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Modulation Of Vaginal Immune Response By Trichomonas Vaginalis With The Correlation Between Bacterial Vaginosis And Infertility

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ABSTRACT

Trichomonas vaginalis (T. vaginalis), a sexually transmitted protozoan, is a major contributor to genitourinary infections, often coexisting with bacterial vaginosis (BV). This parasitic infection not only induces inflammation but also modulates the host immune response, leading to significant reproductive health concerns, including infertility. The interplay between T. vaginalis and BV disrupts the vaginal microbiota, reducing Lactobacillus species and promoting the overgrowth of anaerobic bacteria such as Gardnerella vaginalis. This shift compromises vaginal immunity, allowing chronic infections that contribute to reproductive tract inflammation. T. vaginalis influences the immune system through various mechanisms, including antigenic variation, inhibition of neutrophil apoptosis, and suppression of Th1-mediated responses, leading to prolonged immune tolerance. These immunomodulatory strategies facilitate persistent infections and increase susceptibility to sexually transmitted infections (STIs), including HIV. The parasite also interacts with BV-associated bacterial biofilms, further impairing microbial clearance and exacerbating vaginal dysbiosis. The association between T. vaginalis, BV, and infertility is multifaceted. Chronic inflammation, cervical mucus alterations, and endometrial involvement create a hostile environment for sperm survival and embryo implantation. Additionally, T. vaginalis-induced pelvic inflammatory disease (PID) can lead to tubal scarring and ectopic pregnancies. Given these significant reproductive health implications, routine screening and treatment strategies should be prioritized, particularly in high-risk populations. Future research should focus on understanding the immune mechanisms underlying T. vaginalis and BV interactions to develop targeted therapeutic interventions aimed at mitigating infertility risks associated with these infections.

Keywords: *Trichomonas vaginalis, Bacterial vaginosis. Infertility, pelvic inflammatory disease, Sexually transmitted infections*

1. INTRODUCTION

Trichomonas vaginalis is a parasite that infects the genitourinary tract of both men and women. It is transmitted through sexual contact and can cause trichomoniasis, which may manifest with symptoms or remain asymptomatic. The relationship between the host and parasite is intricate, and it is unlikely that clinical symptoms can be solely attributed to one pathogenic effect. Various factors contribute to the interaction between *T. vaginalis* and host tissues, including contact-dependent and contact-independent mechanisms, as well as the immune response(1). Sexually transmitted infections (STIs) are a significant concern for global health, impacting millions of individuals and posing various risks to reproductive health. *Trichomonas vaginalis*, an extracellular flagellated protozoan, is of particular importance due to its high prevalence and potential consequences. This microscopic parasite primarily colonizes the human genital and urinary tracts, leading to a range of health issues. One area that has received significant attention is the correlation between *Trichomonas vaginalis (T. vaginalis)* infection and reproductive health, specifically the mysterious link to female infertility(2,3).

Bacterial vaginosis (BV) is a complex condition caused by an imbalance in the vaginal microbiota. It is characterized by a decrease in lactobacilli, which are beneficial bacteria, and an overgrowth of various anaerobic and facultative bacteria, including *Gardnerella vaginalis*, *Prevotella spp*, *Bacteroides spp*, *Mobiluncus spp*, gram-positive cocci, and genital mycoplasma. Anaerobic bacteria produce enzymes called sialidases and prolidases, which are believed to play a role in the development of BV(4).

The potential role of *T. vaginalis* in infertility has prompted research efforts to unravel the complexities of this relationship. Furthermore, the interaction between *T. vaginalis* and bacterial vaginosis (BV), a common vaginal infection characterized by an imbalance in microbial flora, has also garnered interest. Understanding the dynamics between T. vaginalis, BV, and their combined impact on fertility is crucial for comprehensive management of reproductive health. This review probes into the multifaceted world of *T. vaginalis*, exploring its prevalence, association with BV, and the current state of knowledge regarding its potential connection to female infertility (5).

2. STUDY SELECTION

Published studies evaluating the association between the presence of bacterial vaginosis (BV) and the risk for acquiring *T. vaginalis* (TV) were included if they met all these inclusion criteria

(1) Study design was observational,

(2) Participants were women of any age,

(3) Exposure was the presence of BV, and

(4) The outcome was incident TV

3. CORRELATIONS

Immune Modulation by Trichomonas vaginalis (6–8)

1. Innate Immune Response and Activation: The first line of defence against *T. vaginalis* is the innate immune system, which includes epithelial cells, macrophages, dendritic cells, and neutrophils.

