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Sperm Cell Damage Induced by Urogenital Tract Infections

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Opinion

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Opinion

Male infertility has traditionally been diagnosed by microscopic assessment of concentration, motility and morphology of sperm in the ejaculate. These tests are essential to provide the fundamental information on sperm quality. However, evidence based medicine shows that sperm DNA fragmentation (SDF) tests can also differentiate fertile from infertile males.

Damaged DNA has been observed in testicular, epididymis and ejaculated sperm. Various internal causes of DNA fragmentation are abortive apoptosis, deficiencies in recombination or oxidative stress, among others. Damage can also occur due to extrinsic factors, the so called iatrogenic damage, and can be a result of storage temperatures, extenders used, handling conditions, lapse of time after ejaculation, infections, reaction to medicines, or post-testicular oxidative stress.

The significance of urinary tract infections (UTIs) by bacteria for sperm quality has been increased in the recent years. As UTIs and inflammations are thought to be responsible for up to15% of the cases of male infertility. There are many reports providing evidence that bacterial UTIs could contribute to deterioration of sperm quality, especially in infertile men. The available date mostly concerns to the well-known causative agents of UTIs such as *Escherichia coli, Staphylococcus aureus, Enterococcus faecalis, Ureaplasma urealyticum, Mycoplasma hominis,* and *Chlamydia trachomatis*. During bacterial semen infection, sperm motility and normal morphology loss may be consequences of adhesion phenomena and sperm agglutination. The sperm surface is rich in glycoproteins and is thus susceptible to bacteria– spermatozoa interactions at the receptor–ligand level [1,2].

Bacterial infections can directly and indirectly influence the sperm parameters; directly by reducing sperm viability and motility as well as by changing sperm morphology, and indirectly through oxidative stress, immune reactions, induction of apoptosis or necrosis and secretory dysfunction of male accessory glands.

The harmful effects of numerous microbial pathogens on spermatozoa not only result from the tight adhesion of interacting cells, but also from the expression of other surface virulent factors, such as lipopolysaccharides (LPS), cytotoxic necrotizing factor, α-haemolysins, β-haemolysins, and from the release of soluble spermatotoxic factors such as sperm immobilization factor (SIF) [3,4]. For example, *E. coli* haemolysins might be involved in the molecular mechanism that ultimately alters the membrane integrity [5]. In turn, SIF can inhibit sperm motility and viability by decreasing mitochondrial ATPase activity [4]. However, a loss of sperm motility concomitant with the integrity of the sperm mitochondrial membrane potential ($\Delta\psi m$) during experimental in vitro semen infection has also been reported in the presence of bacteria that do not produce SIF, such as known pathogenic *C. trachomatis* and the conditionally pathogenic *S. haemolyticus* and *B. ureolyticus* [6]. However, the most critical mechanism leading to the sperm death pathway occurring in ejaculated spermatozoa during genitourinary tract inflammation/infection is related to apoptosis.

With respect to the relationship between sperm apoptotic markers and the fertility potential of spermatozoa, the published data are still controversial. However, the vast majority of authors agree that the determination of apoptotic markers in spermatozoa, usually measured by flow cytometer, has better potential to predict fertility in clinical practice than conventional semen parameters.

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