

sumption probably accounts for the higher dry matter and iodine content of the glands, since the diet is not entirely devoid of iodine. The normal values for rats of similar age in our colony are: thyroid weight 10 mg. per 100 gm. body weight, dry matter 32%, iodine on dry basis 0.200%.³ When compared with these values, the rats of all groups had goiter, and increasing chloride up to the point where it began to retard growth did not affect the severity of the condition, with the possible exception noted above. That the enlargement is not as great as has been reported in previous experiments is probably to be attributed to variations in the iodine content of the yellow corn meal.

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Lipid Content of Blood Leucocytes Following Hemorrhage in Rabbits.

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Harlow and Selye¹ reported that a neutrophilic leucocytosis occurred in rats and mice following the administration of various acute, non-specific, damaging stimuli and they considered that this leucocytosis was part of a general response to such types of stimuli, a response which they described as an "alarm reaction". The "alarm reaction," according to Selye,² represents the first stage of a "general adaption reaction," the "stage of alarm" being followed by a "stage of resistance" and finally, if sufficient stimulation be continued, by a "stage of exhaustion." During the course of certain experiments,³ one-quarter of the total blood volume was removed from rabbits and it was thought that such a stimulus might provide an "alarming stimulus" and evoke an "alarm reaction." This amount of blood was taken as quickly as possible by either cardiac puncture or from the marginal ear veins and constituted a distinct shock to the animal; it was the largest amount of blood which could be removed thus and not cause a considerable number of deaths; even removing this amount produced an occasional death.

³ Remington, R. E., Remington, J. W., and Welch, S. S., *Anat. Rec.*, 1937, **67**, 367.

¹ Harlow, C. M., and Selye, H., *Proc. Soc. Exp. Biol. and Med.*, 1937, **36**, 141.

² Selye, H., *Endocrinol.*, 1937, **21**, 169.

³ Boyd, E. M., and Stevenson, J. W., *J. Biol. Chem.*, 1937, **122**, 147.

Total and differential leucocyte counts were done on these animals before and after bleeding. The phospholipid and free cholesterol contents of the blood leucocytes were simultaneously estimated by methods previously used.⁴ Thirty young, healthy rabbits were employed, 30-40 cc. of blood removed without anesthesia and a second sample of the same volume taken after an interval of 3 hours in one group of 6 animals, after 6 hours in a second group, and after 12, 24, and 48 hours in 3 further groups.

The hemorrhage produced a consistent decrease which averaged 25% in the concentration of hemoglobin and in the red cell count with no evidence of regeneration of red cells or hemoglobin up to and including the 48-hour interval. The mean values and their standard deviation of the total white cell count, phospholipid and free cholesterol of the blood leucocytes before bleeding are given in Table I. The mean values only are listed at the various inter-

TABLE I.
The Mean Phospholipid and Free Cholesterol Content of the Blood Leucocytes Following Bleeding in Rabbits.
Lipid values are expressed in mg. per 100 gm. moist weight.

Hours after Bleeding	Total Leucocyte Count	Phospholipid	Free Cholesterol
0	8,450 ± 2,030	944 ± 424	281 ± 122
3	8,800	1,243	257
6	8,100	1,444	473
12	8,100	884	308
24	12,000	1,150	261
48	8,800	846	273

vals after bleeding since it is obvious that no statistically significant change could occur with the slight and variable differences in these latter means. The total white cell count did not rise appreciably above nor fall appreciably below the initial, indicating that the leucocytes lost during bleeding were rapidly replaced (in less than 3 hours) but that no leucocytosis occurred up to 48 hours after bleeding. There was likewise no consistent change in the differential count with the exception that within the first 6 hours after bleeding all of the animals showed relatively more polymorphs and the lymphocytes increased in number between the 12th and 24th hours.

There was no consistent change in either the phospholipid or free cholesterol content of the white blood cells following bleeding. By usual statistical methods, the values of free cholesterol at 6 hours were significantly elevated but this did not appear likely in view of

⁴ Boyd, E. M., Blenkinsop, J. W., and Mylks, G., PROC. SOC. EXP. BIOL. AND MED., 1937, **36**, 300.

the other results. It would thus appear that the leucocytes mobilized into the blood stream following hemorrhage have the same lipid composition as those previously present.

Summary. Removal of one-quarter of the blood volume of rabbits produced no appreciable change during 48 hours in the phospholipid and free cholesterol content of the white blood cells and no leucocytosis.

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Transfer of Acquired Resistance to Transplantable Leukemia in Mice.*

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It has been demonstrated that graduated doses (cell-immunization) of leukemic cells¹ and certain normal tissues^{2, 3} can induce resistance to transplantable leukemia in normally susceptible mice. Following cell-immunization injected leukemic cells immediately develop small lesions which soon become necrotic.⁴ Lesions do not form immediately following leukemic inoculation of strain C58 mice made resistant by Sto-Li foetal tissues. Once resistance to line I leukemia has been established by means of graduated cell doses it is permanent, while resistance caused by normal tissue implantation may be only temporary with a peak of effectiveness in C58 mice 3 days after implantation of Sto-Li tissue (unpublished data). Of further interest in this problem is the question: Can resistance induced by these 2 methods be transferred to normally susceptible mice?

Accordingly, spleen and liver were removed from cell-immunized mice, minced and injected into susceptible C58 mice. Another

*Aided by a grant from the Carnegie Corporation.

¹ MacDowell, E. C., Taylor, M. J., and Potter, J. S., *PROC. SOC. EXP. BIOL. AND MED.*, 1934, **32**, 84.

² MacDowell, E. C., Taylor, M. J., and Potter, J. S., *Proc. Nat. Acad. Sci.*, 1935, **21**, 8, 507.

³ Rhoads, C. P., and Miller, D. K., *PROC. SOC. EXP. BIOL. AND MED.*, 1935, **32**, 817.

⁴ Potter, J. S., and Findley, M. D., *PROC. SOC. EXP. BIOL. AND MED.*, 1935, **32**, 1338.