degradation of DNA, measured viscosometrically, in samples extracted from *E. coli B/r* immediately after irradiation was not influenced by the presence of iodoacetamide.

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Influence of X-rays on the Incorporation of Deoxyribonucleic Acid Precursors into some Mammalian Cells *in vitro*

Dose-response curves for the influence of ionizing radiations on *in vitro* incorporation of DNA precursors into human bone marrow[1] and experimental tumours[2,3] irradiated in the period of synthesis of DNA are well known. Recently published investigations[4] showed a striking similarity between these curves for human bone marrow and Ehrlich ascites carcinoma while both systems were oxygenated. Our experiments show the relationship between the dose of X-rays and the depression of thymidine incorporation in anoxic suspensions of rat bone marrow and Ehrlich ascites tumour.

The suspensions[5] contained an average amount of $2{\cdot}4 \times 10^7$ nucleated bone marrow cells together with $0{\cdot}7 \times 10^7$ erythrocytes or $1{\cdot}1 \times 10^7$ tumour cells in 1 ml. of medium, consisting of 50 per cent of supplemented Hanks's solution and 50 per cent of rat serum. The suspension of cells was irradiated in room temperature with X-rays, 160 kVp, dose-rate of 140 r./min. Thymidine-2-^{14}C, 0·75 μc./ml. culture, specific activity 16·0 or 18·3 mc./mM, was added immediately afterwards. 2-ml. aliquots of cell suspension were incubated at 37·5° C in a number of screw-cap bottles for 2 h without shaking. After incubation the cells were washed twice in ice-cold solution of 0·85 per cent sodium chloride and once in a 2 per cent acetic acid. Each time the cell suspension was centrifuged for 10 min at 0° C (2,000*g*). Finally, the sediment was suspended in 2 ml. of 2 per cent acetic acid and three aliquots of 0·3 ml. were placed in aluminium planchettes. A windowless gas-flow counter was used for measuring the activity. The mean values of the experimental groups were expressed as a percentage of the control group. For each dose 4–6 separate experiments were carried out. Standard deviation of individual experiments are indicated on the graph (Fig. 1). The tendency of the resulting curves to converge allowed us to draw a common curve for both tissues.

In the foregoing experiments both systems are anoxic: the suspension of Ehrlich ascites tumour cells does not contain red blood cells; the ratio of erythrocytes to nucleated bone marrow cells is about 1 : 3, while Berry *et al.*[4] added 50–100 red blood cells for each tumour cell to the suspensions so as to oxidize

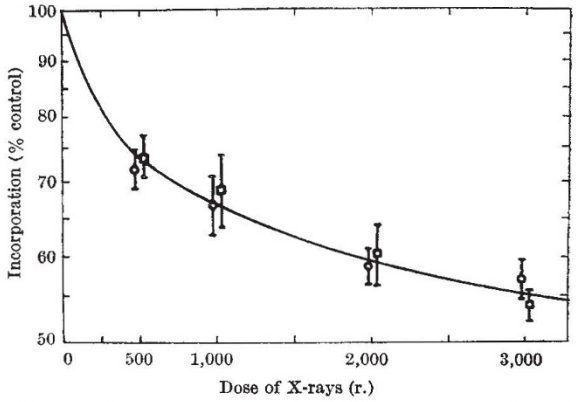

Fig. 1. Dose–response curve for X-rays on incorporation of thymidine-2-^{14}C into rat bone marrow and Ehrlich ascites tumour cells *in vitro* (S period). □, Bone marrow; ○, Ehrlich ascites tumour

the cells of Ehrlich carcinoma. The same X-ray dose-response curves for the inhibition of incorporation of thymidine were obtained even though the compared suspensions contained cells that were taken from two species of animals and were of a different tissue character, one being a rather homogeneous population of tumour cells of epithelial origin, and the other normal, inhomogeneous, hæmopoietic tissue. Finally, they also differ in the rate of incorporation of thymidine that was found in other experiments[6] in which irradiation was not performed.

This lack of specificity of X-ray action on incorporation of thymidine into different tissues depends probably on the environment of the cell. The kind of serum used seems to be an important factor. Berry, Hell, Lajtha and Ebert obtained similar results testing oxygenated suspensions of human bone marrow and Ehrlich ascites tumour cells; in both cases they used human serum. Lajtha[7], on the contrary, noticed changes in the effect of X-rays on synthesis of DNA induced by various sera. This may account for the differences between dose-response curves for Ehrlich carcinoma obtained by Berry *et al.*[2] and by us.

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BIOLOGY

Tumour Induction by an Indolyl-3-Acetic Acid–Kinetin Interaction in a *Nicotiana* Hybrid

It has been demonstrated that auxins influence the ability of bacteria to induce plant tumours[1], and that kinetin in the presence of auxin can substitute for extracts of tumours in the production of tumours on isolated tobacco pith tissue[2].