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Review Article

# Impact of Toxoplasma on Male Fertility: A Review

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**ABSTRACT**

*Toxoplasma gondii* is an obligate intracellular protozoan parasite with a global distribution, infecting humans and animals alike. While much of the research on toxoplasmosis has focused on its effects in women, particularly congenital infections and pregnancy-related complications, emerging evidence indicates that *T. gondii* may also adversely affect male reproductive health. This review explores the epidemiology, life cycle, transmission, and clinical manifestations of toxoplasmosis, emphasizing its potential role in male infertility. Studies from various regions, including Iran, Egypt, India, China, and Brazil, report a higher prevalence of *T. gondii* infection among infertile men compared to fertile controls. The parasite can localize in the male reproductive system, causing inflammation, oxidative stress, and hormonal disturbances that lead to reduced sperm count, poor motility, and abnormal morphology. Experimental studies further suggest that *T. gondii* can directly damage sperm cells, compromising their structural integrity. Additionally, chronic infection may alter testosterone and luteinizing hormone levels, affecting spermatogenesis. Globally, toxoplasmosis prevalence varies widely, from 16% in Asia to over 60% in Africa, influenced by climatic, environmental, and cultural factors. The growing body of evidence supports *T. gondii* as a contributing factor, though not the sole cause, of male infertility. Recognizing toxoplasmosis as a possible infectious determinant of reproductive dysfunction could improve diagnostic approaches and preventive strategies for unexplained infertility cases. Continued multidisciplinary research is essential to elucidate the mechanisms linking *T. gondii* infection and male reproductive impairment.

**Keywords:** Causes of Male infertility , Male Infertility, Parasitic Diseases, Sexual inactivation, *Toxoplasma gondii*.

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## INTRODUCTION

*Toxoplasma gondii* is tissue sporozoites belong to the group of protozoa parasite. This sporozoa infects the wide range of animals including humans and birds. The normal host of this parasite is Cat of the *Felidae* family (Alvarado-Esquivel *et al*., 2011; Ayi *et al*., 2009). Cat is the only host where the oocyst creating sexual stage of Toxoplasma can develops. Toxoplasma invades the mucosal cell of cat small intestine with sporozoite from oocyst or bradyzoites from the cyst that formed along with the tissues. In intestine of cat the sporozoite forms the schizonts and gametocytes. After the sexual fusion of male and female gametes oocyst develops and exit from the host cells to the gut of cat and passes along with the feces from the cat (Afonso *et al*., 2012; Arantes *et al*., 2015; Attia *et al*., 2019). The exited cyst is the infective agents that can re infect another organism and remain infective for 48 hr after releasing from the feces (Barakat *et al*., 2018).

In human most of the times Toxoplasma cause Congenital and Post natal infections. The congenital toxoplasmosis develops to the mother during the pregnancy where she is immunocompromised. The congenital toxoplasmosis is very severe however the postnatal toxoplasmosis is less severe and asymptomatic. This parasite destroys the cells of Reticoloendothelial system directly and enters within the tissues (Benenson *et al*., 1982; Bittencourt *et al*., 2012).

Congenital toxoplasmosis leads the variety of complications like stillbirth, choriorectinitis, hydrocephaly and microcephaly. Prenatal toxoplasmosis is the major causes of blindness are the congenital defects. This parasitic infection also affects the neurons and causes the various serious abnormalities in Central Nervous System (Boothroyd and Grigg, 2002; Boyer *et al*., 2011; Caballero-Ortega *et al*., 2016).

Traditionally, research on the reproductive impact of toxoplasmosis has primarily focused on its well-documented effects on female fertility, particularly the risk of miscarriage and congenital toxoplasmosis. However, growing evidence suggests that *T. gondii*

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may also exert a considerable, though often overlooked, influence on male reproductive health (Dardé, 2008). This introduction aims to explore the multifaceted impact of *T. gondii* infection on male fertility, examining the current understanding of how this widespread parasite may affect sperm parameters, hormone levels, and the overall reproductive potential of infected males. By delving into the mechanisms through which *T. gondii* might compromise male fertility, this discussion seeks to highlight the importance of considering parasitic infections, particularly toxoplasmosis, when investigating cases of unexplained male infertility (Derouin and Pelloux, 2008; Dubey, 2010; Dubey and Jones, 2008).

