

TABLE II. A Comparison of SPI in Jersey and Aberdeen Angus Sera. 5 animals in each series.

Breed	Age		Days in milk	Days pregnant	SPI, $\mu\text{g } \%$					Avg
	Yr	Mo			1	2	3	4	5	
AA*	4	4	60	0	2.6	2.7	2.8	4	4.2	3.3
J†	4	7	72	0	3.7	4.5	5.2	5.6	6.7	5.1
AA	3	0	Dry	216	4.2	4.9	4.9	5.5	5.6	5
J	2	7	Dry	206	4	4.1	4.2	4.3	4.9	4.3
AA	4	4	71	0	4.1	4.3	4.5	4.9	5.1	4.6
J	4	2	51	0	5.2	5.3	5.5	6	6.3	5.7
AA	3	10	Dry	213	3.3	4	4.1	4.2	4.3	4
J	3	10	Almost dry	207	4.4	4.6	4.8	5.2	5.6	4.9

* AA = Aberdeen Angus.

† J = Jersey.

1.2 $\mu\text{g } \%$, the differences between the Jersey and Aberdeen Angus BEI may be negligible. A study is now being planned of SPI and BEI of Jersey and Aberdeen Angus sera.

Discussion. The average amount, 1.6 $\mu\text{g } \%$, by which the SPI of 5 nonpregnant, lactating Jerseys exceed the BEI is greater than the average difference, 0.5 $\mu\text{g } \%$, by which the SPI exceeds the BEI of men and women who are not receiving inorganic iodide. This greater difference for cattle may be the result of the iodide contained in the salt blocks available to the dairy animals. For this reason variations in SPI of cattle receiving iodized salt may not be indicative of differences in thyroid activity unless the averages of SPI vary by more than 1.6 $\mu\text{g } \%$, the average difference between SPI and BEI of mature animals. The greater differences between SPI and BEI in young calves may be the result of greater differences in the ingestion of iodized salt.

Summary. Four comparisons (10 sera in each comparison) were made of the SPI content of Jersey and Aberdeen Angus sera. The

average SPI of the Jersey sera was 5.0 $\mu\text{g } \%$, whereas that of the Aberdeen Angus sera was 4.2 $\mu\text{g } \%$. In a comparison of Jersey and Brown Swiss sera the former was found to be higher in PBI than the latter, 4.6 and 3.7 $\mu\text{g } \%$ respectively; the average BEI of the sera, however, was identical (2.5 $\mu\text{g } \%$). The range of SPI for Jersey and Brown Swiss heifers was so great that no comparison is justified; the average BEI was 2.5 $\mu\text{g } \%$ and 3.9 $\mu\text{g } \%$ respectively.

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Effect of BAL on Survival of Rats after Lethal Doses of Polonium.*† (19324)

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Earlier experiments(1) have shown that

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BAL (2,3-dimercaptopropanol) administered intramuscularly after single intravenous doses of polonium doubles the 10-day total excretion

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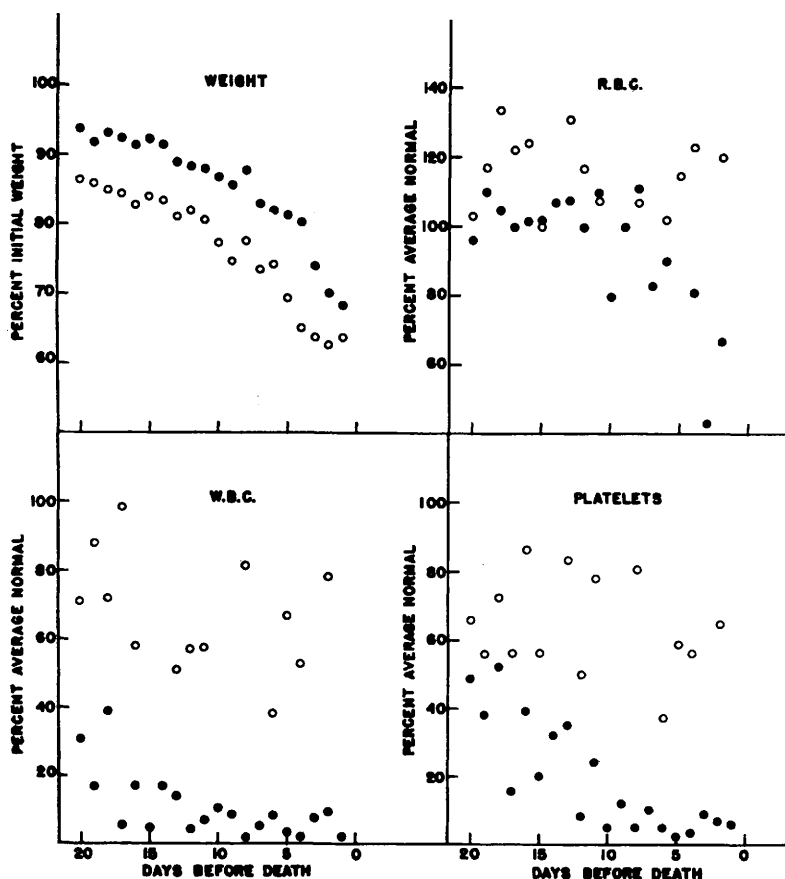


