THE SCIENCE WORLD
A Monthly e Magazine
ISSN:2583-2212
February, 2023; 3(02), 276-280

Popular Article

Effect of oxidative stress on fertility and therapeutic effect of antioxidants

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https://doi.org/10.5281/zenodo.7685603

Introduction

The imbalance between the generation of reactive oxygen species (ROS) and the antioxidant system's beneficial role in neutralizing and removing them causes oxidative stress. An overabundance of ROS generates a pathogenic reaction that damages cells and tissues. Natural antioxidant defense mechanisms exist to reduce the damaging effects of free radicals by neutralizing them and mending damaged cells. Reactive oxygen species (ROS) and antioxidants must coexist in this delicate and challenging equilibrium for healthy cell activity (Pham et al., 2008). ROS are produced as end products when cells use oxygen to survive. A specific level of ROS is necessary for the progression of normal cell processes, which includes reproductive cells and tissue. On the other

hand excess levels become pathological, causing DNA damage and even apoptosis. High ROS levels could be caused by either endogenous or external causes. Environmental pollution, smoking, alcohol, poor diet, and obesity are the most prevalent external sources of oxidative stress in reproductive cells. Endogenous causes include infections, chronic and autoimmune illnesses (Tremellen, 2008). ROS can be directly neutralised by antioxidants, and the harm they cause can be repaired. The body contains both enzymatic and nonenzymatic natural antioxidants (Fig. 1). Catalase, superoxide dismutase, glutathione reductase, and glutathione peroxidase are antioxidant enzymes. Ascorbic acid (vitamin C), alpha-tocopherol (vitamin E), ferritin, and transferrin are further nonenzymatic antioxidants. (Gupta et al., 2007).

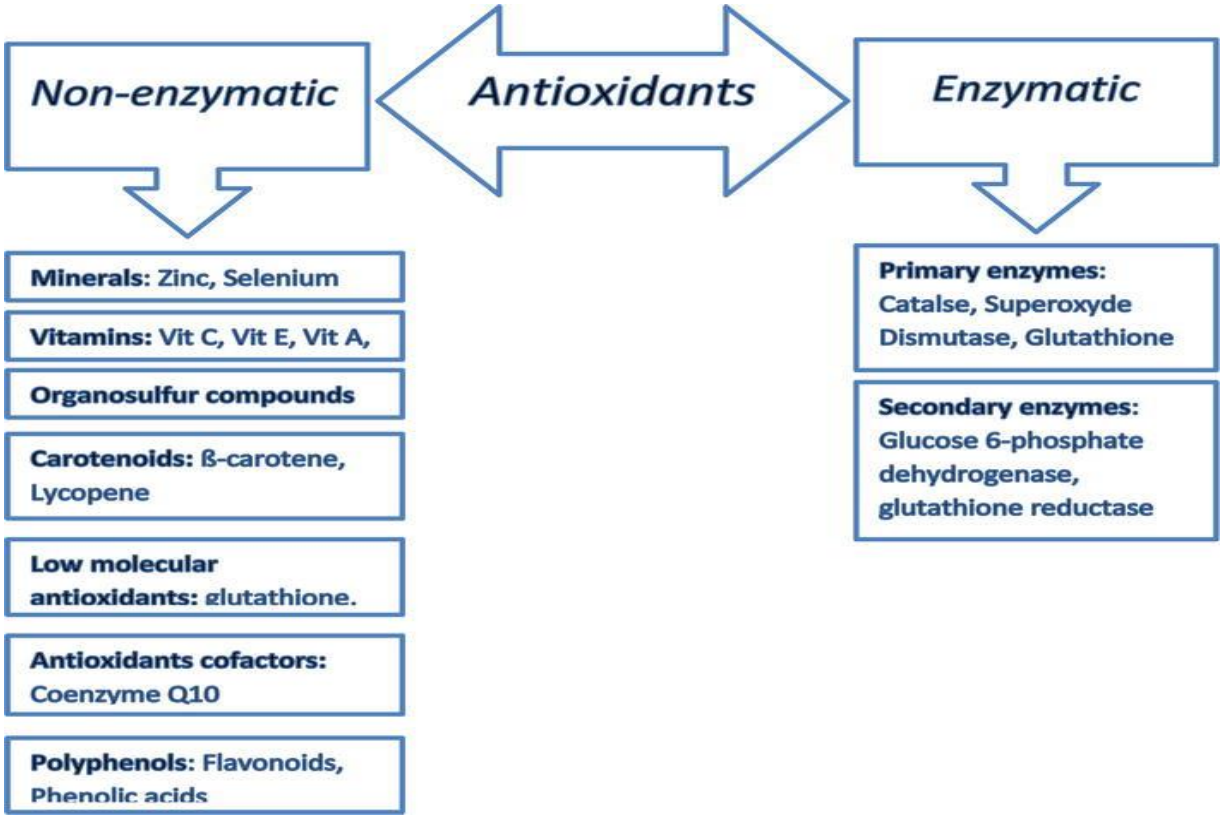

Fig. 1. Enzymatic and nonenzymatic natural antioxidants (Amor et. al., 2021).