Male infertility is a complex and often multifactorial condition affecting a significant proportion of couples attempting to conceive, contributing to roughly half of all infertility cases either as a sole cause or a contributing factor. It is broadly characterized by abnormalities in sperm production, function, or transport, leading to a reduced ability to initiate a pregnancy. Common underlying causes range from structural issues like varicoceles (swollen veins in the testicles) and blockages in sperm-carrying ducts, to hormonal imbalances, genetic disorders such as Klinefelter syndrome or Y chromosome microdeletions, and infections that can impair sperm health or obstruct pathways (Elmore *et al*., 2010; Fekadu *et al*., 2018). Lifestyle factors, including smoking, excessive alcohol consumption, drug use, and exposure to environmental toxins, also play a crucial role in compromising male reproductive health (Fereig *et al*., 2016). Diagnosing male infertility typically involves a comprehensive medical history, physical examination, and detailed semen analysis, which evaluates sperm count, motility, and morphology. Further investigations may include hormone testing, genetic screening, and imaging techniques like ultrasound. While some causes of male infertility may be surgically corrected or managed with hormone therapy, Assisted Reproductive Technologies (ART) such as *in vitro* Fertilization (IVF) and Intracytoplasmic Sperm Injection (ICSI) frequently offer effective solutions for couples facing this challenge (Flegr *et al*., 2011; Furtado *et al*., 2013).

## Types of Male Infertility

Male infertility can be broadly categorized based on the underlying cause, often impacting different stages of sperm production, maturation, or transport. One common classification divides it into pre-testicular, testicular, and post-testicular factors.

Pre-testicular infertility refers to issues originating outside the testicles that affect their ability to produce sperm. This often involves hormonal imbalances, such as low levels of hormones from the pituitary gland or hypothalamus (e.g., hypogonadotropic hypogonadism), which are essential for stimulating sperm production (Galván-Ramírez *et al*., 2015). Certain systemic illnesses, medications (like anabolic steroids or chemotherapy), lifestyle factors (e.g., obesity, excessive alcohol, or drug use), and even some environmental toxins can also fall into this category (Figure 1).

Testicular infertility arises from direct problems within the testicles themselves, affecting sperm production. This is a very common type and includes conditions like azoospermia (complete absence of sperm in ejaculate) and oligospermia (low sperm count). Causes can be genetic, such as Klinefelter syndrome or Y chromosome microdeletions, which impair spermatogenesis. Structural abnormalities like varicoceles (swollen veins in the scrotum) are also a significant cause, as they can elevate testicular temperature and compromise sperm development. Infections (e.g., mumps orchitis), testicular trauma, undescended testicles (cryptorchidism), and even prior cancer treatments can lead to testicular damage and subsequent infertility (Gebremedhin and Tadesse, 2015; Gharavi *et al*., 2013).

Finally, post-testicular infertility involves issues after sperm production in the testicles, affecting their transport or delivery. This can be due to obstructive causes, where the tubes carrying sperm (like the epididymis or vas deferens) are blocked, and preventing sperm from being ejaculated. These blockages can result from infections, previous surgeries (e.g., vasectomy), or congenital absence of parts of the reproductive tract (often linked to cystic fibrosis gene mutations) (Hill and Dubey, 2013; Hotez and Kamath, 2009). Ejaculatory disorders, such as retrograde ejaculation (semen entering the bladder instead of exiting the penis) or an ejaculation (absence of ejaculation), also fall under this classification, preventing sperm from reaching the female reproductive tract. In some cases, male infertility is classified as idiopathic or unexplained when no specific cause can be identified despite thorough investigation, highlighting the complexity and ongoing research in this field (Jones *et al*., 2009).

