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POSSIBLE RENAL FAILURE ASSOCIATED WITH URANIUM TOXICOLOGICAL AND INCORPORATION EFFECTS IN ANIMALS

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Fifteen male Wistar rats, 15 days old, were housed in 7 metabolic cages, forming 6 groups with 2 animals each and one group, the "control group", with 3 animals. They were fed with rat chow doped with uranyl nitrate, except the control group, at concentrations ranging from 0.5 to 100 ppm. The uranium content in the ashes of several organs was measured by the fission track counting technique, following neutron irradiation of the biological samples near the core of a research reactor.

The urine of the animals from the group fed with higher doses of uranyl nitrate (from 20 to 100 ppm) was darker and showed blood, suggesting possible kidneys malfunction condition. The laboratorial examination revealed, however, that pH, protein concentrations, cetonic bodies, nitrite, bilirubin and urobilinogen, in all animals, ranged within the normal parameters for rats.

In fact, acute and chronic renal failure could be provoked by many factors, as e.g. the ingestion of nephrotoxins (heavy metals, pesticides and drugs). The specific toxic burden from nephrotoxins takes place at renal tubule level, which could conduct to celular death. The kidneys almost stop functioning in acute processes where tubules obstruction is verified, but such a condition could be reversed after celular recovery. On the other hand, in chronic renal failure the final result is the irreversible decrease of the function nephrons number [1]. Such occurrence could be verified by many renal function tests where, among them, we mention the analysis of urine sediment performed at this Laboratory: it was observed nearly 50 red blood cells per μ l.

Toxicologic studies carried out in mamalians, show that most of the uranium absorbed by the GI tract, or by injection, is excreted through the urinary tract. However, amounts of non-excreted uranium are mostly acumulated in the kidneys, where inhibition of enzymatic reactions could take place causing, thus, lesions leading to the failure of these organs. We note that the enzymatic reactions control respiration of the renal cells. One of the consequences of such lesions is the alteration of the glomerular filtration[2]. It is not clear if this alteration works toward an increase or decrease of the uranium concentration in the blood. Quite intriguing, on the other hand, is the observation that the measured uranium transfer coefficients f for several organs, as a function of the uranium amount. A (ppm) in the animals food, are increasing for $A > 20$ ppm - this is the U - concentration range where blood was detected in the urine. It is very likely that this increasing character of f is due to an augmented concentration of U in the blood which, by its turn, could be a consequence of kidneys malfunction. A quantitative evaluation of this issue is presented elsewhere [3].

References

- [1] A.C.Guyton, J.E. Hall Textbook of Medical Physiology, 9th edition, 1996, W.B. Saunders Co. Philadelphia, PA 19106.
- [2] D. P. Haley. Morphologic changes in uranyl nitrate-induced acute renal failure in saline - and water-drinking. United States-Canadian Division of the International Academy of Patology 1982.
- [3] J.D.T. Arruda-Neto et al., contribution to this event.