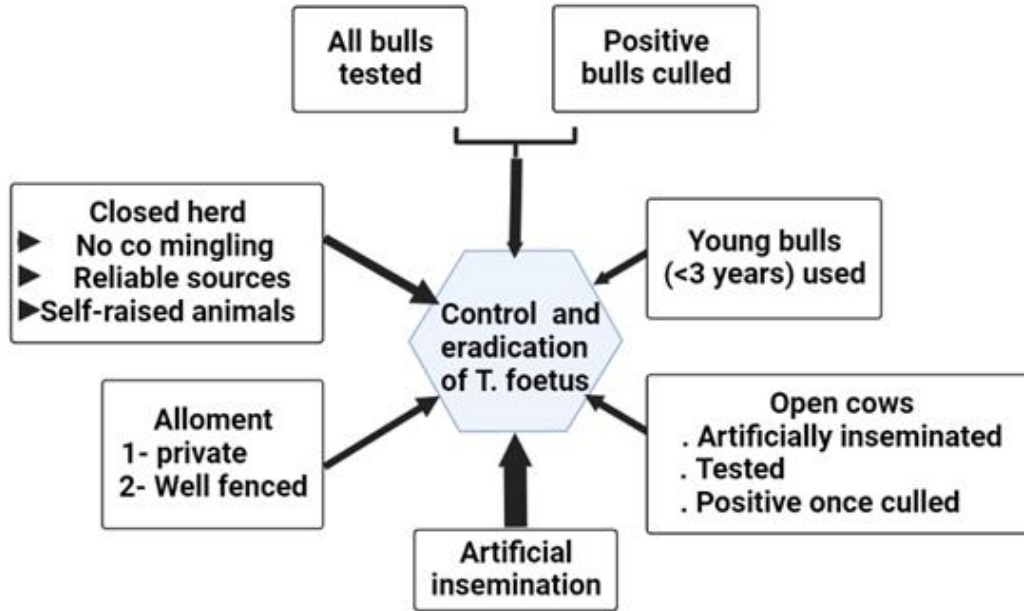
### **1. Causative agent identification**

The tentative diagnosis of trichomoniasis as a reason for reproductive failure on a farm depends on the clinical history, signs of early miscarriage, and recurrent or irregular estrous cycles. However, the infection is confirmed by the manifestation of *T. foetus* in placental fluid, an aborted fetus's stomach contents, vaginal mucus, endometrial washings, inflammatory discharge due to pyometra, or preputial smegma. The most dependable sample to diagnose infected herds is the washings or scrapings of the prepuce or vagina (Corney 2013). The most common diagnostic method is the visualization of motile trichomonads in a saline preparation of the vaginal fluid, which must be done after 10 to 20 minutes of sample collection. Otherwise, the trichomonads will die. The parasites are 10–20 µm long and 5–15 µm wide, near the size of a leukocyte, and may move actively or be observed beating their flagella without the organism's movement (Schwebke and Burgess 2004).

### **2. T. foetus identification by direct examination or in culture**

Many techniques are used to diagnose *T. foetus* with different levels of specificity and sensitivity. An example is the detection of *T. foetus* in Giemsa-stained vaginal smears under the microscope. However, this method cannot detect infections with low parasite numbers. Another way is to grow the parasite in different culture media (Parker et al. 2001), such as Diamond's or Claussen's media, allowing the protozoa to grow *in vitro* until a sufficient number of parasites facilitates detection by light microscopy (Anderson et al. 1994). One drawback of this method is that it takes two to seven days and does not differentiate different *Trichomonas* species (Ginter Summarell et al. 2018). Smegma samples taken either by preputial lavage or scraping seem to be most satisfactory for diagnosing infected bulls and yielding comparable numbers of organisms (Michi et al. 2016). It is preferable to rest bulls sexually for at least seven days before collecting samples to increase the concentration of organisms in the preputial cavity. *T. foetus* trophozoites are microscopically distinguished by their jerky, rolling movement, three anterior flagella, and an undulating membrane (Anderson et al. 1994). Proper diagnosis of *T. foetus* relies on correct collection and handling of samples, suitable growth media and conditions, and proper organism identification by microscopic examination.





