uranium and thorium with high-energy neutrons, being approximately equal, taken as 100 on an arbitrary scale.

Neutron-reaction	Maximum neutron-energy	Uranium		Thorium	
		No Cd	With Cd	No Cd	With Cd
	Mev				
Li + D ..	13.5	100	70	100	100
D + D ..	2.5	100	70	100	100
C + D ..	0.5	100	10	0	0

From these comparisons it apears that the uranium fissions are produced by different processes for fast and slow neutrons, the fast-neutron process requiring more than 0.5 Mev but less than 2.5 Mev for effective operation. For thorium, on the other hand, only the fast-neutron process is effective, but somewhat surprisingly it also appears to require between 0.5 and 2.5 Mev.

A few words with regard to our present knowledge on the efficiency of these processes may be in order. The capture of a neutron with the energy of one thirtieth of an electron-volt gives rise to the release of 200,000,000 electron-volts of energy, but the production of a single slow neutron requires the expenditure of approximately 3,000,000,000 electron-volts of energy by the bombarding beam in the most efficient process yet known (deuterons on beryllium at 9,000,000 volts).

It may also be of interest to record that the measurements on this extremely interesting new process in uranium and thorium were the first experiments carried out with our new 5,000,000-volt equipment for nuclear physics, aside from nuclear measurements performed for voltage-calibration only. We take pleasure in recording our obligation to Dr. John A. Fleming, director of the department, for his vigorous support of our program in fundamental physics.

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SPECIAL ARTICLES

POSSIBLE AVITAMINOSIS K PRODUCED IN MICE BY DIETARY MEANS

RECENT work on the possible relation of a deficiency of the fat-soluble factor, vitamin K, to the bleeding tendency in obstructive jaundice,[1] and loss of coagulability of blood in bile fistula dogs[2] and bile fistula rats[3] suggests that an avitaminosis K might be produced in mammals by dietary means alone. However, as far as the present writer is aware, a hemorrhagic disease in mammals comparable to that in chicks[4,5,6] has not as yet been produced. Consequently, this brief report is being made of a bleeding tendency occurring in mice maintained on a diet low in vitamin K and prevented by supplementation with the vitamin in the form of an ether extract of alfalfa.

In connection with a series of experiments concerned with the nature of the raw egg-white syndrome as produced in mice, it was apparent that when the tails were clipped in order to obtain blood bleeding continued for a longer time than is normally expected. Comparison of the clotting time with that of mice on a stock diet of Dog Chow showed that it took approximately twice as long for the blood of the experimental animals to coagulate as it did for the stock mice.

The diet that was used to produce the syndrome was made up as follows: powdered egg albumin (Merck), 61 per cent.; cornstarch, 27 per cent.; brewer's yeast, 5 per cent.; salt mixture, 4 per cent.; cod liver oil, 2 per cent.; agar, 1 per cent. This diet is relatively low in fat and might, therefore, be expected to contain only a limited amount of the fat-soluble factor. In addition, the albumin which makes up a large proportion of the diet has been reported to lack vitamin K[7] and yeast has been found to contain little or none of this factor.[8] To determine whether a vitamin K deficiency did exist, bleeding time was determined by Duke's method, using the clipped tails of three groups of mice: (1) stock mice on Dog Chow, (2) mice on the basal diet described above, and (3) mice on the basal diet supplemented with an ether extract of alfalfa equivalent to 5 per cent. of the diet. The tests were done after the animals had been on the diets for four to five weeks. Typical results are given in Table I.

TABLE I

Diet	Number of mice	Average bleeding time, minutes
Dog Chow	8	4.6
Basal	17	10.8
Basal plus extract of alfalfa	14	4.9

[1] E. D. Warner, K. M. Brinkhous and H. P. Smith, *Proc. Soc. Exper. Biol. and Med.*, 37: 628, 1938.

[2] W. B. Hawkins and K. M. Brinkhous, *Jour. Exper. Med.*, 63: 795, 1936.

[3] J. D. Greaves and C. L. A. Schmidt, *Proc. Soc. Exper. Biol. and Med.*, 37: 43, 1937.

[4] H. Dam, *Biochem. Jour.*, 29: 1273, 1935.

[5] H. J. Almquist and E. L. R. Stokstad, *Jour. Biol. Chem.*, 111: 105, 1935.

[6] F. Schonheyder, *Nature*, 135: 653, 1935. Since this paper was submitted for publication it has been noted that H. Dam and J. Glavind (*Lancet*, 1: 720, 1938) referred to their unpublished experiments in which this condition was produced in rabbits and cured by vitamin K. They gave no details concerning the type of diet used nor the symptomatic picture that resulted.

