9th Yazd International Congress and Student Award on Reproductive Medicine with 4th Congress of Reproductive Genetics

Key Lectures

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Etiologies of male oxidative stress

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Reactive oxygen species (ROS) are formed during normal cellular metabolism. In the male reproductive system, they are involved in many physiological processes, including capacitation, hyper-activation, acrosome reaction and sperm-oocyte fusion. The generation of the ROS occurs via three methods in spermatozoa: (1) in the cell membranes, using nicotinamide adenine dinucleotide phosphateoxidases, and (2) in the mitochondria, using nicotinamide adenine dinucleotide phosphateoxido-reductase and finally, (3) immature spermatozoa with residual cytoplasm contains high levels of glucose-6-phosphate dehydrogenase, a cytosolic enzyme that utilizes the hexose monophosphate shunt to produce abnormally high levels of NADPH. Excessive NADPH results in a greater production of superoxide anions by NADPH oxidases. Activated leukocytes (peroxidase positive) also produce large quantities of ROS and it has been shown that leukocytes are the predominant source of ROS in raw human semen. When ROS increases beyond the physiological levels, overwhelming opposing antioxidant forces, oxidative stress (OS) results. When this occurs, ROS can lead to lipid peroxidation, DNA damage, and OS-induced apoptosis and autophagy, which can be harmful to the highly susceptible sperm cells.

In addition to several diseases like diabetes and varicocele, there are many extrinsic factors affecting human spermatozoa by elevating ROS level. Smoking is one of the most clinically relevant and preventable causes of OS. Alcohol consumption is considered as another etiological factor for ROS production. bMalnutrition and poor diet can also induce ROS production. On the other hand, obesity and over-nutrition also play a significant role in inducing OS in the reproductive system. Stress is linked to increasing in ROS in the seminal plasma and impaired sperm quality. There are many proposed mechanistic effects of medicines on ROS and its related OS. Bactericidal antibiotics can overproduce ROS and lead to mitochondrial dysfunction in mammalian cells including spermatozoa. Environmental pollutants such as nitric oxide, sulfur dioxide, carbon tetrachloride, ozone, wood dust, particulate matter, volatile organic compounds, bisphenol A, xenoestrogens, and phthalates can potentially induce OS and decrease sperm quality. Additionally, studies have shown that pesticides such as lindane, methoxychlorate and dioxin-TCDD have been related to testicular OS in animals and humans. Finally, the radiation and electromagnetic fields such as cellular phones also can cause an elevation in ROS production and decrease sperm fertility potential.