## Unexplained Causes of Male Infertility

The unexplained reason of infertility means does not mean there is no cause, but rather that current diagnostic tools are insufficient to pinpoint it. Emerging research suggests several cryptic or subtle factors may be at play. These include, but are not limited to, subclinical genetic or epigenetic abnormalities impacting gene expression during spermatogenesis, subtle defects in sperm function that are not captured by routine semen analysis (e.g., poor sperm-egg binding ability or DNA fragmentation), and immunological factors where the body mistakenly produces antibodies against its own sperm (Kamani *et al*., 2010). Furthermore, increasing attention is being paid to environmental exposures, such as pollution and endocrine-disrupting chemicals, which may subtly compromise testicular function over time. Chronic, low-grade infections not typically associated with overt reproductive disease, or even metabolic conditions like obesity and insulin resistance, are also being investigated as potential contributors. The ongoing effort to unravel these hidden

mechanisms is crucial for developing more precise diagnostic tools and targeted therapeutic strategies for couples facing the frustration of unexplained male infertility. (Khalil *et al.*, 2020; Khan *et al.*, 2007).

## Epidemiology of Toxoplasmosis

Toxoplasmosis is a zoonotic parasitic disease caused by the obligate intracellular protozoan, *Toxoplasma gondii*. Its epidemiology is complex and widespread, affecting a significant portion of the global population. The disease has a cosmopolitan distribution, with varying prevalence rates influenced by climate, hygiene, and cultural practices. Humans typically acquire the infection through three main routes: consuming undercooked or raw meat containing tissue cysts, ingesting sporulated oocysts from contaminated soil, water, or vegetables, and congenital transmission from mother to fetus. Cats, as the definitive hosts, play a crucial role in the lifecycle by shedding oocysts in their feces, which can then contaminate the environment (Khurana *et al.*, 2017; Li *et al.*, 2018). The prevalence of antibodies to *T. gondii* is high in many parts of the world, with seroprevalence rates often exceeding 50% in certain regions of Europe and Latin America. However, the majority of infections in immunocompetent individuals are asymptomatic, making the true burden of the disease difficult to quantify. The epidemiology is further complicated by the fact that while acute infections are generally mild, reactivation in immunocompromised individuals can lead to severe and life-threatening conditions, such as toxoplasmic encephalitis. Additionally, congenital toxoplasmosis can result in serious birth defects, highlighting the importance of understanding its transmission dynamics (Lüder *et al.*, 2011).

## Sign and Symptoms of Toxoplasmosis

### *Acute Toxoplasmosis in Immunocompetent Individuals*

In healthy people with a strong immune system, the infection is often mild and self-limiting. Symptoms, if they appear, are often similar to the flu or mononucleosis. These can include:

**Fever:** A low-grade fever may be present.

**Lymphadenopathy:** Swollen lymph nodes, particularly in the neck and armpits, are a very common sign.

**Myalgia:** Muscle aches and pains.

**Malaise and Fatigue:** A general feeling of being unwell and tired.

**Headache:** Mild to moderate headaches.

Sore throat.

**Rash:** A skin rash may occur in some cases.

## Reactivated Toxoplasmosis in Immunocompromised Individuals

For people with weakened immune systems (e.g., those with HIV/AIDS, cancer patients on chemotherapy, or organ transplant recipients), a latent infection can reactivate and cause severe, life-threatening disease. The parasite can spread to various organs, most commonly the brain. Symptoms can include:

**Toxoplasmic Encephalitis:** This is a severe form of the disease that affects the brain, leading to:

- Headaches.
- Confusion and altered mental status.
- Poor coordination and muscle weakness.
- Seizures.

**Ocular Toxoplasmosis:** Severe eye infections can cause:

- Blurred or reduced vision.
- Eye pain and redness.
- Seeing "floaters" in your vision.
- In severe cases, it can lead to blindness.

**Pneumonitis:** Lung inflammation, which can present as a cough, fever, and difficulty breathing.

**Myocarditis:** Inflammation of the heart muscle, which can lead to heart failure.

## Congenital Toxoplasmosis

This occurs when a mother acquires the infection during pregnancy and transmits it to her unborn baby. The severity of the symptoms depends on when the infection occurs during the pregnancy, with earlier infections often leading to more severe outcomes. While many infected newborns may be asymptomatic at birth, they are at risk for developing problems later in life (Mahmoud *et al.*, 2022).