Female subfertility

It has been proposed that cytochrome P450 and the corpus luteum are two major sources that influence the synthesis of ROS. Variable concentrations of ROS and antioxidants have a significant impact on the maturation of oocyte development, meiosis I and II (Behrman et al., 2001). OS has an

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immediate impact on the egg, embryo, and implantation by causing DNA damage, cellular protein oxidation, and cell membrane lipid peroxidation. Moreover, endometriosis, hydrosalpinges,

polycystic ovarian syndrome (PCOS), and unexplained subfertility are linked to oxidative stress (Agarwal et al., 2012). There are various potential modes of action for supplemental antioxidants. Improved endometrial blood flow, reduced hyperandrogenism, reduced insulin resistance, fertile cervical mucus, and an impact on prostaglandin synthesis and steroidogenesis are all advantages for female fertility (Thomson et al., 2012) In vitro research have convincingly demonstrated that ovarian glutathione reduction promotes antral follicle atresia, reflecting the great sensitivity of antral follicles to oxidative stress. The same was observed for the fertilisation and embryonic development processes (Mulla et al., 2018). ROS produced by psychological stress will further disrupt granulosa cell activity, and a decrease in estradiol levels will diminish the quality and number of recovered oocytes.

Male subfertility

Seminal plasma and spermatozoa both include antioxidant mechanisms that can detoxify the damaging effects of ROS. The imbalance between total antioxidant capacity and ROS production in seminal fluid is indicative of oxidative stress and is strongly associated with male infertility (Sharma et al., 1999). Spermatozoa themselves are a prominent producer of ROS, and physiological amounts of ROS are required for a variety of sperm activities including capacitation, maturation, and hyperactivation. An oversupply of ROS triggers a pathogenic reaction that damages cells and tissues. Spermatozoa are particularly vulnerable to the harmful effects of ROS due to the high concentrations of unsaturated fatty acids in their cell membrane that can be oxidised (lipid peroxidation) and there will be low concentrations of the enzyme that can neutralise ROS in the cytoplasm. The process of lipid oxidation causes structural DNA damage, cell apoptosis, deactivation of cellular enzymes, loss of membrane integrity, and an increase in the permeability of the membrane. As a result, sperm count and activity are lowered, motility is diminished, and morphology is abnormal (Fig. 2) (Sanocka et al., 2005; Schuppe et al., 2008). A varicocele, an abnormal expansion of the pampiniform venous plexus in the scrotum, may be a significant endogenous source of ROS in the male reproductive system. High quantities of ROS are produced as a result of the altered microcirculation and elevated scrotal temperature, which harm and even cause the apoptosis of germ cells (Zini et al., 2011).

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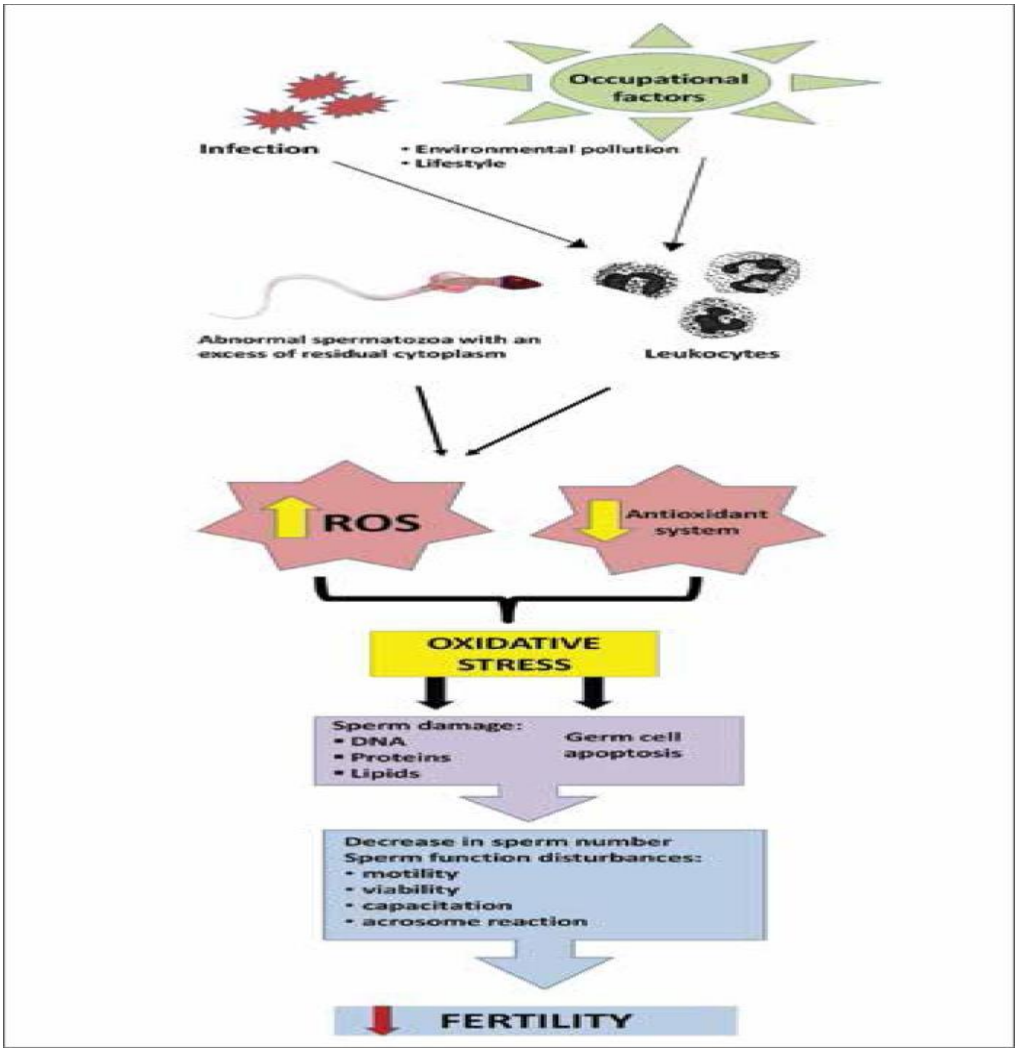

