The rotation of an aqueous solution of l-glyceric aldehyde, if kept in the ice-box, changes in a week's time from $[\alpha]_D^{20°} - 14$ to about -7. Dr. Baer has been able to restore the original optical activity of such a solution by evaporating it and heating the residue to 55–60° C. *in vacuo* for 2 hours. We found that the decrease in optical rotation is not accompanied by any noticeable change in the biological activity: both solutions whether $[\alpha]_D^{20°}$ was -7 or -14 were found to inhibit glycolysis to the same degree when used in equal concentrations.

As dl-glyceric aldehyde (0.5×10^{-2} M.) is known to inhibit respiration of brain cells, experiments were carried out to decide whether this effect is due to the l- or to the d-component. It was found that the inhibition is brought about mainly by l-glyceric aldehyde, whereas the d-form has only a slight effect on respiration. (See figure).

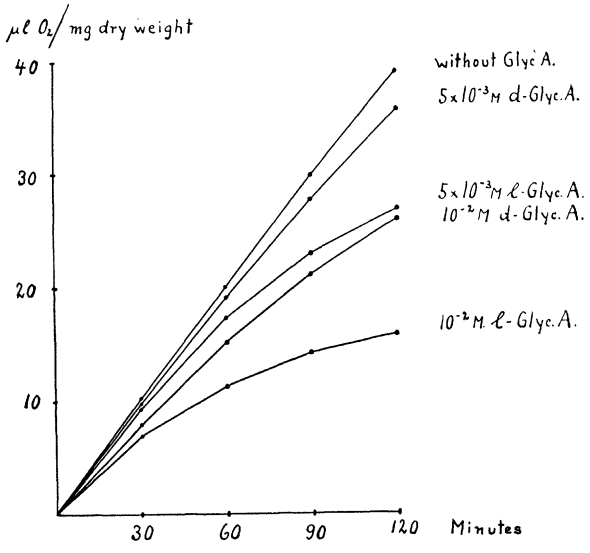

FIG. 1. Effect of d- and l-glyceric aldehyde on respiration of rat brain (grey matter) Ringer Phosphate, pH = 7.4.

The respiration of tumors is less sensitive to glyceric aldehyde than the respiration of brain, and the differences between the two optical forms were found to be less marked in experiments on sarcoma 39.

d- and l-glyceric aldehyde are fermented at the same velocity by slices of rat liver under anaerobic conditions.

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A THEORY OF CORTICO-ADRENAL AND POST-PITUITARY INFLUENCE ON THE KIDNEY

THE possibility of an important relationship between the adrenal cortex and the posterior pituitary lobe in regard to their actions on the kidney has been discussed in previous papers.[1,2,3] We have recently carried out experiments which confirm our earlier work and give fairly clear indication of the mode of action of the hormones concerned. Tests were made of responses of female opossums to orally administered water, salt and urea, and the influence of cortico-adrenal extract and post-pituitary solution (Squibb) was determined. Both normal and adrenalectomized opossums were utilized, and more than three hundred separate experiments were made in this study.

The results are given in summary in Table 1. It

TABLE 1

EFFECT OF CORTICO-ADRENAL EXTRACT AND POST-PITUITARY SOLUTION (SQUIBB) ON RENAL SECRETION

Fluid administered†	Hormone administered	Normal opossums			Adrenalectomized opossums		
		Average water output, cc.*	Concentration in mgm./cc.		Average water output, cc.*	Concentration in mgm./cc.	
			Chlorides	Urea		Chlorides	Urea
Water	none	16.4	0.21	4.51	8.1	0.32	6.66
	cortico-adrenal extract	17.6	0.32	3.58	16.9	0.30	4.91
	post-pituitary solution	5.7	6.50	18.2	3.0	1.72	25.0
NaCl (0.9 %)	none	13.2	7.12	6.51	5.2	7.50	10.2
	cortico-adrenal extract	22.0	6.37	4.18	12.2	4.58	7.60
	post-pituitary solution	17.0	10.0	6.77	8.8	11.1	10.3
NaCl (0.9 %) plus Urea (2 %)	none	10.0	7.60	40.6	8.8	4.20	38.6
	cortico-adrenal extract	15.2	6.31	27.2	13.3	3.31	33.3
	post-pituitary solution	14.8	8.51	26.5	...	...	...