## Symptoms at birth (less common but severe)

**Jaundice:** Yellowing of the skin and eyes.

**Hepatosplenomegaly:** Enlarged liver and spleen.

**Rash:** Small red spots or bruising.

**Hydrocephalus:** A buildup of fluid in the brain.

**Intracranial calcifications:** Calcium deposits in the brain, indicating previous damage.

**Microcephaly:** An abnormally small head size.

**Chorioretinitis:** Inflammation and scarring of the retina, which can lead to vision loss.

## Delayed symptoms (may appear months or years after birth)

**Recurring eye infections:** Leading to permanent vision loss or blindness.

Learning disabilities and intellectual disability.

Developmental delays.

Hearing loss.

Seizures.

## Complications of Toxoplasmosis

*Toxoplasma gondii* infection is often asymptomatic in healthy individuals, it can lead to severe and life-threatening complications, particularly in immunocompromised people and newborns. The most devastating complication is toxoplasmic encephalitis in individuals with weakened immune systems, such as those with HIV/AIDS or organ transplant recipients. This condition involves inflammation of the brain, leading to symptoms like headaches, seizures, confusion, and even coma, which can be fatal if left untreated. Another major complication is ocular toxoplasmosis, where the parasite causes inflammation and scarring of the retina, potentially leading to severe vision loss or blindness (Montoya and Liesenfeld, 2004). This can occur in both immunocompromised and immunocompetent individuals, and it is a leading cause of infectious retinitis globally. Congenital toxoplasmosis, transmitted from mother to fetus, can result in severe birth defects, including hydrocephalus, microcephaly, and intracranial calcifications. These neurological complications can lead to developmental delays, intellectual disabilities, and seizures later in life. Additionally, the parasite can cause inflammation of other organs, such as myocarditis (heart inflammation) and pneumonitis (lung inflammation), which can lead to organ failure and death. These complications underscore the importance of early diagnosis and prompt treatment, especially in at-risk populations (Nayeri *et al*., 2017; Nowakowska *et al*., 2006).

## Life Cycle of Toxoplasmosis

The life cycle of *Toxoplasma gondii* is intricate and involves both definitive and intermediate hosts. The definitive hosts are felines, including domestic cats, where the parasite undergoes its sexual reproductive phase. When a cat ingests tissue cysts from an infected intermediate host (such as a mouse or bird), the parasites differentiate and reproduce asexually and sexually in the cat's intestine, forming oocysts. The cat then sheds millions of these unsporulated oocysts in its feces for a period of one to three weeks. These oocysts become infectious, or sporulated, after a few days in the environment.

Humans and other warm-blooded animals serve as intermediate hosts. We can become infected by ingesting these sporulated oocysts from contaminated soil, water, or vegetables. Alternatively, we can acquire the infection by eating undercooked or raw meat that contains dormant tissue cysts, particularly from animals like pigs and sheep (Pappas *et al*., 2009).

Once ingested by an intermediate host, the parasites invade various tissues, where they multiply and form tissue cysts, most commonly found in the brain, heart, and muscle. These dormant cysts can persist for the lifetime of the host (Robert-Gangneux and Dardé, 2012). While the parasite cannot complete its sexual cycle in intermediate hosts, it can be transmitted to a new host when the intermediate host is eaten by a definitive or another intermediate host, thereby continuing the cycle (Figure 2).