Fig. 2. Effect of oxidative stess on spermatozoa (Walczak et al., 2013).

Conclusion

There are indications that oxidative stress is a significant factor in both male and female subfertility. Antioxidants might make for a cheap remedy to improve fertility outcomes. Sufficient evidence exists that oxidative stress can jeopardize fertilization, implantation and embryo viability.

References

Agarwal, A., Aponte-Mellado, A., Premkumar, B. J., Shaman, A., & Gupta, S. (2012). The effects of oxidative stress on female reproduction: a review. *Reproductive biology and endocrinology : RB&E*, *10*, 49. https://doi.org/10.1186/1477-7827-10-49

Aitken J, Fisher H. Reactive oxygen species generation and human spermatozoa: the balance of benefit and risk. Bioessay 1994:16.259-67

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Published 28.2.2023

Amor, H., Shelko, N., Mohammed, M., Michael Jankowski, P., & Eid Hammadeh, M. (2021). Role of Antioxidants Supplementation in the Treatment of Male Infertility. Antioxidants - Benefits, Sources, Mechanisms of Action. doi: 10.5772/intechopen.95891

Behrman, H. R., Kodaman, P. H., Preston, S. L., & Gao, S. (2001). Oxidative stress and the ovary. *Journal of the Society for Gynecologic Investigation*, *8*(1 Suppl Proceedings), S40–S42. https://doi.org/10.1016/s1071-5576(00)00106-4

Gupta S, Agarwal A, Banerjee J, Alvarez JG. The role of oxidative stress in spontaneous abortion and recurrent pregnancy loss: a systematic review. Obstet Gynecol Surv 2007;62:335–47

Mulla, A. , Fazari, A. , Elkhouly, M. and Moghaddam, N. (2018) Role of Antioxidants in Female Fertility. *Open Journal of Obstetrics and Gynecology*, **8**, 85-91. doi: 10.4236/ojog.2018.82011.

Pham-Huy, L. A., He, H., & Pham-Huy, C. (2008). Free radicals, antioxidants in disease and health. *International journal of biomedical science : IJBS*, *4*(2), 89–96

Sanocka–Maciejewska D, Ciupinska M and Kurpisz M. Bacterial infection and semen quality. J Reprod Immunol. 2005; 67: 51–56. 15.

Schuppe HC, Meinhardt A, Allam JP, Bergmann M, Weidner W and Haidl G. Chronic orchitis: a neglected cause of male infertility? Andrologia. 2008; 40: 84–91

Sharma, R. K., Pasqualotto, F. F., Nelson, D. R., Thomas, A. J., Jr, & Agarwal, A. (1999). The reactive oxygen species-total antioxidant capacity score is a new measure of oxidative stress to predict male infertility. *Human reproduction (Oxford, England)*, *14*(11), 2801–2807. https://doi.org/10.1093/humrep/14.11.2801

Thomson, R. L., Spedding, S., & Buckley, J. D. (2012). Vitamin D in the aetiology and management of polycystic ovary syndrome. *Clinical endocrinology*, *77*(3), 343–350. https://doi.org/10.1111/j.1365-2265.2012.04434.x

Tremellen K. Oxidative stress and male infertility—a clinical perspective. Hum Reprod Update 2008;14:243–58.

Walczak-Jedrzejowska, R., Wolski, J. K., & Slowikowska-Hilczer, J. (2013). The role of oxidative stress and antioxidants in male fertility. *Central European journal of urology*, *66*(1), 60–67. https://doi.org/10.5173/ceju.2013.01.art19

Zini A, Dohle G. Are varicoceles associated with increased deoxyribonucleic acid fragmentation? Fertil Steril 2011;96:1283–7.