**Fig. 3:** An integrated approach for controlling and eradicating *T. foetus* infections. The increasing thickness of the arrows indicates the increasing importance of each approach. Yao C, 2013. Diagnosis of *Tritrichomonas foetus*-infected bulls, an ultimate approach to eradicate bovine trichomoniasis in US cattle? *Journal of medical microbiology* 62(1): 1-9.

In samples where the concentration of organisms is sufficiently high, it is possible to further characterize the organisms by phase contrast microscopy (Skirrow and BonDurant 1990) or staining methods (Lun and Gajadhar 1999) to help visualize vital diagnostic features of *T. foetus*.

### 3. Polymerase Chain Reaction (PCR)

An alternative test that can detect *T. foetus* infection is the polymerase chain reaction (PCR) diagnostic assay, which is of particular value if the number of organisms in the culture remains low (Ginter Summarell *et al.* 2018). The PCR widely detects *T. foetus* DNA using primers such as TF1, TF2, TF3, and TF4. This technique was about 90% sensitive, using TFR3 and TFR4 primers for *T. foetus* detection (Mukhufhi *et al.* 2003; Alobaidii *et al.* 2021). PCR has provided vital improvements over the culture techniques, such as enabling the detection of pseudocysts (non-motile forms) (Pereira-Neves *et al.* 2011), short duration, and high specificity. However, PCR techniques still encounter many challenges (Ginter Summarell *et al.* 2018). To minimize false positive results, the authors utilized a complementary DNA enzyme immunoassay to efficiently discriminate between false-negative amplification products and *T. foetus* DNA (Higgins 2006).

### 4. Serological Tests

Serological tests like mucus agglutination and ELISA can be applied to diagnose *T. foetus*. However, these methods have limited use since they are not highly sensitive or specific, and

bulls do not develop adequate immune reactions for serological diagnoses (Voyich *et al.* 2001).

### Control and Prevention

Strategies for preventing and controlling bovine trichomoniasis depend upon the distinctive epidemiologic characteristics of bovine trichomoniasis. In this sexually transmitted infection, bulls are asymptomatic carriers and are a permanent source of infection, while infections are usually temporary in cows and heifers (Florin-Christensen and Schnittger 2018).

Bovine trichomoniasis is best controlled by proper management (Fig. 3). All bulls in the herd and subsequent replacements should be tested for trichomonads at least three weekly intervals before being used for breeding. Infected bulls should be removed from the herd and replaced with young ( $\leq 2$  years) virgin bulls (Fort *et al.* 2016). Alternatively, AI can control the transmission of *T. foetus* effectively, but a complete change from natural services to AI may not be practical. If the cow herd was exposed to *T. foetus*, cows should be examined, and all those with recent pregnancy loss or pyometra should be culled. A cow herd exposed to trichomoniasis can be divided into two groups; pregnant cows should be observed for abortion, and nonpregnant cows should be rested sexually for at least four months to eliminate the *T. foetus* organisms immunologically from their urogenital tracts (BonDurant and Honigberg 1994). After successful calving, cows in the infected group also should be given sexual rest for a 90-day postpartum interval, or no less than two normal estrous periods after the

breeding season begins, before being moved into a herd with uninfected cattle (Mancebo et al. 1995). Trichomoniasis can be prevented by testing all additions to an established herd. Because testing procedures for individual cows are not well established, additions to established herds should be limited to animals from familiar herds or virgins. If that is not possible, all other female additions should be tested by culture on multiple samples before entering the herd. One commercial "bacterin-type" vaccine and several experimental antigen vaccines (Skirrow and BonDurant 1990) have been shown to induce an immunity *T. foetus* in female cattle vaccinated before breeding.

