ing axolemmal membrane of the presynaptic fiber in a manner such that a single membrane of double thickness is sometimes produced. 3. Pre- and postsynaptic axolemmal membranes are structurally similar and are about 300 Å in thickness. 4. The axoplasm of the postsynaptic fiber and of the synaptic processes contains a distinctly increased number of axoplasmic filaments per unit volume over that of presynaptic axoplasm.

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Hepatic Glycogen in Acute Radiation Death.* (20072)

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In radiobiological research involving acute x-radiations, interest is directed toward discovering sensitive indicators of irradiation damage. Liver glycogen may prove to be such an indicator. It might be hoped that glycogen depletion has a quantitative relationship to irradiation dosage.

Materials and methods. Hamsters (*Cricetus auratus*), mice (Swiss albino), and salamanders (*Triturus pyrrhogaster*) were exposed to x-radiation until they were moribund. The animals were then sacrificed by decapitation, and their livers were immediately removed and fixed in cold Rossman's solution. These tissues were studied for glycogen by the histochemical technic of Hotchkiss and McManus (1,2) which involves an oxidative treatment of the sections with periodic acid and subsequent treatment with Schiff's leuco-fuchsin followed by sodium bisulfite. The specificity of the reaction is controlled by treating alternate sections with saliva or diastase before staining. As an additional check for the presence of glycogen in the hamster livers, the microchemical method of Boettiger(3) was used.

The irradiation conditions were as follows: The hamsters were killed under the 2 million volt x-ray machine made available through the courtesy of Dr. Vincent Collins of the Delafield Memorial Hospital. The dose rate was 534 r/minute at a distance of 61 cm, and the animals were moribund at 110,000 r in 3 hours and 26 minutes. The mice and salamanders were exposed between parallel tube sources at the Radiological Research Laboratory, stabilized at 110 KV and 30 MA, without filtration, with a distance of 30 cm from the source to the center of the body of the animals. The dose rate was 2200 r/minute. In all cases the animals were enclosed in plastic boxes. The salamander bodies were kept moist with cotton dampened with tap water.

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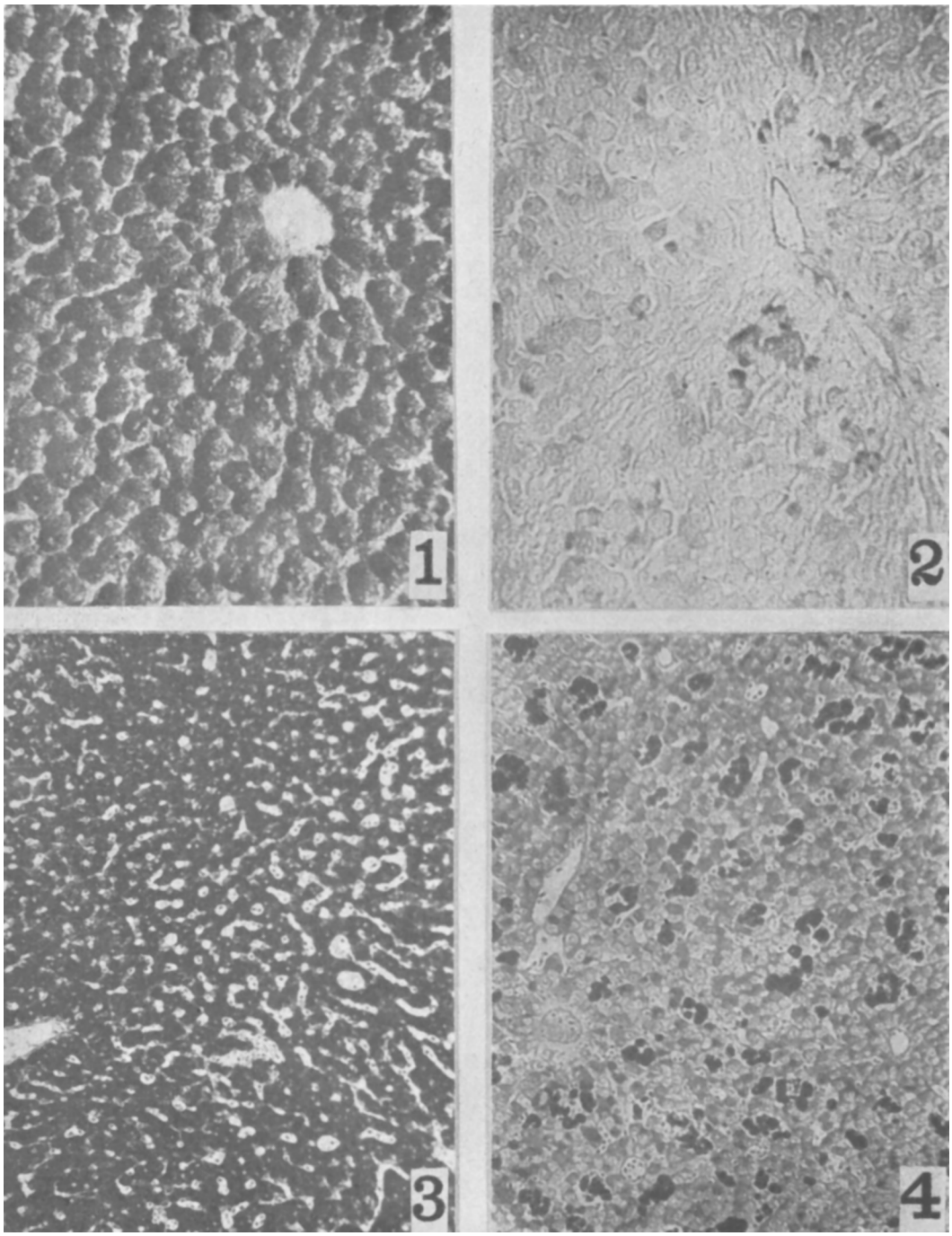

