metabolic product of α-chlorohydrin which directly affects GA3Pdh, TPI and aldolase. Addition of DL-1-chloro-1-deoxyglycerol-3-phosphate (0.1 mM) severely inhibited the activity of GA3Pdh (53%) in the reaction cuvette containing spermatozoal sonicate. One hundred times that concentration of inhibitor was required for significant inhibition of TPI and aldolase. We suggest that α-chlorohydrin is phosphorylated on entry into the spermatozoa. The phosphorylated compound may then act as a competitive inhibitor of GA3Pdh because of its structural similarity to the normal substrate of the enzyme, glyceraldehyde-3-phosphate. Further investigations of this hypothesis are presently under way.

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Localisation of plutonium in mouse testes

THE plutonium isotope ^{239}Pu is a known carcinogen but its ability to produce genetic damage has not been so well investigated. The radionuclide is found in gonads after intravenous injection into humans[1] and after administration to animals by various routes[2]. Retention of ^{239}Pu in the gonads is prolonged and the genetic effects there have been estimated on the basis of average dose in the gonads[3]. Inhomogeneity of distribution of plutonium within gonads has, however, been noted[4] giving rise to the possibility that some cells may receive a greater radiation dose than others. No investigations of the effects of this inhomogeneity upon dose-rate to the cells involved in gametogenesis have been reported. Here we report briefly that a non-uniform distribution of ^{239}Pu in the testis results in increased radiation of the spermatogonial stem-cells.

Groups of 12-week-old male CBA mice were killed 4 or 12 weeks after intravenous injection of ^{239}Pu citrate. The plutonium in each right testis was measured radiochemically (Table 1). Autoradiographs prepared from 6-μm sections of frozen left testes showed that some 90% of the plutonium seemed to be deposited within the intertubular spaces and in the peritubular tissue immediately surrounding them.

A total of 12,000 α tracks were counted in more than 700 randomly selected fields from 58 sections.

Table 1 Radiochemical analysis of the right testis of CBA mice following intravenous injection of ^{239}Pu in 1% trisodium citrate solution

Weeks after injection	4	12
Number of testes	5	6
Testis mass (mg, mean ± s.e.)	73±1	67±4
Plutonium (pCi, mean ± s.e.)	20±3	23±2
Plutonium (pCi g^{-1}, mean ± s.e.)	280±45	350±38
Average dose rate (rad d^{-1}, mean ± s.e.)	0.072±0.012	0.092±0.010

A mean activity of 88 nCi ^{239}Pu was injected into each mouse; 3.2 μCi per kg body mass.

The α tracks could be categorised into: those which lay entirely above the intertubular spaces (47%); those which lay above the peritubular membrane or above the adjoining 10-μm layer of cells containing the spermatogonial stem cells (42%); and those which lay entirely within the peritubular membranes but excluding those in group 2 (11%).

On the assumption that the testis consists of a close-packed series of cylinders of uniform diameter (experimentally determined mean value =206 μm) it was calculated that the 10-μm shell of tissue surrounding each tubule and containing the spermatogonial stem-cells, constituted 17% of the whole testis mass. It was then assumed that the spermatogonial stem-cells were randomly distributed in these 10-μm peritubular shells and a dose inhomogeneity factor N (ref. 5) was calculated:

$$N = \frac{\text{average dose to spermatogonial stem-cells}}{\text{average dose to whole testis}}$$

$$= \frac{\text{fraction of all } \alpha \text{ tracks in group 2}}{\text{fraction of testis mass in 10-}\mu\text{m peritubular shells}}$$

$$= \frac{0.42}{0.17} = 2.5$$

Therefore, from the data given in Table 1, dose rates to spermatogonial stem-cells in our experiment were 0.18 and 0.23 rad d^{-1} at 4 and 12 weeks respectively. A value $N=2$ was also calculated directly from the model system assuming random distribution of ^{239}Pu in the intertubular spaces and taking into account the transfer of energy along the path of each α particle (M. C. Thorne, personal communication).

Our results show that for mice the radiation dose rate to the spermatogonial stem-cells is greater by a factor of 2–2.5 than the average for the testis as a whole calculated directly from the total amount of ^{239}Pu deposited in the gland. This implies that the genetic effects may be proportionately greater. The validity of the factor N as calculated and the biological effectiveness of α particles relative to other radiations are matters now undergoing cytological investigation (A. G. Searle, personal communication). It would also be important to establish comparable values for ovaries. Much more work is required to decide on appropriate values for men.

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