Editorial

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The relationship between oxidative stress and sperm physiology

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Editorial

Oxidative stress is a physiological mismatch between ROS production and the body's ability to detoxify or repair damage.[1] ROS are crucial for cell signaling and pathogen defense, however excessive ROS generation can damage cells.[2] Oxidative stress has been studied in sperm physiology. This editorial examines how oxidative stress affects sperm function and fertility.Male fertility relies on spermatogenesis, which creates numerous spermatozoa cells in the testis.[3] ROS can damage spermatogenic cells, while oxygen is necessary for aerobic metabolism.[4] Significant positive link has been documented between ROS levels and proportion of spermatozoa with defective heads, acrosomes, midpieces, cytoplasmic droplets, and tail defects.[5] Within semen there are two principal sources of production of free radicals; leukocytes and sperm. The vast majority of semen specimens contain leukocytes, with neutrophils being the predominant leukocyte type.[6] As the production of ROS is one of the principal mechanisms by which neutrophils destroy pathogens, it is not surprising that seminal leukocytes have the potential to cause oxidative stress.[7] However, a link between the presence of leukocytes in semen and male oxidative infertility is still under debate. Sperm and critical stages of spermiogenesis are vulnerable to ROS-induced damage due to factors such as chromatin condensation, lack of DNA repair mechanisms, high poly unsaturated fatty acid content in the membrane, ROS production during epididymis, and low cytoplasmic antioxidant enzyme levels.[8] Dietary deficits are associated to sperm oxidative damage by various studies. The Age and Genetic Effects in Sperm (AGES) study compared sperm quality with self-reported dietary consumption of antioxidants and minerals (vitamins C and E, β-carotene, folate, and zinc) in 97 healthy non-smokers.[9] Vitamin C intake correlated with sperm concentration and vitamin E with total progressively motile sperm in this study. This is also consistent with past findings that seminal plasma vitamin E levels improve motile sperm percentage. The AGES study found no link between poor antioxidant consumption and sperm DNA damage.[9] This was surprising as prior researchers had connected low seminal plasma vitamin C to sperm DNA damage.Seminal antioxidants such β-mercaptoethanol, protein, vitamins E and C, cysteamine, cycteine, taurin, and hypotaurin limit the quantity of seminal ROS.[10] In fact, these chemicals improve sperm parameters and embryo development. Seminal leukocytes and aberrant spermatozoa generate most ROS in human ejaculate. Morphologically defective spermatozoa produce ROS extensively.[11] Among sperm abnormalities, leftover cytoplasm or cytoplasmic droplet may produce the greatest ROS. Sertoli cells normally omit these extra cytoplasms during spermiogenesis.[12] This condition causes immature, functionally impaired spermatozoa. Studies have shown that cytoplasmic droplets generate ROS via "glucose-6-phosphate dehydrogenase" (G6PD), which induces high ROS production via two pathwaysSperm DNA can be directly damaged by free radicals targeting purine, pyrimidine, and deoxyribose bases. Sperm DNA is protected from free radicals by protamines.[13] However, infertile men often lack protamination, making sperm DNA sensitive to ROS assault). Alternatively, free radicals can cause sperm apoptosis and caspase-mediated DNA destruction.[14] Using terminal deoxynucleotidyl transferase-mediated dUTP nick-end labeling (TUNEL), sperm chromatin structure assay (SCSA), and 8-hydroxydeoxyguanosine detection, several researchers have linked oxidative stress to sperm DNA damage.[15]

Functional role of ROS in male reproduction

The body produces extremely reactive oxygen molecules called reactive oxygen species (ROS) from many cellular processes. ROS are frequently associated with oxidative stress and cell damage, but they also play complex roles in male reproduction.[16] Several studies show that spermatozoa need small quantities of ROS to fertilize oocytes. Moreover, spermatozoa require tiny quantities of ROS for capacitation, hyperactivation, motility, acrosome response, and fertilization.[17] ROS and other spermatozoa components increase intracellular cAMP, which activates protein Kinase A.[18] Capacitation is driven by tyrosine phosphorylation, which increases with these changes. Increased cAMP levels promote sperm motility or hyperactivation. Capacitation also makes the acrosome membrane fragile, releasing hydrolytic enzymes such acrosin and allowing sperm to bind oocyte.[19] ROS may regulate spermatozoa nuclear maturation. These reactive agents create lipid peroxides, which may offer a substrate for GPX4, oxidizing nuclear proteins and facilitating nuclear condensation. GPX4 can utilise nuclear protein thiols instead of glutathione.[20] Additionally, men with leukospermia-associated oxidative stress have dramatically lower GPX activity in their spermatozoa. Finally, glutathione reductase regenerates reduced glutathione for GPX action. Selective GTR inhibition lowers reduced glutathione for GPX activity, exposing sperm to oxidative stress. GPX, GTR, and glutathione work together to protect sperm from oxidative damage.[21] Good reproductive health requires a careful ROS equilibrium. Diet, smoking, alcohol, and environmental pollutants can affect ROS production and male fertility. Antioxidants, which neutralize ROS, are also being studied for oxidative stress-related male infertility. Understanding ROS' complex roles in male reproduction is crucial to creating successful reproductive treatments.[22]

Conclusion

A healthy male reproductive system balances ROS generation and antioxidant activity. Overproduction of ROS in semen can damage sperm or seminal plasma antioxidant defenses and produce oxidative stress. Although reactive oxygen species are necessary for capacitation

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and acrosome reaction, increased ROS and decreased antioxidant defense cause OS status, which leads to sperm membrane lipid peroxidation, reduced motility, sperm DNA damage, poor pregnancy and ART outcomes, and increased offspring genetic disease risk. OS has many diagnostic and treatment options. Oxidative stress damages sperm DNA/chromatin. Intratesticular, post-testicular, and external factors like alcohol, smoking, varicocele, diabetes, and others have been linked to increased ROS and sperm DNA damage, which can affect male fertility. Antioxidant therapy is thought to improve sperm quality and male fertility by lowering oxidative stress. However, OS test selection, antioxidant treatment kind and duration, and patient group definition are not agreed upon. To overcome these restrictions and boost fertility, more research is needed.

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Conflicts of interest

The author declares no conflicts of interest.

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