varying periods of time, following the procedure of Howell and Cekada.[9] The blood was collected in paraffin-coated tubes which were kept cool in an ice bath. At regular intervals of time blood smears were made on a glass slide, dried, and stained by Wright's method.[10] The specimens were examined for the relative number of platelets and for the degree of agglutination that had occurred. The data presented in Table III show that the platelets disintegrate more rapidly in normal than in abnormal blood. These studies indicate that the platelets from defective animals are abnormally stable, and at present this is the most obvious explanation of the abnormalities observed.

The symptoms resemble hemophilia more closely than they do any other disease that has come to our attention.

TABLE III.
Stability of Platelets.

Type of animal*	Min. after collection	No. platelets per 1000 erythrocytes	Remarks
Normal	5	39	A considerable number adhering in pairs
Defective	5	32	No agglutination
Normal	30	17	Agglutinated and ragged
Defective	30	27	No agglutination

*The data are averages of 2 normal and of 2 defective animals.

13276

Return of Hepatic Vitamin A in Rats after Depletion by Methylcholanthrene.

T. U. MARRON. (Introduced by H. A. Mattill.)

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Goerner[1] demonstrated that a gelatin suspension of 1,2,5,6-dibenzanthracene injected intraperitoneally into rabbits markedly decreased the vitamin A content of the liver mitochondria. In surviving animals the vitamin content of these structures was slowly restored after the drug was discontinued. Later Goerner and

[9] Howell, W. H., and Cekada, E. B., *Am. J. Physiol.*, 1926, **78**, 500.

[10] Wright, J. H., *J. A. M. A.*, 1910, **55**, 1979.

[1] Goerner, A., *J. Biol. Chem.*, 1937, **122**, 529.

Goerner,[2] in examining livers from dibenzanthracene-treated rabbits and rats, found decreases in hepatic vitamin A that ranged to zero.

At the time the latter paper appeared we had completed analogous experiments on the Sprague-Dawley strain of white rats. Twenty-five animals of both sexes and of various ages were given intraperitoneal injections of methylcholanthrene (Eastman) in lard filtered through a medium paper at 37°C. Doses of 6 mg or less were given over a period of 3 weeks; some animals received more frequent doses than others. For every methylcholanthrene animal another was similarly injected with phenanthrene in 37° lard to find the effect of that non-carcinogenic hydrocarbon. Controls for both groups were animals injected with the lard alone. Animals were sacrificed at periods of one to 20 days after the last injection, and the excised whole livers were digested and extracted by a modification of the method of Davies.[3] Vitamin A was determined, excluding carotene, by a modified Carr-Price reaction.[4, 5] All of the rats that had received 22 to 54 mg of methylcholanthrene and had been allowed to live 22 days or more showed very little or no hepatic vitamin A. The average liver weight, expressed as percent of body weight, was greater than that of the controls or stock animals, but not abnormal according to Donaldson.[6] The livers of phenanthrene and lard treated animals compared well in vitamin A content and size with stock animals examined.

These animals had not been allowed to live long enough after the drug administration had been discontinued to demonstrate any recovery effect similar to that mentioned by Goerner[1] for rabbits that had received dibenzanthracene. Nor did Goerner and Goerner[2, 7] allow dibenzanthracene-treated rats to survive so that studies of the hepatic vitamin A could be made in the recovery period. Therefore, 20 white rats of an unknown, inbred strain, weighing 100 to 200 g, were each given 2 intraperitoneal injections of 6 mg methylcholanthrene in 37° lard 30 days apart. About 75 days after the last injection normal weight gains were observed; and 10 days later half of the animals with an equal number of lard controls of both sexes were sacrificed. Fluorescence microscopy[8] was substituted

2 Goerner, A., and Goerner, M. M., *Am. J. Cancer*, 1939, **37**, 518.

3 Davies, A. W., *Biochem. J.*, 1933, **27**, 1770.

4 Rosenthal, E., and Erdelyi, J., *Biochem. Z.*, 1933, **267**, 119.

5 Rosenthal, E., and Wiltner, M., *Biochem. J.*, 1935, **29**, 1036.

6 Donaldson, H. H., *The Rat*, Memoirs of the Wistar Institute, No. 6, 1924.

7 Goerner, A., and Goerner, M. M., *J. Nutr.*, 1939, **18**, 441.

8 Popper, Hans, PROC. SOC. EXP. BIOL. AND MED., 1940, **43**, 133.

for a chemical method of detecting vitamin A. The methylcholanthrene-treated animals were found to have hepatic vitamin stores equal to those of the lard controls and stock animals. After 9 months the remaining animals were still normal as to both vitamin A stores and liver histology.

The purity of the hydrocarbon used was confirmed by the manufacturer, and a new rechecked lot was furnished, with which the experiments were repeated on 10 animals with 5 lard controls. The dosage was 20 mg methylcholanthrene divided into 3 portions given during 30 days. Weight gains were resumed about 90 days after the first injection. At 120 days the livers of the 15 animals were alike as to normal stores of vitamin A[8] and absence of liver pathology; complete blood counts were normal as established for the colony.

All of the animals in the 3 experiments were on a stock diet of Purina Dog Chow. No exceptional loss of appetite was ever noticed. No tumors ever appeared; and no animals died except of internal injury at injection.

Conclusion. Methylcholanthrene will deplete rat livers of vitamin A in 3 to 6 weeks; phenanthrene or lard injections will not. Some strains of white rats are not readily susceptible to tumor production by intraperitoneal injections of the drug and regain their ability, after discontinuance of injections, to store vitamin A in the liver.

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13277

Non-Effect of Ovariectomy on the Twenty-fifth Day of Pregnancy in the Rhesus Monkey.

CARL G. HARTMAN. (Introduced by George W. Corner.)

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It was demonstrated[1] that the pregnant Rhesus monkey is able to carry its fetus to term despite surgical removal of the ovaries. Five cases were cited which were castrated on days 95, 89, 46, 35, 31 of gestation, respectively. To these it is now possible to add another and more crucial case castrated 25 days after ovulation and fertilization.

[1] Hartman, Carl G., *Anat. J. Obst. and Gynec.*, 1939, **37**, 287.