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# CORRELATION BETWEEN ABNORMAL URANIUM INCORPORATION AND RENAL FAILURE IN ANIMALS

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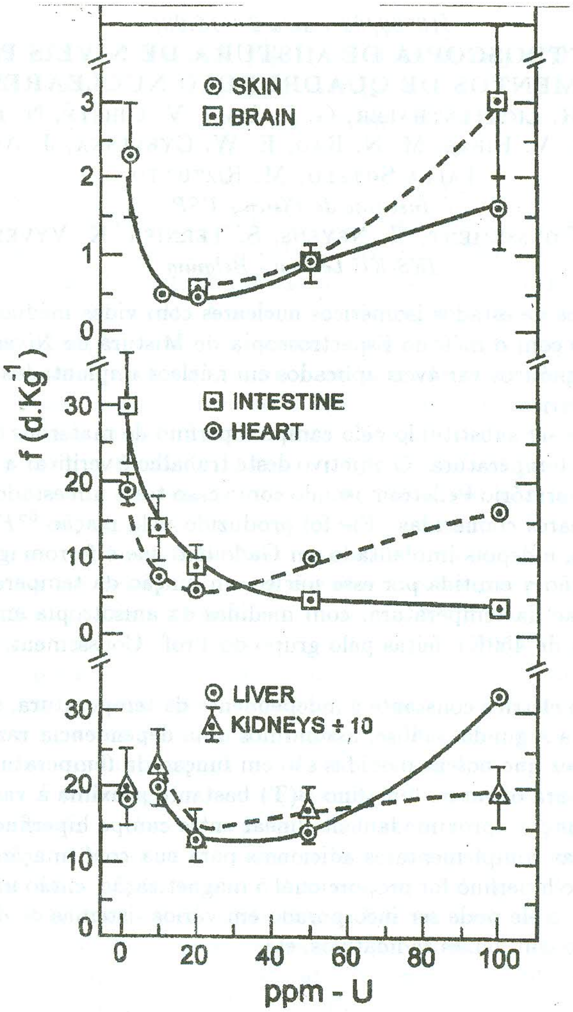
We report on an investigation of a long-lasting uranium ingestion from food, starting after weaning, and lasting until the animal maturity. The mode and interval of ingestion, in this case, simulate a common real-life scenario, which is valid for both animals and humans. Thus, the transfer coefficients of U to the organs of Wistar rats were determined as a function of the U concentration in the food. Groups of animals were fed with rat chow doped with uranyl nitrate at concentrations ranging from 0.5 to 100 *ppm*. The U content in the ashes of liver, kidneys, heart, brain, intestine, skin and testicles, was measured by the fission track counting technique, following neutron irradiation of the biological samples near the core of a research reactor; the results are shown in the figure.

We note that the transfer coefficient $f$ for a given organ is defined by

$$f = \frac{C}{A}(d \cdot kg^{-1}), \tag{1}$$

where $C\ (mg - U\ /\ kg)$ is the U content per $kg$ of organ mass, and $A\ (mg - U\ /\ day)$ is the amount of U daily consumed by the animal during its last, and prolonged, feeding period.
It has been argued recently if $C = C(A)$ is indeed a linear function, say $C = a \cdot A$, where $a$ is a constant; in this case, $f = a\ (constant)$. It has been demonstrated for vegetables that, in fact, $C = a \cdot A^b$, $0 < b < 1$ [1], leading to the hyperbolic function $f(A) = a \cdot A^{b-1}$. A visual inspection of the figure reveals that for $A < 1\ mg - U\ /\ day$ ($\sim 20\ ppm$ in the food) the transfer coefficient function $f = f(A)$ is hyperbolic, while only for intestine a hyperbola is verified up to $A = 5.2\ mg - U\ /\ day$ ($100\ ppm$ in the food ). From least square fittings we got $b \approx 0.5$ for all organs; which is quite similar to vegetables.
Incidentally, in the animals from the groups fed with $A > 20\ ppm$ we detected $\sim 50$ red blood cells per $\mu\ell$ of urine, indicating thus the occurrence of lesions, which could lead to alteration of the glomerular filtration [2]. If, in this scenario, the U concentration in the blood increases, it may well be possible that $C\ (mg - U\ /\ kg)$ for each organ increases as well, except for the intestines where U is transferred mostly from the earlier uptake stage (at the GI tract).
Therefore, we are led to the conclusion that the deviation of $f = f(A)$ from the normal hyperbolic trend, for $A \geq 20\ ppm$, is a signature for renal malfunction. Note that for the intestines $f = f(A)$ mantains its hyperbolic shape in the whole range of $A$.



By extrapolating the functions $f = a \cdot A^{b-1} \cong a \cdot A^{-0.5}$, obtained by fitting in range $A$: $0-20$ *ppm*, up to $A = 100$ *ppm* we obtain an estimate for $f$ in the absence of renal malfunction: $f_h$ ("hyperbolic transfer coefficient"). Naming the actual, experimental, transfer coefficients by $f_e$, we define the following ratio

$$r = \frac{f_e - f_h}{f_h}, \tag{2}$$

which expresses the relative difference between $f_e$ and $f_h$ and, also, it could provide a quantitative evaluation for the renal failure problems affecting the incorporation of U. In our case, the average $< r >$ for skin, heart and brain is $(2.0 \pm 1.6)$ for $A = 50$ *ppm - U*, and is equal to $(7.6 \pm 1.6)$ for 100 *ppm-U*

## References

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