FIG. 1. Normal hamster liver, P.A.S. preparation.

FIG. 2. Liver of hamster irradiated until moribund. There is almost no Schiff positive (glycogen) material.

FIG. 3. Liver of normal salamander, P.A.S. preparation.

FIG. 4. Liver of irradiated salamander. Many hepatic cells free of glycogen. P.A.S. preparation.

Results. In the livers of those mammals irradiated as described above, there was little or no glycogen as shown by the Hotchkiss-McManus technic (Fig. 1 and 2). The microchemical determinations specific for liver glycogen confirmed this finding. The normal hamster liver contained 3% glycogen (by weight) while the liver of the irradiated animals, after only 3½ hours and 110,000 r, contained only 0.3% glycogen.

In the cold-blooded salamanders exposed to the acute lethal dose of x-radiation, there was a marked decrease in liver glycogen as illustrated by Fig. 3 and 4, but the depletion was not as complete as it was for the mammalian livers.

Discussion. The apparently contradictory report of Ross and Ely(4) that there was an increase in hepatic glycogen when fasted rats were x-irradiated up to 500 r is not applicable here since our animals were not fasted, and we used extremely high dose rate and levels of exposure. Louran and Lartigue(5) suggested that x-radiation interfered with the synthesis of glycogen in guinea pigs exposed to 500 r total body x-radiation. Their findings, however, probably have little relevance to ours, since their observations were made over a period of days and all of our animals died within a maximum of 3½ hours after the start of x-ray exposure.

The cold-blooded salamanders lived 80 minutes under a dose rate of 2200 r/minute which meant a total accumulation of 176,000 r. It is difficult to determine the death instant of these sluggish animals, and some might have "survived" further exposure under the x-ray beam. The livers of these animals did show some glycogen depletion but not as complete as that seen in the mammalian livers. This difference may be accounted for by their slower metabolic rate.

Since there was a drastic depletion of liver glycogen in all animals studied, there must be a common radiogenic cause. One cannot rule out the possibility of an adrenal-stress syndrome. There may also be an acute syndrome involving other endocrine systems such as the pituitary-pancreas-adrenal complex. A recent paper by the authors(6) points up histological manifestations of acute exposure in the lymphocytic organs, and a further paper(7) illustrates changes in the endocrine organs. It would be ill-advised to emphasize depletion of hepatic glycogen as an independent reaction. Nevertheless, the effect is so drastic and dramatic that it compares well with the known radio-sensitivity of lymphocytes.

Conclusions. In both warm-blooded animals and in cold-blooded amphibia, the liver responds to acute x-radiation by a drastic depletion in liver glycogen, as determined both by histochemical and microchemical tests.

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