## Conclusion

Bovine trichomoniasis is a sexually transmitted host-specific disease of cattle that continues to pose a severe economic loss on cattle production due to infertility and abortion. The disease's asymptomatic nature, particularly in the bull, makes diagnosis complex and challenging. The infection can be diagnosed by direct smear examination, culturing, and molecular or serological techniques. The control and eradication of *T. foetus* can only be done by culling positive bulls upon testing.

## REFERENCES

- Adeyeye A et al., 2012. Bovine trichomoniasis: An overview. *Animal Health and Production* 60(1): 7-18.
- Alobaidii WA et al., 2021. Detection of trichomoniasis in cattle in Nineveh province. *Iraqi Journal of Veterinary Sciences* 35(2): 287-290.
- Anderson ML et al., 1994. Protozoal causes of reproductive failure in domestic ruminants. *Veterinary Clinics of North America: Food Animal Practice* 10(3): 439-461.
- Bawa E et al., 1991. Prevalence of bovine campylobacteriosis in indigenous cattle of three states in Nigeria. *Tropical Animal Health and Production* 23(3): 157-160.
- Benchimol M, 2004. Trichomonads under microscopy. *Microscopy and Microanalysis* 10(5): 528-550.
- Benchimol M et al., 2006. Interaction of *Tritrichomonas foetus* and the bovine oviduct in an organ culture model. *Veterinary Parasitology* 140(3-4): 244-250.
- BonDurant R, 1985. Diagnosis, treatment, and control of bovine trichomoniasis. The Compendium on continuing education for the practicing veterinarian. Food and Agricultural Organization of the United Nations.
- BonDurant R and Honigberg B, 1994. Trichomonads of veterinary importance. In: Kreier JP, editor. *Parasitic protozoa*. New York: Academic Press; pp: 111–206.
- BonDurant RH, 2005. Venereal diseases of cattle: natural history, diagnosis, and the role of vaccines in their control. *Veterinary Clinics: Food Animal Practice* 21(2): 383-408.
- Campero CM and Cobo ER, 2006. *Tritrichomonas foetus*: patógenesis de la mortalidad embrionaria/fetal, caracterización de antígenos vacunales y respuesta inmune inducida. *Revista de Medicina Veterinaria-Buenos Aires* 87(2): 47.
- Campero CM et al., 2003. Aetiology of bovine abortion in Argentina. *Veterinary Research Communications* 27(5): 359-369.
- Clark B et al., 1977. Studies on the transmission of *Tritrichomonas foetus*. *Australian Veterinary Journal* 53(4): 170-172.
- Corney B, 2013. Bovine trichomoniasis. *Australian and New Zealand Standard Diagnostic Procedure* 2013: 1-25.
- Danan G and Teschke R, 2015. RUCAM in drug and herb induced liver injury: the update. *International Journal of Molecular Sciences* 17(1): 14.
