

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 ADRENAL¹

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

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

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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.)

amounts of the sodium factor but very little cortin, as illustrated in Table I.

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 in-

TABLE II

Days after complete adrenal- ectomy	Weight Kgm.	Extract	Plasma Sodium mEq./l
<i>Cat ES</i>			
61	3.70	Cortin alone, enough for maintenance	143.0
110	3.70	Cortin alone, de- creased to point of insufficiency	141.0
130	3.66	Cortin alone, main- tenance dosage	142.0
137	3.66	Cortin plus sodium factor	151.5
167	3.74	Whole extract	149.8
<i>Dog 19</i>			
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

jected 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.

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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