	M
Long diameter of tubule bow	185.0
Short diameter of tubule bow	111.0
Long diameter of glomerular knot	148.0
Short diameter of glomerular knot	61.5
Arterioles of knot	7.5
Maximum epithelial height	9.0
Minimum epithelial height	4.5

Discussion. For a number of years research in this laboratory has very largely concerned itself with a study of the processes of epithelial repair in the kidney and the liver when these organs were effecting such a process as a result of either slight or severe mechanical or chemical injury(3-7). The observation has been made that the factor of tissue difficulty, strain, as these organs attempt their cytological restoration influences the morphological characteristics, the type of epithelial cell, which either transitorially or permanently establishes the process of repair. A slight injury is followed by the reappearance of a normal order of cell for the location in which the injury has occurred. A very severe injury registers its influence in the process of repair by a reversion to an embryonic order of epithelial tissue, frequently syncytial in nature and showing no or very poor cellular differentiation. In a certain number of instances such an order of repair remains over months or even years as a permanent morphological transition while in other instances there would appear to remain in this embryonic order of tissue of repair the urge to return to the differentiated type of cell normal for this area that has participated in the severe injury.

The injury which has been described for the opossum kidney shows itself in a dominant fashion, not in the tubular epithelium of this organ, but to a similar tissue (epithelium) found in the glomeruli. In a certain number of these ancestrally old animals, the marsupials, it would appear that the repair process may also revert to an embryonic status not only in the type of cell which is found in the two layers of Bowman's membrane but furthermore and rarely, this reversion may take on structural proportions with an attempt to form new bodies atypically glomerular in their configuration.

3. MacNider, Wm. deB., *J. Med. Research,* 1911, v20, 369.

4. MacNider, Wm. deB., *J. Med. Research.,* 1916, v29, 177.

5. MacNider, Wm. deB., *J. Exp. Med.,* 49:387.409; Harvey Society Lecture 1928-29.

6. MacNider, Wm. deB., *J. Pharm. and Exp. Therap.,* 1936, v56, 359.

7. MacNider, Wm. deB., *J. Pharm. and Exp. Therap.,* 1937, v59, 393.

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Differential Effect of Desoxycorticosterone and the Sodium Hormone on Eosinophil Count in the Mouse. (18245)

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Certain steroids from the adrenal cortex cause a reduction in the number of circulating eosinophils. Seventeen-hydroxy-11-dehydrocorticosterone and related compounds are most potent. Desoxycorticosterone in large doses will cause eosinopenia(1). Meyer and Speirs(2) have recently modified the Thorn(3) test for the assay of the compounds. It occurred to us that this might be another means of comparing desoxycorticosterone and the sodium hormone. We have been able to show that the sodium hormone is without effect on the eosinophils, while we

1. Speirs, R. S., and Meyer, R. K., *Endocrinol.,* 1949, v45, 403.

2. Meyer, R. K., and Speirs, R. S., *Anat. Rec.,* 1950, v106, 249.

3. Thorn, G. W., The diagnosis and treatment of adrenal insufficiency. Charles C. Thomas, Springfield, Ill., 1949.

TABLE I. Effect of DC and Sodium Hormone on Eosinophil Cells in Mice.

Dose, γ	No. of determinations	Eosinophil count 4 hr	Eosinophil count 7 hr	Change, %
	Sodium hormone			
300	6	62	56	—10
380	6	50	54	+ 8
758	2	25	19	—24
1516	2	53	56	+ 6
	DC			
25	4	62	70	+13
80	4	56	43	—24
150	8	62	22	—65
200	2	31	12	—61

have confirmed the observation that desoxycorticosterone (DC) causes eosinopenia. This furnishes additional evidence that desoxycorticosterone is not the sodium hormone.

Methods. The sodium hormone* was prepared from beef adrenals and separated from the gluconeogenic hormones by chromatography according to the procedure of Thatcher and Hartman(4).

Two strains of mice were used for the test since they showed no difference in the response. These were the C-57 Bar Harbor mice and an albino strain obtained from an Indiana dealer. Because the eosinophil counts vary greatly from time to time depending on the condition of the animal, young animals of the same age, weight, diet, and cage conditions were used(1). Forty-eight hours after adrenalectomy 5 γ of epinephrine hydrochloride were injected subcutaneously to eliminate any response due to adrenal rests. The eosinophil count at the end of four hours was used for comparison with the count made three hours after injection of the substance to be tested. The test substance was injected subcutaneously at the end of the 4 hour period.

Results. The results shown in Table I are typical. Additional determinations were made with similar effects but different dosages. It will be noted that the sodium hormone had no significant effect even in large doses while DC was effective in sizeable doses (150 γ or more).

Discussion. It has been shown previously that sodium hormone and DC differ in their chemical properties(4). They both produce marked sodium retention when injected into normal dogs. The preparations of sodium hormone used in our experiments were recently tested for this property and proven potent.

Speirs and Meyer(1) found that DC in relatively large doses (150 γ) caused maximum eosinopenia. Cortisone is very potent, as little as 5 γ producing maximum eosinopenia (unpublished results in our laboratory).

We conclude from our experiments that the sodium hormone does not produce eosinopenia whereas DC, in comparatively large doses, does.

Summary. Sodium hormone potent in respect to its ability to produce sodium retention failed to produce eosinopenia in adrenalectomized mice while large doses of desoxycorticosterone did. This is further evidence that the two substances are not identical.

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4. Thatcher, J. S., and Hartman, F. A., *Arch. Biochem.*, 1946, v101, 195.