- de Oliveira JMB et al., 2015. Prevalence and risk factors associated with bovine genital campylobacteriosis and bovine trichomonosis in the state of Pernambuco, Brazil. *Tropical Animal Health and Production* 47(3): 549-555.
- Eaglesome M et al., 1995. A detection assay for *Campylobacter fetus* in bovine semen by restriction analysis of PCR amplified DNA. *Veterinary Research Communications* 19(4): 253-263.
- Florin-Christensen M and Schnittger L, 2018. Introduction into Parasitic Protozoa. In: Florin-Christensen M, Schnittger L, editors. *Parasitic Protozoa of Farm Animals and Pets*. Springer, Cham. pp: 1-10. [https://doi.org/10.1007/978-3-319-70132-5\\_1](https://doi.org/10.1007/978-3-319-70132-5_1)
- Fort M et al., 2016. Evaluation of the performance of bovine trichomonosis control program in La Pampa-Argentina. *Proceedings of the XXI Inter Congress ANEMBE*, Santiago de Compostela, Spain, 2016.
- Ginter Summarell CC et al., 2018. Improvements in *Tritrichomonas foetus* molecular testing. *Journal of Veterinary Diagnostic Investigation* 30(4): 603-608.
- González-Carmona LC et al., 2012. Determination of presence of *Tritrichomonas foetus* in uterine lavages from cows with reproductive problems. *Revista Brasileira de Parasitologia Veterinária* 21: 201-205.
- Goodger W and Skirrow S, 1986. Epidemiologic and economic analyses of an unusually long epizootic of trichomoniasis in a large California dairy herd. *Journal of the American Veterinary Medical Association* 189(7): 772-776.
- Griffiths I et al., 1984. Levels of some reproductive diseases in the dairy cattle of Colombia. *Tropical Animal Health and Production* 16(4): 219-223.
- Güven E et al., 2013. Molecular determination of *Tritrichomonas* spp. in aborted bovine fetuses in Eastern Anatolian Region of Turkey. *Veterinary Parasitology* 196(3-4): 278-282.
- Higgins MR, 2006. Identification of novel virulence factors and mechanisms of pathogenesis from the sexually transmitted protozoan *Tritrichomonas foetus*. Montana State University, Bozeman, Montana.
- Issa R, 2014. Nonpathogenic protozoa. *International Journal of Pharmacy and Pharmaceutical Sciences* 6(3): 30-10.
- Lun Z-R and Gajadhar AA, 1999. A simple and rapid method for staining *Tritrichomonas foetus* and *Trichomonas vaginalis*. *Journal of Veterinary Diagnostic Investigation* 11(5): 471-474.
- Mancebo O et al., 1995. Persistence of *Tritrichomonas foetus* in naturally infected cows and heifers in Argentina. *Veterinary Parasitology* 59(1): 7-11.
- Mardones F et al., 2008. Risk factors associated with *Tritrichomonas foetus* infection in beef herds in the Province of Buenos Aires, Argentina. *Veterinary Parasitology* 153(3-4): 231-237.
- Martin KA et al., 2021. Bovine trichomonosis cases in the united states 2015–2019. *Frontiers in Veterinary Science* 8: 692199.

