

could expect just measurable changes within this range if the phenomenon fits a semi-log plot and if sufficient numbers of animals were used.

Fig. 2 presents data obtained from Table I with specific regard to AET. The upper 2 curves present time-mean delta pH plot of the 2 controls (i.e. with and without AET), while the lower present 300 r, with and without AET (Table II).

Discussion. The finding that apparent threshold levels of gamma irradiation in the rat are in the vicinity of 20 to 30 r for whole blood cholinesterase is in complete agreement with Sabin(1) who found similar activity with x-irradiation down to 25 r in the mouse red cell cholinesterase. Thus the two species are remarkably similar despite differences in type of irradiation and different enzyme entities.

The fact that AET fails to protect whole blood cholinesterase from gamma irradiation is evident from Fig. 2 as the 2 levels are virtually identical, i.e. 1.2% apart, on the tenth day, the time of expected peak depression. AET at the high levels tested did not appreciably affect cholinesterase in the surviving animals, though mild significant depression

attributable to AET alone was noted on the third and fourteenth days (Table II).

Summary. 1. Whole body gamma irradiation in the rat produced significant whole blood cholinesterase depression on the tenth day at a dosage level of 75 r. 2. The levels tested when plotted and extrapolated indicated threshold changes in cholinesterase activity would be in the vicinity of 20 to 30 r. 3. AET alone, while producing some mild cholinesterase depression, failed to protect whole blood cholinesterase activity from the effects of gamma irradiation at the levels of agent and irradiation tested.

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Effects of Source of Dietary Carbohydrate on Survival Time of Sublethally X-irradiated Mice.* (26417)

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During an investigation of the effects of diet on response of mice exposed to multiple sublethal doses of total body x-irradiation, it was observed that length of survival of x-irradiated animals was significantly affected by the source of dietary carbohydrate employed. Our findings are reported here.

Procedure. The basal ration employed consisted of carbohydrate, 59%; casein[†], 24%; cottonseed oil, 10%; salt mixture[‡],

5%; cellulose[§], 2%; and the following vitamins per kg of diet: thiamine hydrochloride, 10 mg; riboflavin, 10 mg; pyridoxine hydrochloride, 10 mg; calcium pantothenate, 60 mg; nicotinic acid, 100 mg; ascorbic acid, 200 mg; biotin, 4 mg; folic acid, 10 mg; para-aminobenzoic acid, 400 mg; inositol, 800

[†] Vitamin-free Test Casein, General Biochemicals, Inc., Chagrin Falls, Ohio.

[‡] Wesson Modification of Osborne-Mendel Salt Mixture, General Biochemicals, Inc., Chagrin Falls, Ohio.

[§] Solka Floc, Brown and Co., Boston, Mass.

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TABLE 1. Effects of Source of Dietary Carbohydrate on Survival Time of Mice Exposed to Multiple Sublethal Doses of Total Body X-irradiation.*

Dietary carbohydrate	No. of animals	Avg body wt at time of 1st x-irrad. (g)	Day after 1st x-irrad. when 50% of animals in group were dead	No. surviving†	Avg length of survival after 1st x-irrad. (days)‡§
Glucose	29	31.8	66	8	73.3 ± 6.6
Sucrose	29	32.9	55	1	54.3 ± 3.8
Dextrin	29	30.0	44	1	45.6 ± 3.3
Cornstarch	26	30.1	34	2	41.8 ± 4.8

* Each group initially consisted of 30 mice. Animals that died before 4th x-irradiation were not included in tabulation of data. Dextrin employed (Dextrin White from corn) was obtained from Mallinkrodt Chemical Works, St. Louis, Mo.; cornstarch from Mefford Chemical Co., Los Angeles, Calif.

† Experiment was terminated 118 days after 1st x-irradiation.

‡ Data were calculated on basis of an avg survival time of 118 days for animals alive at termination of experiment.

§ Including stand. error of mean.

mg; vit B₁₂, 150 µg; 2-methyl-1,4-naphthoquinone, 5 mg; choline chloride, 2 g; vit A, 5000 U.S.P. units; vit D₂, 500 U.S.P. units; and alphatocopherol acetate, 100 mg. The vitamins were added in place of an equal amount of dietary carbohydrate. Four carbohydrates were employed, diet A, glucose; diet B, sucrose; diet C, dextrin; and diet D, cornstarch.