## Diagnosis of Toxoplasmosis

The diagnosis of toxoplasmosis is typically a multi-faceted approach, relying on a combination of clinical assessment, laboratory tests, and imaging studies. For most asymptomatic individuals, a diagnosis is not necessary. However, for at-risk groups like pregnant women, immunocompromised patients, or newborns, a definitive diagnosis is crucial. The primary method for diagnosis is serological testing, which detects the

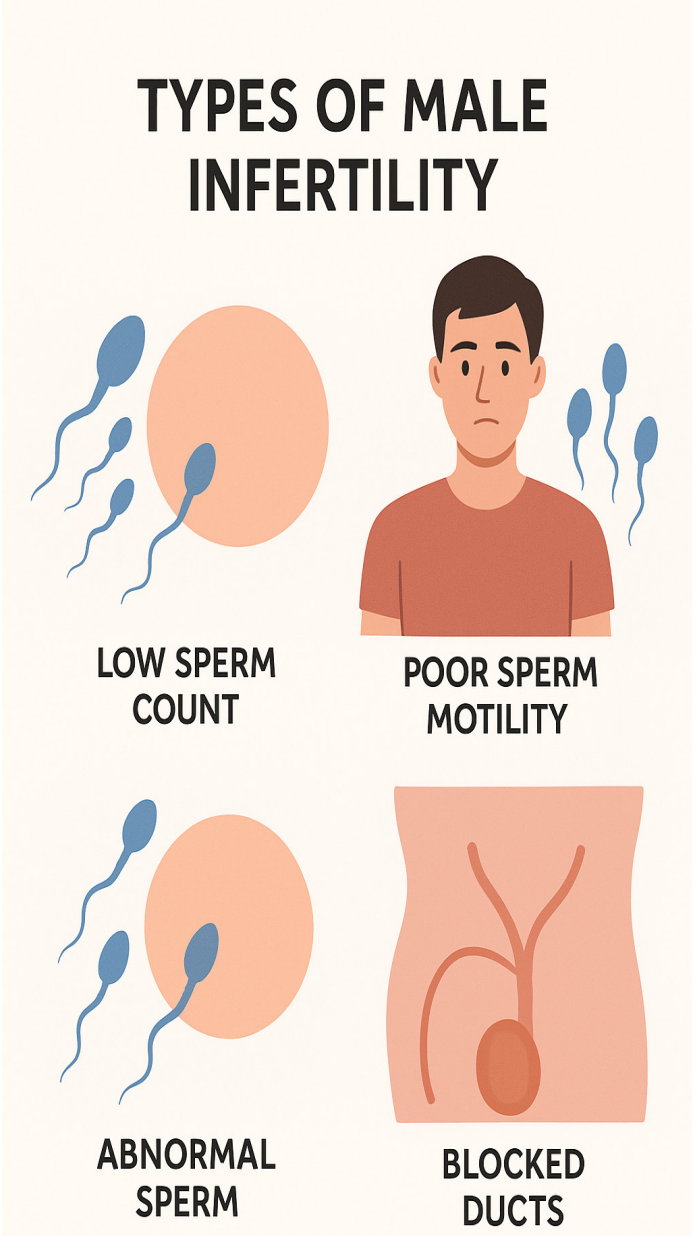


**Figure 1:** Showing the possible factors of Male infertility.

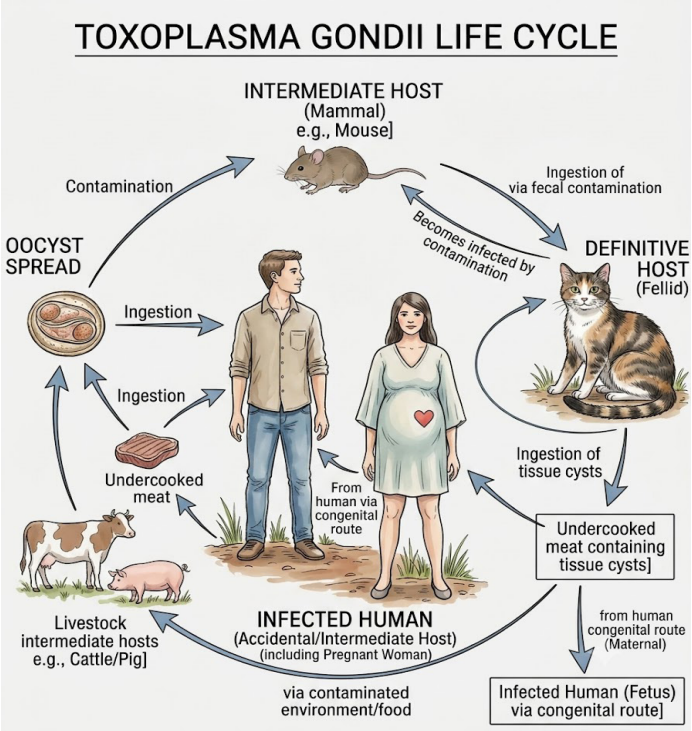


**Figure 2:** Showing the life cycle of Toxoplasma.

presence of *Toxoplasma gondii* antibodies in the blood. The presence of Immunoglobulin G (IgG) antibodies indicates a past exposure, while Immunoglobulin M (IgM) antibodies suggest a recent or active infection. However, interpreting these results requires caution as IgM can persist for months after the initial infection. For more precise diagnosis, particularly in cases of suspected congenital or reactivated toxoplasmosis, tests like the Sabin-Feldman dye test or PCR (Polymerase Chain Reaction) can be used. PCR is particularly valuable as it directly detects the parasite's genetic material in bodily fluids, such as amniotic fluid, blood, or cerebrospinal fluid. In addition to laboratory tests, imaging techniques like MRI or CT scans of the brain are essential for diagnosing toxoplasmic encephalitis, as they can reveal characteristic brain lesions. Furthermore, an ophthalmologist may perform a detailed eye examination to look for signs of chorioretinitis when ocular toxoplasmosis is suspected. The final diagnosis is often confirmed by combining these various diagnostic tools to paint a complete clinical picture (Rostami *et al*., 2018; Rostami *et al*., 2020).

## Prevention of Toxoplasmosis

Preventing toxoplasmosis primarily involves avoiding exposure to the parasite, *Toxoplasma gondii*, through its various transmission routes. A key preventive measure is practicing proper food hygiene. This includes cooking meat, especially pork, lamb, and venison, thoroughly to a safe internal temperature (at least 145°F or 63°C for whole cuts, and 160°F or 71°C for ground meat), as undercooked meat can harbor tissue cysts. Freezing meat at sub-zero temperatures can also effectively kill the parasite. Additionally, washing fruits and vegetables thoroughly before consumption is crucial to remove any oocysts from contaminated soil. When handling raw meat, a separate cutting board and utensils should be used, and all surfaces should be washed with hot, soapy water afterward to prevent cross-contamination (Shapiro *et al*., 2019).

