

sectomy were found to abolish the ovarian hyperemia response to human chorionic gonadotrophin. Corticotropin and cortisone acetate restored this effect in hypophysectomized rats, and cortisone acetate, as well as other steroids, in the adrenalectomized rats.

Adrenalectomy but not hypophysectomy prevented this response to an anterior pituitary preparation. Cortisone acetate reinstituted the effect in the adrenalectomized anterior pituitary injected animals.

Received May 14, 1951. P.S.E.B.M., 1951, v77.

Heme Synthesis and Erythrocyte Life Span in the Cat.* (18737)

W. N. VALENTINE, M. L. PEARCE, RICHARD F. RILEY, ESTHER RICHTER, AND
JOHN S. LAWRENCE.

From the School of Medicine, University of California at Los Angeles.

The interpretation of the hematologic syndromes resulting from exposure to ionizing radiations depends in large measure on the life span of the formed elements in the circulating blood(1,2). The rate of utilization of leukocytes(3) and thrombocytes(4) has already been determined for the cat. In other experiments(5,6) the functional regeneration of myeloid and erythroid elements in the cat was compared. In order to interpret this work a measure of the life span of the red cell in this species was needed.

While a variety of methods have been used for determining erythrocyte life span, the method of isotope tagging of the porphyrin ring is probably the least objectionable and was the one chosen.

Material and methods. Five apparently healthy adult cats from the stock colony, immune to infectious feline agranulocytosis,

were used. They were maintained on a diet of canned commercial dog food, a small weekly allotment of fresh horse meat and an admixture of ground Rockland Rat Chow. Each was given 100 mg/kg of glycine- N^{15} containing 63.3 atoms % excess of N^{15} . The isotopic glycine divided into 15 doses was fed over a period of 3 days. The animals weighed 2.7 to 3.1 kg with a mean of 2.9 kg. Erythrocyte counts, white blood cell counts and hemoglobin values were determined weekly. Blood samples for analysis were obtained at about 2 week intervals by placing the animals under light ether anesthesia and cutting down on the femoral artery with the usual aseptic precautions. The blood samples were drawn in oxalate, the packed red cells re-suspended in physiological saline, re-centrifuged and finally lysed with distilled water. Hemin was obtained from the lysed cells by routine procedures(7,8). The hemin N^{15} concentrations were determined on a Consolidated-Nier Isotope Ratio Mass Spectrometer. The blood volume of each cat was determined at the end of the experiment by the method of Evans Blue dilution.

Results. During the course of the work it became apparent that individual cats gave somewhat different hemin N^{15} concentrations at a time when their red cell heme was maxi-

* This paper is based on work performed under Contract No. AT-04-1-GEN-12 between the Atomic Energy Commission and the University of California at Los Angeles.

1. Lawrence, J. S., Dowdy, A. H. and Valentine, W. N., *Radiology*, 1948, v51, 400.

2. Jacobson, L. O., Marks, E. K., and Lorenz, E., *Radiology*, 1949, v52, 371.

3. Lawrence, J. S., Erwin, D. M., and Wetrich, R. M., *Am. J. Physiol.*, 1945, v144, 284.

4. Lawrence, J. S. and Valentine, W. N., *Blood*, 1947, v2, 40.

5. Valentine, W. N. and Pearce, M. L., To be published.

6. Valentine, W. N., Pearce, M. L., and Lawrence, J. S., To be published.

7. Shemin, D. and Rittenberg, D., *J. Biol. Chem.*, 1946, v166, 621.

8. Fischer, H., *Organic Synthesis*, Edited by N. L. Drake, John Wiley and Sons, Inc., New York, 1941, v21, 53.

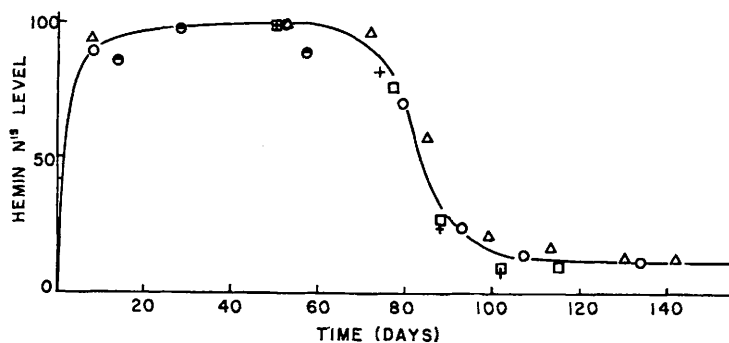


FIG. 1. Composite curve for 5 animals derived from corrected hemin N^{15} excess ratios expressed as per cent of plateau values.

mally labeled (about 50 days). Further, it was apparent that repeated sampling of blood, the volume of each sample being approximately 10% of the circulating blood volume, was in itself decreasing the hemin isotope level. Accordingly, two corrections were applied to the data to make them more comparable and to provide a mean curve from which the half-life of the red cell could be calculated. First each value was multiplied by a factor derived from the sample volume and the cat's blood volume, itself corrected for dilution by previous sampling. Second, observed N^{15} excess ratios, corrected for sampling, were expressed as percentages of the

plateau N^{15} level for that animal. The corrected data are presented in Fig. 1.

Using the method of calculation presented by Shemin and Rittenberg(9), the mean life span, corrected for the initial labeling period, was determined to be 77 days, while half of the cells had a life span between $72\frac{1}{2}$ and $81\frac{1}{2}$ days.

Summary. The mean life span of the erythrocyte in the cat as determined by labeling heme with N^{15} is 77 days.

9. Shemin, D. and Rittenberg, D., *J. Biol. Chem.*, 1946, v166, 627.

Received May 15, 1951. P.S.E.B.M., 1951, v77.

Passive Sensitization of Human Skin by Sera of Guinea Pigs Anaphylactically Sensitized to Ovalbumin. (18738)

WILLIAM B. SHERMAN AND E. J. COULSON.

From Institute of Allergy, Roosevelt Hospital, New York and Allergen Research Division, Bureau of Agricultural and Industrial Chemistry, U. S. Department of Agriculture, Washington.

Previous studies have shown that immediate urticarial reactions of the Prausnitz-Kustner type may be produced in human skin after passive sensitization with sera from rabbits sensitized with ovalbumin and with sera from guinea pigs sensitized to pollens of grass or ragweed(1,2). The present study concerns

the activity in passively sensitizing human skin of sera from guinea pigs sensitized with crystalline ovalbumin, and the relationship of that activity to the capacity of the same antisera to produce passive anaphylactic sensitivity in the guinea pig.

Samples of sera obtained from guinea pigs sensitized by various methods at the Allergen Research Division of the Department of Agriculture were tested in the passive sensitization of human skin at Roosevelt Hospital. Other

1. Sherman, W. B., Menzel, A. E. O., Seebohm, P. M., *J. Exp. Med.*, 1950, v92, 191.

2. Sherman, W. B., Stull, A., and Hampton, S. F., *J. Immunol.*, 1939, v36, 447.