Uranium exposure increases spermatocytes metaphase-apoptosis in rats: inhibitory effect of thymoquinone and N-acetylcysteine

H Waly, SM Ragab, Khaled M A Hassanein, NS Khalil Abou, EA Ahmed

Abstract:

Uranyl acetate (UA), a commercial stock from depleted uranium (DU), has a combined effect of chemical toxicity and mild radioactivity. Here, we investigated the potential antioxidant, antiapoptotic and cytoprotective effects of thymoquinone (TQ) and N-acetylcysteine (NAC) against UA-induced testicular damage in rats. UA reduced testicular superoxide dismutase (SOD) activity and nitric oxide (NO) and glutathione (GSH) levels relative to the control group. Interestingly, the testicular SOD activity and NO and GSH levels of UA/TQ-and UA/NAC-treated groups were also significantly lower relative to the control. A marked increase in spermatocytes metaphase apoptosis was found (stage XIII) in UA-treated rats, which is probably due to difficulties in segregation of homologous-chromosomes. This may clarify why UA exposure decreased round spermatids numbers and fertility in previous studies. To check the reason of partial metaphase arrest, the presence of DNA-damage-related γ-H2AX foci in late spermatocytes of all groups was checked, but only insignificant increase was found in UA-treated group. TQ or NAC supplementation reduced the apoptosis and improved the testicular histological alterations. Thus, TQ and NAC attenuate UA adverse effects on the testicular microenvironement through anti-apoptotic and cytoprotective but not antioxidant effects.

Keywords:

Uranyl acetate, thymoquinone, apoptosis, antioxidant

Published In:

General physiology and biophysics, NULL, NULL