FIG. 1. Average loss of weight and average changes in formed elements of circulating blood are plotted for 20 days preceding death. The dots refer to the control group of rats, open circles to the BAL-treated group.

and brings about a shift of polonium from the bone marrow, spleen, and testis into muscle tissue. On the basis of both these effects it was believed that BAL would significantly modify the toxicity of polonium and the following experiments were performed to test that belief.

Methods. Eighteen male, Wistar, albino rats averaging about 250 g in weight were injected with a polonium dose of $36 \mu\text{C}/\text{kg}$ introduced into the caudal vein of the unanesthetized rat. Immediately thereafter 9 rats received an intramuscular injection of 0.04 ml of 10% BAL in peanut oil, followed by subsequent dosage at the rate of 3 doses per day, 3 hours apart for 3 days. Each rat was weighed daily and clinical signs were noted in the record. Blood counts were made twice a week for the first 40 days, once a week for

the next 40 days, and once every 2 weeks for the next 58 days.

Results. The survival times in days as found for the 9 BAL-treated and 9 control rats were as follows:

BAL-treated: 37, 57, 58, 75, (89), 103, 174, 219, 250
Control: 18, 19, 20, 20, (22), 23, 26, 29, 32

Fig. 1 presents the average weight in per cent of initial weight, the average per cent normal red blood count, white blood count, and platelet count for the 20 days immediately preceding death. Since each rat was weighed daily, each point on the weight curve is the average of nine values. Since the blood sampling was staggered, the average value plotted for a single day is based on data from 2 to 4 rats.

The average normal values used to calculate the data in Fig. 1 are derived from an unpub-

lished study of 116 adult male rats of the Wistar strain yielding per cu mm blood: for red blood cells, 8.55 millions (range 7-11); for white cells, 18.82 thousands (range 6-30); for platelets, 8.0 hundred thousand (range 5-10).

The clinical signs recorded for the rats dying in the early stages (the control rats) presented the typical radiation injury syndrome, *i.e.*, lassitude, rough coat, bloody nose, loss of appetite. The BAL-treated animals which died at longer times displayed lassitude and loss of weight without the hematological signs.

Discussion. The median survival time of the untreated rats injected with a polonium dose of 36 $\mu\text{C}/\text{kg}$ was 22 days. This compares with a figure of 25 days taken from a curve relating survival time and dose of polonium published by Fink and others(2). The corresponding median survival time for the BAL-treated group was 89 days, which is equivalent to a dose of 17.8 $\mu\text{C}/\text{kg}$ based on the Fink curve.

It may be shown that the increased polonium excretion promoted by BAL does not completely account for the prolongation of survival time. On the basis of excretion data published earlier(1) it is known that at the end of 10 days, BAL-treated rats retained 46.5% of the dose and untreated rats retained 74.3% of the dose. After 10 days excretion is not significantly affected by BAL treatment. Therefore, taking account only of excretion we might predict that the treated group would behave as if the effective dose were no less than $46.5/74.3 \times 36 = 22.4$ $\mu\text{C}/\text{kg}$ which is equivalent to a median survival time of 57 days on the basis of the Fink curve.

The experimental finding of 89 days median survival time suggests the conclusion that the detoxifying effect of BAL cannot depend only upon increased excretion but must also consist

in a mobilization of polonium from radiation-sensitive tissue such as bone marrow and spleen into radiation-resistant muscle tissue.

The effect of BAL is reflected in the contrasted hematology of the treated versus the untreated rat groups just prior to death. Fig. 1 shows that the untreated animals suffer precipitous falls in red and white cell, as well as platelet, counts. Damage to the hemopoietic system was clearly a major cause of death in this group. On the other hand, while the average white cell and platelet counts of the treated animals were reduced somewhat from average normal values, the reduced levels were well maintained and probably presented no serious functional handicap. Beyond the loss of weight preceding death, a common late effect of lethal radiation, the experiment provides no information as to the cause of death for the treated group.

Summary. (1) Control rats injected with a lethal dose of polonium (36 $\mu\text{C}/\text{kg}$) had a median survival time of 22 days, whereas the BAL-treated group had a median survival time of 89 days. (2) Blood studies of control rats showed the typical picture of hemopoietic failure in contrast with well sustained cell and platelet levels found for the BAL-treated group. The difference is believed to be due to a reduced radiation exposure of the spleen and marrow caused by the action of BAL in accelerating excretion and in diverting polonium from the hemopoietic organs into muscle.

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