- **Epithelial Cell Activation:** *T. vaginalis* attaches to the vaginal epithelium, leading to cytokine secretion (e.g., IL-6, IL-8, TNF-α), which promotes inflammation.
- **Neutrophil Recruitment and Activation:** Majority involvement of Neutrophils in controlling the infection by producing reactive oxygen species (ROS) and releasing extracellular traps to capture the parasite. However, *T. vaginalis* can inhibit neutrophil apoptosis, leading to prolonged inflammation.
- **Macrophage Polarization:** The parasite influences macrophage polarization, skewing the response toward an M2-like (anti-inflammatory) phenotype that supports chronic infection.

2. Adaptive Immune Response and Modulation: *T. vaginalis* also affects adaptive immunity, altering both humoral and cell-mediated responses.

- **T-cell Suppression:** The parasite induces a shift from a Th1 to a Th2 response, reducing the effectiveness of parasite clearance. Increased IL-10 production contributes to immune tolerance.
- **B-cell and Antibody Response:** Although infected individuals produce antibodies against *T. vaginalis*, these responses are often non-protective due to antigenic variation of parasite surface proteins.

3. Immune Evasion Strategies: To persist in the host, *T. vaginalis* has developed sophisticated immune evasion mechanisms:

- **Antigenic Variation:** The parasite frequently alters its surface proteins, making it difficult for the immune system to recognize and eliminate it.
- **Complement System Inhibition:** *T.vaginalis* secretes proteases that degrade complement proteins, reducing opsonization and immune clearance.
- **Modulation of Host Microbiome:** The presence of T. *vaginalis* is linked with bacterial vaginosis (BV), as it disrupts the normal vaginal flora, allowing for the overgrowth of pathogenic bacteria like *Gardnerella vaginalis*.

Association Between *T. vaginalis* and BV(9)

Several studies have established a strong correlation between *T. vaginalis* infection and BV, suggesting bidirectional interactions that worsen vaginal health.

1. Disruption of the Vaginal Microbiome: A healthy vaginal microbiome is dominated by *Lactobacillus* species, which produce lactic acid and hydrogen peroxide to maintain an acidic pH and prevent bacterial overgrowth. *T. vaginalis* disrupts

this balance by increasing vaginal pH and promoting the overgrowth of anaerobic bacteria, such as *Gardnerella vaginalis* and *Atopobium vaginae*, which are characteristic of BV.

2. Biofilm Formation and Persistence of BV Bacteria: BV-associated bacteria often form biofilms that protect them from host immune responses and antibiotic treatment. *T. vaginalis* has been shown to interact with these biofilms, enhancing bacterial survival and making BV more difficult to eradicate.

3. Synergistic Immune Evasion: *T. vaginalis* and BV bacteria share immune evasion strategies, including the degradation of complement proteins and suppression of antigen presentation. This synergy contributes to the chronic nature of both infections and their association with complications such as infertility and preterm birth.

Infertility Concerns Associated with *T. vaginalis* and BV(10–13)

1. Impaired Sperm Function and Cervical Mucus Alterations: Inflammation caused by *T. vaginalis* and BV can alter cervical mucus properties, making it hostile to sperm motility and survival. Elevated vaginal pH and the presence of inflammatory mediators can reduce sperm viability and fertilization potential.

2. Fallopian Tube Inflammation and Blockage: Chronic inflammation from persistent *T. vaginalis* and BV infections can lead to salpingitis and tubal scarring. This increases the risk of tubal infertility and ectopic pregnancies.

3. **Endometrial Involvement and Implantation Failure:** *T. vaginalis* infection has been linked to endometrial inflammation, disrupting implantation processes. Persistent immune activation may lead to a hostile uterine environment, contributing to early pregnancy loss.

4. **Increased Risk of Pelvic Inflammatory Disease (PID):** When *T. vaginalis* and BV-related inflammation ascend to the upper reproductive tract, it can result in PID. PID is a leading cause of infertility due to damage to the fallopian tubes and ovarian structures.

4. CONCLUSION

Trichomoniasis is considered one of the neglected parasitic infections by the CDC, and efforts should be made to prioritize public health actions to control its spread. Screening symptomatic women and their partners, as well as treating infected individuals, is crucial in preventing further transmission of the disease. Additionally, screening may be recommended for asymptomatic women at high risk of infection, such as those with a history of STDs or multiple sexual partners.

Trichomonas vaginalis infection has been associated with infertility, with higher infection rates observed in infertile populations compared to fertile groups(10). The pathogen interacts with the host immune response, primarily involving neutrophils, macrophages, and cytokines like IL-8, IL-6, and TNF-α(8). *T. vaginalis* infection can impair sperm quality and trigger immune responses affecting reproductive function(10). Additionally, bacterial vaginosis (BV), characterized by a shift in vaginal microbiota, has been linked to infertility, endometritis, and pelvic inflammatory disease(14). BV is associated with a nearly two-fold increased risk of acquiring *T. vaginalis* infection. The relationship between these conditions and infertility is likely multifactorial, involving inflammation, immune responses, bacterial toxins, and increased susceptibility to sexually transmitted infections(14–16).

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