Letter to Editor

Metals and male reproduction: The possible mechanisms

Sir,

Men are inevitably exposed to metals due to their ubiquity in nature, wide use in industry and long-term persistence in the environment. The information on the most important occupational and environmental sources of metal exposure has mainly been collected from the toxicological profiles issued by the Agency for Toxic Substances and Disease Registry (ATSDR).[1] Metals may affect the male reproductive system directly, when they target specific reproductive organs, or indirectly, when they act on the neuroendocrine system. These effects can be long lasting and irreversible if Sertoli cells are disrupted during the foetal development. The number of Sertoli cells determines the number of sperm produced in adulthood, because each Sertoli cell can support only a finite number of germ cells that develop into sperm. According to Sharpe *et al*.,[2] Sertoli cells proliferate during the foetal, neonatal and pre-pubertal period, and each of these periods is particularly sensitive to the adverse effects of metals. The disruption of spermatogenesis in men at any stage of cell differentiation can decrease the total sperm count, increase the abnormal sperm count, impair the stability of sperm chromatin or damage sperm DNA.[3] Accumulating in the epididymis, prostate, vesicular seminalis or seminal fluid, metals may impair progressive sperm motility.[3] In addition, metals can cause hormonal imbalance by affecting the neuroendocrine system, disrupting the secretion of androgens from Leydig cells or Inhibin B from Sertoli cells.[4,5] There is growing evidence that oxidative stress is implicated in the pathogenesis of male infertility.[3,4] It is known that human spermatozoa are particularly vulnerable to oxidative stress. An excessive generation of reactive oxygen species (ROS) in the spermatozoa results in the peroxidation of polyunsaturated fatty acids within their plasma membrane.[3] Several metals, including iron, copper, nickel, lead, calcium and cadmium, may increase ROS production, decrease glutathione and other antioxidant levels, enhance the lipid peroxidation of the cell membrane, cause apoptosis, and contribute to the oxidative damage of DNA. Damage to the sperm membrane reduces sperm's motility and ability to fuse with the oocyte, whereas damage to the sperm DNA compromises paternal genomic contribution to the embryo and increases the risk of infertility, miscarriage, or serious disease in the offspring.[6,7-12] Some malformations of the male reproductive system, such as cryptorchidism, hypospadias, and prostate and testicular cancers may originate from exposure to endocrine disruptors.[3-5] Cadmium, calcium, mercury, lead, and arsenic are suspected to affect the endocrine system. Evidence is usually limited to animal data or to *in vitro* studies.[4] The clinical and epidemiological findings are scarce and controversial, and often difficult to interpret because of multiple exposures to different agents and latency of effects.

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Access this article online	
Quick Response Code:	Website: www.advbiores.net
	DOI: 10.4103/2277-9175.133253