For cat owners, especially pregnant women and immunocompromised individuals, several precautions are essential. Since cats can shed oocysts in their feces, it's recommended to have someone else change the litter box daily, as oocysts only become infectious after 1-5 days. If a person at risk must change the litter, they should wear disposable gloves and wash their hands thoroughly afterward. Keeping cats indoors and feeding them only commercial cat food or cooked table scraps can also help prevent them from becoming infected by hunting. Finally, wearing gloves while gardening or working with soil is an important step, as the soil may be contaminated with cat feces. These simple yet effective measures can significantly reduce the risk of infection (Singh, 2003).

## Prevalence of Toxoplasmosis across the world

Table 1 shows the average seroprevalence rate of *Toxoplasma gondii* infection - that is, the percentage of people who have been exposed to the parasite and have developed antibodies against it - across different continents.

Africa (61.4%) –Highest prevalence: Africa shows the highest seroprevalence, suggesting that a large proportion of the population has been exposed to *Toxoplasma gondii*. This could

be due to factors such as warm and humid climates (which favor the survival of parasite oocysts in soil), poor sanitation, higher exposure to cats (the definitive hosts), and consumption of undercooked meat.

Oceania (38.5%) and South America (31.2%): These regions also show relatively high rates. In South America, particularly, more virulent strains of *T. gondii* circulate, and traditional diets or environmental conditions may increase exposure risk.

Europe (29.6%): Europe has a moderate seroprevalence. The rate is lower than in tropical regions but still significant due to consumption habits such as eating raw or undercooked meat and the presence of domestic cats.

USA/Canada (17.5%): North America shows a lower prevalence, likely reflecting better meat inspection, widespread freezing of meat (which kills the parasite), and improved hygiene practices.