† 20 cc./kilo body weight/2 hours.
* Each figure represents an average of 12–21 metabolic runs. Water excretion on basis of cc/kilo body weight/2 hours.

will be observed that in every case the action of cortico-adrenal extract on chloride excretion (per cc of urine) by the kidney is the direct opposite of that of the post-pituitary effect. Further, in every case the elimination of water under the influence of adrenal extract is greater than that under post-pituitary solution. It would appear that the latter hormone acts to bring

1 H. Silvette, *Am. Jour. Physiol.*, 117: 405, 1937.
2 H. Silvette and S. W. Britton, *Am. Jour. Physiol.*, 121: 528, 1938.
3 H. Silvette, *Proc. Am. Physiol. Soc.*, fiftieth annual meeting, Baltimore, 1938, p. 188.

about a highly concentrated urinary output, containing large amounts of both chlorides and urea, while the hormone of the adrenal cortex induces a large urinary volume having a relatively low concentration of urea and chlorides. It is noteworthy, too, that the concentrations of both urea and chlorides in the urine tend to vary together, in one direction or the other, according to the hormone used.

A true diuretic substance is said to be one which will "consistently and reproducibly elevate the urine flow, as determined by catheterized specimens in well-controlled experiments, from moderate rather than very low levels; the diuresis should reach a rate at least comparable to that observed after a moderate dose of water, and it should persist for a period of at least 30 minutes."[4] In our experiments the hormone of the adrenal cortex as a diuretic factor fulfils these conditions in every respect.

The hypothesis that the cortico-adrenal hormone acts as a diuretic agent and in antagonism to the antidiuretic hormone of the posterior lobe of the pituitary explains the puzzling fact that hypophysectomized animals show only a transient polyuria, while animals from which the posterior lobe alone is removed show a persistent diuresis. When the posterior lobe only is excised, lack of its antidiuretic hormone plus the presence of the diuretic adrenal hormone would lead to polyuria through decreased water reabsorption from the renal tubules. When both lobes are removed, the uncompensated activity of the diuretic cortico-adrenal hormone would persist until the adrenal cortex (in the absence of the corticoadrenotropic hormone of the anterior lobe) becomes atrophic, at which time both posterior-lobe antidiuretic hormone and cortico-adrenal diuretic hormone would be absent from the bloodstream and polyuria would disappear.

From many considerations, now omitted for lack of space, it appears that while post-pituitary action may be exerted chiefly in the direction of inhibition of the aqueous portion of urinary excretion by the kidney, the influence of cortico-adrenal extract (in so far as its renal action is concerned) is directed towards increased water elimination. Thus the present experiments seem to establish the physiological antagonism of the post-pituitary and cortico-adrenal principles in their action on urinary secretion.

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[4] H. W. Smith, "The Physiology of the Kidney," New York, 1937, p. 229.

ISOLATION OF THE FUNGUS CAUSING THE RED STELE OR RED CORE DISEASE OF STRAWBERRIES

A SERIOUS root rot of the strawberry was described in Scotland in 1926, under the name "Lanarkshire disease."[1] The term "red core root" was proposed for the disease in 1929,[2] as being descriptive of its most characteristic symptom. A disease presumably identical with "red core root" was first reported in the United States in 1935 from Illinois, under the name "black-stele root rot."[3] In 1937[4] the "black stele" or "red stele" disease was reported from Virginia, Maryland, New Jersey, New York and Michigan, in addition to Illinois. This disease now appears to be spreading in the Middle Atlantic States and is becoming decidedly injurious in restricted areas where soil and climate are favorable to its development.

The red-stele disease may be easily distinguished from other strawberry root rots by the fact that the causal organism, while completely destroying fine, fibrous roots, advances up the central cylinders of larger roots, killing the interiors and causing them to turn dark red, while the surrounding cortex still appears alive and healthy. The fungus eventually advances to the stem of the plant, but apparently does not invade it. The larger roots die gradually, beginning at the distal ends, some time after the steles have been killed. As a consequence of the injury to the root system, affected plants wilt, older leaves die, and newly formed leaves remain very small with conspicuously short petioles. Badly diseased plants often succumb during the heat of spring and summer, but lightly affected plants grow normally through the summer, only to suffer a renewed attack by the fungus late in the season.

Red stele was first attributed to such causes as poor cultural conditions and parasitism by mycorrhizal fungi and by Pythium species. Since 1929[5] there appears to have been general agreement that the disease is caused by an undetermined species of Phytophthora. Oospores of the Phytophthora type occur commonly and abundantly in diseased roots, and sporangia and zoospores characteristic of the species may be induced by submerging recently infected young fibrous roots in cool water for a period of 18 hours

[1] Claude W. Wardlaw, "Lanarkshire Strawberry Disease, a Report for the Use of Growers (with preface by Professor J. M. F. Drummond)". Carluke (Scot.). Printed by J. Bell, 1926. Issued by Botany Department, University of Glasgow.

[2] Nora L. Alcock, Gard. Chron., 3 (86): 14–15, illus., 1929.

[3] H. W. Anderson, *Phytopathology*, 25: 5, 1935.

[4] J. B. Demaree and G. M. Darrow, U. S. D. A. *Plant Dis. Rptr.*, 21 (22): 394–399, December 1, 1937.

[5] Nora L. Alcock, *op. cit.*