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Impact of *Chlamydia trachomatis* on Male Fertility

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Introduction

In male, fertility relies upon the normal function of male reproductive organs, normal spermatogenesis, normal parameters of semen. Spermatogenesis comprises 3 phases: self-renewal of spermatogonial stem cells and differentiation, spermiogenesis and spermatocytes meiotic division. Spermatogenic cells are highly sensitive to infections of pathogenic microorganism or environmental factors [1].

Bacteria, parasites, viruses and fungi are some important clinical pathogens that may cause the microbial infections of male reproductive tracts, and also interfere with the reproductive function in male [2]. 15% of the cases of male infertility are due to reproductive tract inflammations and infections [3]. Several organs of the male reproductive system, such as male accessory sex glands, testis and epididymis all can be infected by pathogenic microorganism [2].

Male reproductive tract infections with *Chlamydia trachomatis* (*C. trachomatis*) is one of the most prevalent sexually-transmitted bacterial diseases. In men, infection by *C. trachomatis* can result in urethritis, prostatitis, epididymitis and orchitis [4]. Sobinoff et al. used the animal model to investigate the effects of *C. trachomatis* infection on spermatogenesis of mice, data showed that *C. trachomatis* infection not only disrupted structure of the seminiferous tubules, caused loss of germ cells at 4 and 8 week after infection, but also injuried severely the testicular tubules with only Sertoli cells. Further examination showed that the DNA repair, and apoptosis in spermatogonial cells and damaged germ cells were evident in atrophic tubules; Sertoli cells with abnormal morphology that were casp3 positive in tubules of infected mice; sperm count, motility and morphologically normal spermatozoa were decreased in infected mice [5]. Owing to few literatures about human testis infected by *C. trachomatis*, the real effects of *C. trachomatis* on spermatogenesis of human testis is not clear yet, this study projected a light on the harmness of *Chlamydia trachomatis* on mammal spermatogenesis.

As to the prostate, *C. trachomatis* is considered a factor of the chronic prostatitis. *C. trachomatis* can cause scarring in the prostatic and ejaculatory ducts, resulting in decreased seminal volume. Many of these infected men present with severe oligozoospermia or azoospermia [6]. In 2014, Cai et al. performed an investigation in patients suffering from chronic prostatitis-related symptoms from a centre of sexually transmitted diseases (STD) in Italy, data indicated that co-infection with human papillomavirus and prostatitis-related symptoms attributable to *C. trachomatis* infection play a key role in decreasing male fertility, particularly related to sperm motility and morphology [7].

Urogenital infections can affect spermatozoa at different levels of their development, maturation and transport [8]. *C. trachomatis* infections also may affect male fertility by directly damaging the sperm, because sperm parameters, acrosome reaction capacity and DNA fragmentation are injured by chlamydial infection [9]. Another study indicated that semen of infertile men with *C. trachomatis* infection when compared to uninfected men showed statistical decrease in semen count and rapid decrease in progressive motility of spermatozoa. Flow cytometry analyses demonstrated a significant increase rate of low $\Delta\Psi m$ and caspase 3 activation in infertile men positive for *C. trachomatis*. These data indicated the negative direct impact of *C. trachomatis* infection on sperm fertilizing ability [10].

In brief, the purpose of this editorial is to review the deleterious effects of *C. trachomatis* on the male fertility and male genital tract. An increased awareness of harmness induced by *C. trachomatis* infection, an increasing health prevention consciousness and early medicine treatment are the key points to reduce the harmness induced by *Chlamydia trachomatis* in human.

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