hydroxylamino group of hydroxamic acids does not seem to have been reported.

This work is being extended, and a detailed report will be published at a later date.

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Effect of Posterior Pituitary Hormone on the Release of Adrenocorticotrophic Hormone

WE have recently found that the decrease of ascorbic acid concentration of rats' adrenals due to epinephrine is smaller in rats to which pituitrin has been administered and in dehydrated rats than in normal rats[1]. This suggests that the posterior pituitary hormone depresses the adrenal cortical activity. The questions arise, whether the posterior pituitary hormone acts directly on the adrenal or indirectly through the depression of the anterior pituitary secretion, and which fraction of the posterior pituitary hormone, pressor or oxytocic, is responsible for this effect. The present work was undertaken to clarify these points.

Male adult rats were used. Ascorbic acid concentration was determined by the method of Roe and Kuether[2]. Adrenal ascorbic acid concentration of normal rats was 401 ± 16·4 mgm. per cent. When 0·02 mgm. of epinephrine per 100 gm. of body-weight was injected, this concentration decreased to 257 ± 9·8 mgm. per cent; and it remained at a value of 360 ± 22·1 mgm. per cent after injection of the same amount of epinephrine following that of 20 m.u. of pituitrin. When, in place of epinephrine, adrenocorticotrophic hormone (1 u. per 100 gm. body-weight) was administered, the adrenal ascorbic acid concentration was 258 ± 10·9 mgm. per cent, and in this case pituitrin did not alter the value (264 ± 20·1 mgm. per cent). If pituitrin inhibited the adrenocortical secretion directly, it would be expected to suppress the decrease of the ascorbic acid concentration both after epinephrine and after adrenocorticotrophic hormone. As, however, the effect of pituitrin is only seen when epinephrine is used, it may be inferred that the exogenous posterior pituitary hormone inhibits the release of adrenocorticotrophic hormone from the anterior pituitary gland.

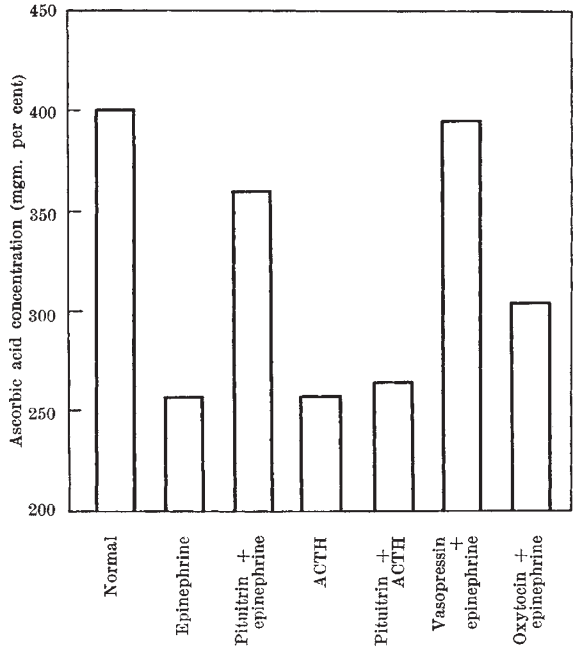

Fig. 1. Changes of adrenal ascorbic acid concentration after epinephrine or ACTH and the effect of posterior pituitary hormone on them

Pressor and oxytocic fractions were separately administered to rats, 20 m.u. per 100 gm. body-weight each, and epinephrine (0·02 mgm. per 100 gm.) was injected, while the adrenal ascorbic acid concentration was determined as in the above experiment. The mean value of ascorbic acid was 395 ± 24·2 mgm. per cent for the pressor fraction and 305 ± 15·8 mgm. per cent for the oxytocic fraction. That is, the former fraction alone shows the above effect.

Details of the experiments will be published in the *Japanese Journal of Physiology*.

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Oxidative Dissimilation of Glycerol studied with Variants of *Bacillus subtilis*

COMPARING the quantitative aspect of growths of two parent strains of *B. subtilis*, we concluded[1] that, in one of them (S_1^-), the glycerol is oxidized through the tricarboxylic acid cycle, whereas in the other strain (M_2^-) the glycerol, although actively oxidized, does not reach the tricarboxylic acid cycle.

It has already been shown that, in animal tissues[2] and in bacteria[3], the oxidation of glycerol goes through the two following steps:

$$\text{Glycerol} + \text{ATP} \rightarrow \text{glycerol-phosphate} + \text{ADP}$$

$$\text{Glycerol phosphate} \xrightarrow{-2H} \text{dihydroxyacetone-phosphate or glyceraldehyde-phosphate}$$

This mechanism cannot explain the results we have recently obtained; and we have been able to show the existence of an alternative pathway consisting of the two reactions:

(1) $\text{Glycerol} + \text{DNP}^+ \rightleftharpoons \text{dihydroxyacetone} + \text{DPNH} + \text{H}^+$.

(2) $\text{Dihydroxyacetone} + \text{ATP} \rightarrow \text{dihydroxyacetone-phosphate} + \text{ADP}$.

We have observed activity due to both the enzymes, glyceroldehydrogenase and triokinase, in cell-free