Asia (16.4%): Lowest prevalence: Asia has the lowest average rate, which may be linked to cultural and dietary habits (e.g., cooking meat thoroughly), better sanitation, and climatic conditions less favorable to parasite survival.

## Prevalence of Toxoplasmosis in India

The table presents data on the anti-Toxoplasma IgG seroprevalence-an indicator of previous exposure to *Toxoplasma gondii* infection-across different regions of India Table 2. The findings show considerable geographical variation, influenced largely by climatic and environmental conditions. The highest prevalence is observed in South India (Karnataka) with 37.3%, which may be due to the region's warm and humid climate that supports the survival of *T. gondii* oocysts in soil and water. East India (Assam) shows a moderate prevalence of 21.2%, though some studies from the northeastern states report even higher rates, up to 48%, possibly due to similar climatic and environmental factors. In North India (Delhi NCR), the rate is 19.7%, also within the intermediate range, but variability exists, as some studies among pregnant women have reported rates as high as 45%, reflecting population-specific differences in exposure and lifestyle (Tenter *et al*., 2000; Torrey and Yolken, 2013). The lowest prevalence is recorded in West India (Gujarat) with 8.8%, likely due to its dry climate, which reduces oocyst survival and transmission risk. Overall, the national average seroprevalence is 22.4% among women of reproductive age, suggesting that approximately one in five women in India has been exposed to *T. gondii*. This regional variation underscores the role of environmental, climatic, and cultural factors in influencing the spread of toxoplasmosis across the country (Wang *et al*., 2019).

## Relation of Toxoplasmosis with Male Infertility

Recent research suggests a potential link between *Toxoplasma gondii* infection and male infertility, although the exact mechanisms are still under investigation. Studies in both humans and animal models have shown that the parasite can affect key parameters of male fertility. For instance, some research has found a higher prevalence of anti-Toxoplasma antibodies in infertile men compared to fertile men, suggesting a correlation. The parasite is known to migrate to various organs, including the male reproductive tract, where it can lead to chronic inflammation and direct damage to reproductive tissues, such as the testes (Weiss and Dubey, 2009).

This can result in a range of negative effects on sperm quality, including decreased sperm count, reduced motility, and abnormal morphology. In some animal studies, *T. gondii* has been

**Table 1: Showing the prevalence of Toxoplamosis in different continents**

| Continent | Average Seroprevalence Rate |
|---|---|
| Africa | 61.4% (Highest) |
| Oceania | 38.5% |
| South America | 31.2% |
| Europe | 29.6% |
| USA/Canada | 17.5% |
| Asia | 16.4% (Lowest) |

**Table 2: Showing the prevalence of Toxoplamosis in India.**

| Geographical Region | Representative State/ Area | Anti-Toxoplasma IgG Seroprevalence Rate | Notes |
|---|---|---|---|
| South India | Karnataka | 37.3% (Highest) | Higher prevalence is often linked to warmer, more humid climates which favor oocyst survival. |
| East India | Assam | 21.2% | Intermediate rate. Studies from Northeast India have sometimes reported rates as high as 48%. |
| North India | Delhi NCR | 19.7% | Intermediate rate. Prevalence varies; one study on pregnant women in North India reported 45%. |
| West India | Gujarat | 8.8% (Lowest) | The lower rate is possibly attributed to drier conditions, which negatively impact the survival of the *T. gondii* oocysts in the environment. |
| Overall (National) | Across 4 regions | 22.4% | Average rate from the study on women of reproductive age. |