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- McCool C et al., 1988. Prevalence of bovine venereal disease in the Victoria River District of the Northern Territory: likely economic effects and practicable control measures. *Australian Veterinary Journal* 65(5): 153-156.
- Mendoza-Ibarra JA et al., 2012. High prevalence of *Tritrichomonas foetus* infection in Asturiana de la Montaña beef cattle kept in extensive conditions in Northern Spain. *The Veterinary Journal* 193(1): 146-151.
- Michi AN et al., 2016. A review of sexually transmitted bovine trichomoniasis and campylobacteriosis affecting cattle reproductive health. *Theriogenology* 85(5): 781-791.
- Molina L et al., 2013. Spatial and temporal epidemiology of bovine trichomoniasis and bovine genital campylobacteriosis in La Pampa province (Argentina). *Preventive Veterinary Medicine* 110(3-4): 388-394.
- Mukhufhi N et al., 2003. Evaluation of a PCR test for the diagnosis of *Tritrichomonas foetus* infection in bulls: effects of sample collection method, storage and transport medium on the test. *Theriogenology* 60(7): 1269-1278.
- Parker S et al., 2001. Application of a PCR assay to enhance the detection and identification of *Tritrichomonas foetus* in cultured preputial samples. *Journal of Veterinary Diagnostic Investigation* 13(6): 508-513.
- Parthiban S et al., 2015. Review on emerging and reemerging microbial causes in bovine abortion. *International Journal of Nutrition and Food Sciences* 4(4-1): 1-6.
- Pefanis S et al., 1988. Trichomoniasis and campylobacteriosis in bulls in the Republic of Transkei. *Journal of the South African Veterinary Association* 59(3): 139-140.
- Pereira-Neves A et al., 2011. Identification of *Tritrichomonas foetus* pseudocysts in fresh preputial secretion samples from bulls. *Veterinary Parasitology* 175(1-2): 1-8.
- Pereira-Neves A et al., 2003. Pseudocysts in trichomonads—new insights. *Protist* 154(3-4): 313-329.
- Petropolis DB et al., 2008. The binding of *Tritrichomonas foetus* to immobilized laminin-1 and its role in the cytotoxicity exerted by the parasite. *Microbiology* 154(8): 2283-2290.
- Rae DO et al., 2004. Epidemiology of *Tritrichomonas foetus* in beef bull populations in Florida. *Theriogenology* 61(4): 605-618.
- Rhyan J et al., 1999. Demonstration of *Tritrichomonas foetus* in the external genitalia and of specific antibodies in preputial secretions of naturally infected bulls. *Veterinary pathology* 36(5): 406-411.
- Schlafer DH and Foster RA, 2016. Female genital system. In: Maxie G, editor. *Jubb, Kennedy and Palmer's Pathology of Domestic Animals: Volume 3*, Elsevier; pp: 358-464. <https://doi.org/10.1016/B978-0-7020-5319-1.00015-3>.
- Schuster JP and Schaub GA, 2001. *Trypanosoma cruzi*: the development of estrus cycle and parasitemia in female mice maintained with or without male pheromones. *Parasitology Research* 87(12): 985-993.
- Schwebke J and Burgess D, 2004. Trichomoniasis. *Clinical microbiology reviews* 17(4): 794-803.
- Skirrow S and BonDurant R, 1990. Induced *Tritrichomonas foetus* infection in beef heifers. *Journal of the American Veterinary Medical Association* 196(6): 885-889.
- Skirrow S et al., 1985. Efficacy of ipronidazole against trichomoniasis in beef bulls. *Journal of the American Veterinary Medical Association* 187(4): 405-407.
- Swai E et al., 2005. Prevalence of genital campylobacteriosis and trichomonosis in crossbred breeding bulls kept on zero-grazed smallholder dairy farms in the Tanga region of Tanzania. *Journal of the South African Veterinary Association* 76(4): 224-227.
- Szonyi B et al., 2012. Spatio-temporal epidemiology of *Tritrichomonas foetus* infection in Texas bulls based on state-wide diagnostic laboratory data. *Veterinary Parasitology* 186(3-4): 450-455.
- Van Bergen MA et al., 2006. Molecular epidemiology of *Campylobacter fetus* subsp. *fetus* on bovine artificial insemination stations using pulsed field gel electrophoresis. *Veterinary Microbiology* 112(1): 65-71.
- Voyich JM et al., 2001. Antibody responses of cattle immunized with the Tf190 adhesin of *Tritrichomonas foetus*. *Clinical Diagnostic Laboratory Immunology* 8(6): 1120-1125.
- Waldner C et al., 2013. Application of a new diagnostic approach to a bovine genital campylobacteriosis outbreak in a Saskatchewan beef herd. *The Canadian Veterinary Journal* 54(4): 373.
- Walker R et al., 2003. Comparison of the 5.8 S rRNA gene and internal transcribed spacer regions of trichomonadid protozoa recovered from the bovine preputial cavity. *Journal of Veterinary Diagnostic Investigation* 15(1): 14-20.
- Yao C, 2012. Opportunistic human infections caused by *Tritrichomonas* species: a mini-review. *Clinical Microbiology Newsletter* 34(16): 127-131.
- Yao C, 2015. *Tritrichomonas foetus* infections in female beef cattle with abortion in Wyoming, USA. *JMM Case Reports* 2(2): e000028.
- Yao C et al., 2011. *Tritrichomonas foetus* infection in beef bull populations in Wyoming. *Journal of Bacteriology and Parasitology* 2(5): 104172.
- Yule A et al., 1989. Bovine trichomoniasis. *Parasitology today* 5(12): 373-377.