One hundred and sixty male mice of the Webster strain were selected at 11 to 14 g in body weight and were divided into 4 comparable groups consisting of 40 animals per group. These were placed in metal cages with raised screen bottoms (5 animals per cage) and were fed the various diets indicated above. Food and water were provided *ad libitum*. The animals were fed daily and all food not consumed 24 hours after feeding was discarded. After 6 weeks of feeding 10 of the mice in each dietary group were selected at random to serve as non-irradiated controls. The remaining mice received an exposure of 200 r total body x-irradiation, repeated once weekly until a total dose of 1200 r (6 exposures) had been administered. Animals were continued on their respective diets for 118 days after first x-ray exposure or until death, whichever occurred sooner. All mice that died before the 4th x-ray exposure were not included in tabulation of data. Data were obtained as to the day after the first x-ray exposure on which 50% of the animals in each group were dead and average length of

survival on each of the diets employed.

The following radiation factors were employed: GE Model Maximar 250; 250 kv; 15 ma; 0.5 mm Cu and 1 mm Al filters plus a Cu parabolic filter‡; HVL, 2.15 mm Cu; target distance to top of box, 82 cm; and dose rate, 15.6 r per minute (measured in air, at top of box). The animals to be irradiated were placed in a wooden box divided into 60 equal compartments 1¼" wide, 3" long, and 1½" deep. The partitions and top were made of ⅛" cellulose acetate sheeting; and the top and bottom of each compartment were perforated with holes for purposes of ventilation. The container was rotated slowly on an electrically driven turntable to insure equivalent irradiation.

Results. Significant differences were observed between the various dietary groups as to the day on which 50% mortality occurred and average length of survival following exposure to multiple sublethal doses of total body x-irradiation (Table 1). Survival time was least for the mice fed dextrin and cornstarch (diets C and D), slightly longer for those fed sucrose (diet B) and significantly longer for those fed glucose as the source of dietary carbohydrate. The increased survival on the latter diet occurred despite the

‡ A nonuniform filter which produces a flat isodose surface of x-ray intensity constructed by the method of Greenfield and Hand(7). The center of the filter had a thickness of 1.7 mm Cu; the edge, 0.5 mm Cu.

fact that mice in this group received more total radiation than those in the other groups. This situation obtained because irradiation was continued as the experiment progressed, hence the mice which survived were exposed to a greater total dose of x-irradiation than those which succumbed earlier in the period. No significant differences in survival were observed among non-irradiated mice in the various groups with at least 80% of the animals in all dietary groups surviving the experimental period of 160 (42 + 118) days.

No data are available as to the cause of the diverse results obtained with the different carbohydrates. It is well established that the intestinal epithelium is highly sensitive to x-irradiation (1-6), and it is possible that multiple sublethal doses of total body x-irradiation induced changes in respect to digestion and absorption of carbohydrates that resulted in a simple sugar such as glucose being utilized more efficiently and with less toxic effects than was the case when more complex carbohydrates were fed.

Summary. Experiments were conducted to determine the effects of source of dietary carbohydrate on survival time of mice exposed to multiple sublethal doses of total body x-irradiation. Findings indicate that average survival time of x-irradiated mice was significantly longer on a glucose-containing diet than on rations containing sucrose, dextrin or cornstarch as the source of dietary carbohydrate.

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A Colorimetric Method for Assay of Erythrocytic Glucose-6-phosphate Dehydrogenase.* (26418)

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A disorder known as primaquine-sensitive hemolytic anemia(1,2) in humans is proving to be increasingly useful as a tool for study of genetic homeostasis(3) and biochemical genetics(4,5,6). This disorder was first reported by Carson(7) to be associated with a deficiency in activity of glucose-6-phosphate dehydrogenase in red blood cells. More recently, 2 different enzymic expressions of

the deficiency have been uncovered(8). Investigations of these disorders have been handicapped by the necessity, when the enzyme is to be measured accurately in hemolysates, for a spectrophotometer capable of operation in the ultraviolet range and equipped with a photomultiplier to compensate for the very high optical density of the hemolysate-blank. Motulsky and Campbell(9) have adapted the technic of Dickens and Glock(10) to permit useful visual estimation of the activity of erythrocytic glucose-6-phosphate dehydrogenase in genetic field work. There would seem to be a need for an objective assay requiring only relatively commonplace equipment.

A method of assaying human erythrocytic

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