[7] H. J. Almquist and E. L. R. Stokstad, *Jour. Nutrition*, 12: 329, 1936.

[8] H. J. Almquist, C. F. Pentler and E. Mecchi, *Proc. Soc. Exper. Biol. and Med.*, 38: 336, 1938.

It should be noted that one of the symptoms of the raw egg-white syndrome in mice is the excretion of large amounts of bile salts and bile pigments in the urine. However, the icteric condition is not responsible for the defect in bleeding, since the administration of vitamin K ameliorated the latter without decreasing elimination of the bile constituents.

While the results are not so striking as those reported for chicks, the constancy of the bleeding defect on the basal diet and its prevention by supplements of alfalfa extract suggest that a vitamin K deficiency has been produced. No more definite conclusion can be drawn until measurements of the prothrombin concentration of the blood have been made.

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THE SODIUM FACTOR OF THE ADRENAL[1]

IN our study of the adrenal factor, which causes retention of sodium in normal animals, we discovered that it could be separated from the vital factor, cortin, by repeated extractions with ethyl ether. The cortin content of our extracts was assayed on adrenalectomized cats,[2] while the presence of the sodium factor was determined by the effect on sodium retention in normal dogs.[3] In the latter instance injections were made subcutaneously in order to avoid the development of the refractory state.[4] A six-hour test period was employed.

We have made many preparations with high cortin content but no sodium-retaining power. For example, six different preparations, injected in amounts containing 20 to 80 cat units of cortin, caused no retention of sodium. On the other hand, extracts in which no separation of cortin and sodium factors had been made (which will be called whole extracts) gave very positive results. Seven different preparations injected in amounts containing 30 to 60 cat units of cortin caused a sodium retention of 37 to 54 per cent.

Extracts have also been prepared containing large amounts of the sodium factor but very little cortin, as illustrated in Table I.

TABLE I

Extract	Cortin content Cat units	Sodium retention Percentage
N	3	47
O	0	32
P	2	61
Q	1+	70
R	0	50
S	5	55

The effects of these two factors have been studied on adrenalectomized animals. Two adrenalectomized male cats were treated with cortin alone for 130 days. The results were similar in each, the plasma sodium being maintained at the level characteristic of untreated adrenalectomized animals in the advanced state of insufficiency. The animals remained in good condition, showing no significant change in weight (Table II). Reduction of the cortin to the point of insufficiency produced little change in plasma sodium, while addition of the sodium factor to the cortin treatment caused a rise in plasma sodium to normal levels. Treatment with whole extract (cortin and sodium factor not separated) had a similar effect. The inability of the sodium factor to maintain adrenalectomized cats was demonstrated in the assay for cortin content of such extracts (Table I).

The effect of these factors has also been studied on two adrenalectomized female dogs, of which Dog 19 (Table II) is typical. When whole extract was injected the plasma sodium was maintained at normal levels, while treatment with cortin alone caused it to fall to approximately the level characteristic of adrenal insufficiency. As in the cats, the animals remained in good condition with cortin alone. Addition of the sodium factor to the cortin treatment caused a rise in plasma sodium to normal level.

TABLE II

Days after complete adrenalectomy	Weight Kgm.	Extract	Plasma Sodium mEq./l
		Cat ES	
61	3.70	Cortin alone, enough for maintenance	143.0
110	3.70	Cortin alone, decreased to point of insufficiency	141.0
130	3.66	Cortin alone, maintenance dosage	142.0
137	3.66	Cortin plus sodium factor	151.5
167	3.74	Whole extract	149.8
		Dog 19	
146		Whole extract	140.3
173	8.0	Whole extract	143.0
225	8.0	Cortin alone	130.5
243	8.0	Cortin alone	132.3
250	8.0	Cortin plus sodium factor	143.8

Our evidence indicates that there is a separate adrenal hormone responsible for sodium retention. In the absence of this hormone cortin maintains the adrenalectomized animal in spite of the diminished plasma sodium.

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[1] Aided by a grant from the Rockefeller Foundation.

[2] F. A. Hartman and W. D. Pohle, *Endocrinology*, 20: 795–800. 1936.

[3] G. A. Harrop and G. W. Thorn, *Jour. Exper. Med.*, 65: 757. 1937.

[4] F. A. Hartman, L. A. Lewis and K. P. McConnell, *Endocrinology*. (In press.)