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Potential testicular toxicity of sodium nitrate in adult rats.

Aly HA, Mansour AM, Abo-Salem OM, Abd-Ellah HF, Abdel-Naim AB.

Department of Pharmacology and Toxicology, Faculty of Pharmacy, Al-Azhar University, Nasr City, Cairo, Egypt.

hamdyaali@yahoo.com

Nitrate is a common contaminant in groundwater aquifers. Current study aimed at evaluating the potential testicular toxicity of sodium nitrate in rats. Sodium nitrate was given orally to rats at doses of 50, 100 or 200 mg/kg/day for 60 consecutive days. Sperm count and motility, daily sperm production and testis weight were significantly decreased specially at high doses. Testicular activity of lactate dehydrogenase-X, glucose-6-phosphate dehydrogenase, and acid phosphatase were inhibited in a dose-related manner. Lipid peroxides and hydrogen peroxide production were significantly increased in all treated animals. This was accompanied by inhibition of testicular activities of superoxide dismutase and glutathione peroxidase. Fifty mg/kg of sodium nitrate did not significantly alter catalase or glutathione reductase activity. Glutathione was significantly decreased by sodium nitrate in a dose-related manner. The decrease in sperm count and motility and daily sperm production was confirmed by histopathological studies which indicated chromatolysis, pyknosis and necrosis in spermatocytes. In conclusion, subchronic exposure of rats to sodium nitrate results in testicular toxicity as evidenced by decreased sperm count and motility, daily sperm production and testis weight, inhibited activity of enzyme markers of spermatogenesis and induction of histopathological changes. These effects are attributed, at least partly, to testicular oxidative stress.

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