experiment 2), there was no indication of prevention of C_3H marrow transplantation, as evidenced by the survival data, compared with the group given C_3H bone marrow alone. The limited results of experiment 4 suggest that the administration of normal homologous mouse blood (in this instance from C_3H strain) can, indeed, alter the course of transplantation of isologous marrow in lethally irradiated hosts. Note that in this group the mean survival time (19 days) was significantly increased, as compared with the mice injected with homologous bone marrow plus blood.

These experimental results provide further support for the presence of immunologically reactive cells in peripheral blood. Presumably, when injected under the experimental conditions described, such cells or their progeny come in direct contact with the donor bone marrow cells (of different geno-type from the injected blood cells), causing their rejection by a homograft type of reaction. As a result, the host animals succumb, as do control irradiated mice, from the sequelæ of marrow failure. In any event, regardless of the specific mechanisms involved, empirical evidence is given here for the deleterious effect of injected blood with respect to the protection of lethally X-irradiated mice by means of bone marrow transplantation. Although the present experiments deal with specific, inbred mouse strains, their possible implications for marrow therapy in irradiated humans[6] are evident.

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Effect of Whole-body X-Irradiation on the Insulin Resistance of Fasted Rats

An increase of the insulin resistance was observed in fasted adult albino rats if they previously were exposed to 600 r. of X-rays.

The number of animals which survived a lethal dose of insulin for 24 hr. was used as a measure of the insulin resistance. Rats of our laboratory strain were used. In the first group, 20 adult males were deprived of liver glycogen stores by withdrawing food for 46 hr. before the insulin was injected. 1 hr. before the injection animals were 'sham irradiated' and weighed. Body-weights were 250–350 gm. Insulin-zinc ('Prolek' Beograd, 40 U./ml.) was injected subcutaneously in the hind limb. The dose was 6 U./100 gm. body-weight. After the injection animals were kept fasting. All animals died in the interval between 2 hr. 55 min. and 6 hr. 15 min. after insulin injection.

The second group of 20 males was treated in the same way, except that they were irradiated 1 hr. before injecting insulin. They received 600 r. whole-body irradiation (measured in air), with 180 kV. X-rays (10 m.amp.). The dose was given 40 cm. from the target at 55 r./min. Filters used were 0·5 mm. copper and 1 mm. aluminium. In this group 14 animals died, the first one 3 hr. 10 min. and the last one 6 hr. after insulin injection. Six of the animals were alive 24 hr. after injection.

The same experiment was carried out with female rats which weighed 200–300 gm. before insulin injection. Of the control 'sham irradiated' group, 11 animals died. The first one died 4 hr. 45 min. and the last two died between 13 hr. and 22 hr. after the insulin injection. Of the females exposed 5 animals died, the first one 3 hr. 10 min. and the last two between 13 hr. and 22 hr. after the injection.

The results suggest that irradiated fasted rats were to a certain degree more resistant to insulin than animals which were fasted only, and that insulin resistance was more pronounced in the female than in the male rats. The influence of irradiation could be mediated through a 'contra-insular' effect of endogenous glucocorticoids, since hyperactivity of the adrenal cortex, especially in the first 24 hr. after exposure, has been repeatedly reported, and starvation does not alter the ability of the pituitary-adrenal system to respond to the stimulus provided by whole-body irradiation[1]. This view is supported by the observed decrease of hexokinase activity in rat's tissues following 600 r. of whole-body X-irradiation[2]. The observed sex difference is more difficult to explain. It is possible that this is connected with the known protein anabolic effect of androgens, and so in males these would be more antagonistic to glucocorticoids.

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Hormone Effects on Early Distribution and Disappearance from the Blood of Promethium-147 in Rats

Growth hormone, as well as cortisone, has been shown to alter metabolic balances and tissue distribution of potassium and sodium[1]. With injected calcium-45, growth hormone restored the diminished uptake by bone observed in hypophysectomized rats, but in normal rats it had no effect on uptake of calcium-45[2]. The present work with normal rats demonstrates effects of growth hormone and cortisone on early uptake by tissue and on disappearance from the blood with time of the lanthanide fission metal promethium-147.

Thirty-one young adult female Wister rats (200 ± 25 gm. weight) were bilaterally adrenalectomized and maintained on 2 per cent saline, and/or treated with cortisone (dosage: 45 mgm./kgm. body weight) or growth hormone (dosage: 8 mgm./kgm. body weight), and intravenously injected with 10 μc. promethium-147 (as chloride) in 0·25 ml. of dilute citrate solution, *p*H 2, according to the