site. At present, the second and third explanations, which may be termed transmission and translation of the stimulus, respectively, seem the most probable, but it is impossible to differentiate between them; we need to know more about the mechanism of flagellar contraction and the way in which it results in directed movement of the organism. However, the fact that it is possible to differentiate between motility in general and directed movement caused by a light stimulus should help to further investigations into the mechanism of the phototactic response (*5*).

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Angiotensin II: Its Effects on Corticoid Production by Chicken Adrenals in vitro

Abstract. *Mammalian angiotensin II, added to the incubation medium, failed to increase corticoid production by chicken adrenal tissue in vitro. Mammalian corticotropic hormone (ACTH) stimulated both aldosterone and corticosterone production.*

Angiotensin II, in addition to its known pressor activity (*1*), has been implicated in the regulation of aldosterone secretion by the adrenal cortex of the human (*2*) and dog (*3*). Kaplan and Bartter (*4*) recently demonstrated that angiotensin II, like ACTH (corticotropin), acts directly on bovine adrenal cortex tissue in vitro, stimulating the production of aldosterone, as well as corticosterone and cortisol. However, angiotensin II failed to stimulate significantly the production of aldosterone and corticosterone by the adrenal cortex of the laboratory rat.

Angiotensinogen, the inactive precursor of angiotensin, has been demonstrated in chicken plasma. Renin, the enzyme responsible for the liberation of angiotensin from angiotensinogen, has been extracted from chicken kidney tissue. The concentration of angiotensinogen and renin in the chicken approximate the concentration determined for the dog, cow, and rat (*5*). Accordingly, it was of interest to examine the possibility that the avian renin-angiotensin system is involved directly in the biosynthesis of aldosterone and corticosterone by the chicken adrenal (interrenal) glands.

Table 1. Effects of angiotensin II and ACTH on steroidogenesis by chicken adrenal tissue. All data presented per 100 mg of incubated tissue.

Angiotensin II (μg)	ACTH (units)	Number of determinations	Steroid production* (μg)	
			Corticosterone	Aldosterone
None	None	4	0.23 ± 0.08	0.33 ± 0.11
5	None	2	0.13 to 0.62	0.13 to 0.55
25	None	4	0.16 ± 0.07	0.15 ± 0.06
50	None	3	0.00 to 0.18	0.00 to 0.18
25	1.0	1	4.90	0.73
None	1.0	4	6.64 ± 0.66†	1.39 ± 0.19†

* Either the mean $\pm$ the standard error, or the range is given. † Significant increase in corticosterone production ($p < .05$) and aldosterone production ($p < .05$) with added ACTH.

One hundred and forty-four, inbred, white leghorn cockerels were obtained as one-day-old chicks and raised in an incubator until they were 32 to 38 days old. Tissue was obtained from the adrenal glands, and after a preincubation period of 30 minutes, the incubation medium was changed and discarded. The tissue was then incubated for an additional 3 hours with a change of medium after 1½ hours. Further details of the procedures used for incubation, extraction, chromatography, and quantitative evaluation, are described by deRoos (*6*). The angiotensin II (*7*) and ACTH (*8*) were added to the incubation medium after the preincubation period and were renewed at the second change of the medium. Different concentrations of angiotensin II were used in three series of experiments. Additional experiments with ACTH, or angiotensin and added ACTH, and control experiments, were performed. The results are presented in Table 1.

The addition of angiotensin II to the incubation medium failed to result in an increase in corticoid production. In contrast, the addition of mammalian ACTH resulted in a significant increase in both aldosterone and corticosterone production.

The failure of mammalian angiotensin II to stimulate corticoid production by the chicken adrenal would not appear to be due to the dose levels employed. A similar experimental procedure, in which 4 μg of angiotensin II per 100 mg of incubated tissue was used, resulted in a significant stimulation of corticoid secretion by the bovine adrenal cortex (*4*). Our data suggest that the renin-angiotensin system is not involved directly in the biosynthesis of aldosterone or corticosterone by the chicken adrenal. The possibility of species specificity within the renin-angiotensin system represents an alternative explanation. Mammalian renin has been reported to cause a pressor response only among mammals, and chicken renin to be active only with chicken blood—that is, angiotensinogen (*5, 9*). However, chicken angiotensin II, obtained by incubation of chicken renin with homologous serum, resulted in a pressor response when administered to rats (*5*). It remains to be demonstrated whether chicken angiotensin II will stimulate corticoid production in mammals. This would appear to be probable, since mammalian studies suggest that the pressor and aldosterone-stimulating activities of angiotensin II depend on the same functional groups (*10; 11*).

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