**Table 3: Showing the data of Male infertility due to Toxoplasmosis.**

| Study Region / Population | Sample Size | Anti-Toxoplasma IgG/IgM Positivity (%) | Observed Effect on Male Fertility Parameters | Key Notes |
|---|---|---|---|---|
| Iran (Infertile men) | 200 | 28.5% | Significantly lower sperm motility and count compared to controls | Strong association between chronic toxoplasmosis and asthenozoospermia (reduced motility). |
| Egypt (Infertility clinic) | 150 | 22.0% | Decreased sperm concentration, increased abnormal morphology | Suggests *T. gondii* may impair spermatogenesis. |
| India (North region) | 120 | 16.4% | Slight reduction in sperm motility; mild oxidative stress markers elevated | Indirect effect through inflammation and oxidative damage. |
| China (General male population) | 300 | 11.8% | Minor decrease in sperm count; no major morphological changes | Chronic infection may influence reproductive hormones. |
| Brazil (Fertility center) | 180 | 25.7% | Lower testosterone and LH levels in infected men | Possible hormonal imbalance linked to chronic infection. |
| Control (Fertile men) | 500 | 8-10% | Normal sperm count, motility, and morphology | Indicates lower prevalence among fertile males. |
| Overall (Average) | - | ~18-22% among infertile men | Reduction in sperm quality, motility, and count | Toxoplasma infection may act as a contributing, not sole, cause of infertility. |

observed to directly attack and damage sperm cells. Additionally, the parasite's presence and the host's immune response may alter hormonal balances, such as testosterone levels, which are critical for sperm production and overall reproductive health. While more extensive research is needed to fully understand this relationship, the accumulating evidence points to *Toxoplasma gondii* as a possible contributing factor to male infertility (World Health Organization [WHO], 2022).

A recent study found that Toxoplasma directly attack on the human sperm, causing them to lose their heads within minutes and that sperm cells are severely impair the male fertility. The parasite is known to invade the male reproductive organs.

The data of Table 3 Shows that *Toxoplasma gondii* infection (especially chronic IgG-positive cases) is more prevalent among infertile men than fertile controls. The infection is associated with impaired sperm quality**,** reduced motility, and sometimes hormonal imbalances**.** Mechanistically, the parasite may induce oxidative stress, testicular inflammation, and altered hormone levels, leading to decreased fertility potential. However, *Toxoplasma gondii* is not a direct or exclusive cause of male infertility - it acts as a contributing factor among multiple infectious, environmental, and physiological influences (Xu *et al.*, 2021; Ybañez *et al.*, 2019).

## CONCLUSION

*Toxoplasma gondii* is a widespread protozoan parasite that can infect a variety of animals, including humans. While the definitive host is the cat, which sheds oocysts in its feces, humans can become infected by ingesting these oocysts from contaminated soil or by eating undercooked meat containing tissue cysts. Although most infections in healthy individuals are asymptomatic or cause mild, flu-like symptoms, the parasite can lead to severe and life-threatening complications in immunocompromised people and newborns. For example, congenital toxoplasmosis can result in serious birth defects such as hydrocephalus and microcephaly, while reactivation in individuals with weakened immune systems can lead to toxoplasmic encephalitis.

Traditionally, research on toxoplasmosis has focused on its impact on female fertility, including the risk of miscarriage and congenital transmission. However, recent evidence suggests that *Toxoplasma gondii* may also affect male reproductive health. Studies have shown a correlation between the presences of anti-*Toxoplasma* antibodies and male infertility. The parasite can migrate to male reproductive organs, causing inflammation and damage to testicular tissues. This can negatively impact sperm quality, leading to reduced count, decreased motility, and abnormal morphology. The parasite has been observed to directly attack sperm cells, causing them to lose their heads. Additionally, the infection and the body's immune response may disrupt hormone levels, such as testosterone, which are essential for sperm production. The accumulating evidence indicates that *Toxoplasma gondii* may be a contributing factor in cases of unexplained male infertility.

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## ABBREVIATIONS

**T. gondii:** *Toxoplasma gondii;* **ART:** Assisted reproductive technologies; **IVF:** *In vitro* fertilization; **ICSI:** Intracytoplasmic sperm injection; **IgG:** Immunoglobulin G; **IgM:** Immunoglobulin M; **PCR:** Polymerase chain reaction; **MRI:** Magnetic resonance imaging; **CT:** Computed tomography; **LH:** Luteinizing hormone; **DNA:** Deoxyribonucleic acid; **HIV:** Human immunodeficiency virus; **AIDS:** Acquired immunodeficiency syndrome.

## CONFLICT OF INTEREST

The authors declare that there is no conflict of interest.

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