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Impact of Microorganisms on Human Infertility

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Abstract

Infertility affects millions of couples worldwide, with microbial factors representing a significant yet often underestimated cause. Recent advances in microbiology and molecular diagnostics have expanded our understanding of how bacteria, viruses, fungi, and other microorganisms contribute to both male and female infertility. This review summarizes the current evidence for microbial involvement in infertility, discussing mechanisms, clinical implications, and future research directions. We highlight the importance of early detection and appropriate management of reproductive tract infections to improve fertility outcomes.

Introduction

Infertility, defined as the inability to conceive after 12 months of regular unprotected intercourse, impacts approximately 10–15% of couples of reproductive age globally [1]. While hormonal, genetic, anatomical, and environmental factors are well-documented contributors, infectious and microbial causes have gained increasing attention in recent years [2]. The human reproductive tract harbors a complex microbiome that plays a crucial role in fertility. Disruption of this delicate microbial ecosystem—through infection or dysbiosis—can result in adverse reproductive outcomes for both sexes [3,4]. This review provides the current understanding of how microbes contribute to infertility, focusing on key pathogens, mechanisms, diagnostic advances, and potential therapeutic strategies.

Microbes and female infertility

Vaginal microbiota and dysbiosis

The vaginal microbiota is dominated by Lactobacillus species, which help maintain an acidic environment and inhibit pathogen colonization [5]. Bacterial Vaginosis (BV), characterized by a reduction in Lactobacillus and overgrowth of anaerobic bacteria such as Gardnerella vaginalis and Atopobium vaginae, is associated with adverse reproductive outcomes including subfertility and increased risk of Pelvic Inflammatory Disease (PID) [6,7]. Studies have shown that BV increases susceptibility to Sexually Transmitted Infections (STIs) and may impair embryo implantation through endometrial inflammation [8].

Sexually transmitted infections

Chlamydia trachomatis and Neisseria gonorrhoeae are leading causes of tubal factor infertility in women [9]. These pathogens can ascend from the lower to the upper genital tract, causing PID, tubal scarring, and occlusion [10,11]. Notably, C. trachomatis infection is often asymptomatic, leading to delayed diagnosis and irreversible reproductive damage [12]. Mycoplasma genitalium and Ureaplasma urealyticum have also been implicated in infertility, although their precise role remains under investigation [13].

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Viral and fungal infections

Human Papillomavirus (HPV) and Herpes Simplex Virus (HSV) infections have been associated with altered cervical mucus properties and recurrent pregnancy loss, though causality is still debated [14,15]. Additionally, chronic endometritis, frequently caused by microbial imbalance or infection, can impair endometrial receptivity and embryo implantation [16]. While less common, vulvovaginal candidiasis (caused by Candida albicans) may contribute to infertility through immune-mediated mechanisms and mucosal barrier disruption [17].

Microbes and male infertility

Seminal microbiome and dysbiosis

The male reproductive tract is not sterile; the seminal microbiome includes both commensal and pathogenic species [18]. Dysbiosis, particularly the presence of Escherichia coli, Staphylococcus aureus, and Ureaplasma spp., has been associated with reduced sperm motility, abnormal morphology, and increased DNA fragmentation [19,20]. These alterations can negatively impact fertilization and early embryonic development.

Genital tract infections

Genital tract infections such as epididymitis, prostatitis, and orchitis are common causes of male infertility [21]. Chlamydia trachomatis, N. gonorrhoeae, and Ureaplasma urealyticum are frequently implicated [22]. Mumps virus, which can cause orchitis, is a well-known cause of acquired male infertility due to testicular atrophy and impaired spermatogenesis [23].

Viral and other microbial causes

Human Immunodeficiency Virus (HIV) and hepatitis B and C viruses may indirectly affect fertility by causing chronic inflammation, systemic illness, or through the effects of antiviral therapy [24]. Additionally, Human Papillomavirus (HPV) DNA has been detected in semen, with some studies suggesting an impact on sperm quality and male fertility [25].

Mechanisms of microbial-induced infertility

Microbes can affect fertility via multiple mechanisms:

1. Inflammatory Damage: Infection triggers inflammatory responses, leading to tissue damage, fibrosis, and scarring, especially in the fallopian tubes or epididymis [26].
2. Immune Modulation: Chronic infections may disrupt immune tolerance at the maternal-fetal interface, increasing the risk of miscarriage or implantation failure [27].
3. Direct Cellular Injury: Certain pathogens invade and damage epithelial or germ cells, impairing gamete production and function [28].
4. Alteration of Mucosal Barriers: Dysbiosis weakens natural defenses, facilitating pathogen entry and persistence [29].
5. Hormonal Disruption: Chronic infections can interfere with the hypothalamic-pituitary-gonadal axis, affecting hormonal balance and reproductive function [30].

Diagnostic approaches

Accurate diagnosis of microbial infertility factors is critical. Traditional methods include culture and microscopy, but these have limited sensitivity, especially for fastidious organisms [31]. Molecular techniques such as Polymerase Chain Reaction (PCR) and Next-Generation Sequencing (NGS) have greatly improved detection rates for both symptomatic and asymptomatic infections [32]. Testing for C. trachomatis, N. gonorrhoeae, Mycoplasma, and Ureaplasma species is recommended in infertility evaluations, especially for patients with tubal or unexplained infertility [33]. Assessment of the vaginal and seminal microbiome using NGS is a promising tool for identifying dysbiosis associated with infertility [34]. However, standardized protocols and interpretation guidelines are still being developed.

Treatment and clinical implications

Treatment of infection-related infertility typically involves targeted antibiotic or antiviral therapy. Early intervention is crucial to prevent irreversible damage, particularly in cases of PID or orchitis [9,21]. For some pathogens, partner treatment and prevention of reinfection are essential [35].

Restoring a healthy microbiome—through probiotics, prebiotics, or even fecal and vaginal microbiota transplantation—is an emerging area of interest [36]. Preliminary studies suggest that probiotic supplementation may improve reproductive outcomes in women with BV or endometrial dysbiosis [37]. More research is needed to establish efficacy, safety, and optimal protocols.

Future directions and research needs

Despite advances, many questions remain about the role of microbes in infertility. The complex interplay between host immune factors, microbiome composition, and reproductive outcomes requires further investigation [38]. Longitudinal studies and randomized clinical trials are needed to determine the causality, optimal diagnostic strategies, and effective interventions for microbe-associated infertility.

The impact of the reproductive tract microbiome on Assisted Reproductive Technology (ART) outcomes is a promising field, with evidence suggesting that microbial profiles may predict success rates in In Vitro Fertilization (IVF) and embryo implantation [39]. Personalized medicine approaches targeting the microbiome could revolutionize infertility care in the coming years.

Conclusion

Microbial factors are significant contributors to infertility in both men and women. Infections, dysbiosis, and chronic inflammation of the reproductive tract can impair gamete quality, disrupt reproductive tract function, and reduce the chances of successful conception. Advances in molecular diagnostics and microbiome research have enhanced our understanding of these complex interactions. Early detection, appropriate treatment, and restoration of microbial balance hold promise for improving fertility outcomes. Ongoing research will be essential to translate these insights into clinical practice.

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