10181

Succinic Acid and Glucose in Pituitary Ketonuria.

A. W. TERRELL. (Introduced by F. D. W. Lukens.)

From the George S. Cox Medical Research Institute, University of Pennsylvania, Philadelphia.

Deuel, Murray and Hallman[1] have shown that sodium succinate is ineffective in preventing the ketonuria in fasting rats previously fed a high fat diet. Since Rietti[2] has shown that the ketonuria of pancreatic diabetes is reduced by hypophysectomy and since extracts of the anterior pituitary produce ketonuria in the fasted rat, a study of the effect of succinic acid in pituitary ketonuria was undertaken.

Male albino rats weighing between 180 and 200 g were fasted for 3 days in metabolism cages and the urine collected in 20% copper sulphate. Each 24-hour volume was analyzed for total acetone bodies by the method of Van Slyke. The results are expressed in mg per 100 g of body weight of rat per day.

On the 2nd and 3rd day of fasting, anterior pituitary extract was given subcutaneously, 0.5 cc per 100 g, and 2 cc of physiological saline was given intraperitoneally to insure a satisfactory urine volume. Since about 25% of our rats failed to respond well to pituitary extract, only those rats which developed satisfactory ketonuria were used. Among these animals there was a variation in the degree of acetonuria, but since the same group of animals, 17 in number, was used repeatedly for the 3 experiments, this factor is controlled. Between each period of food withdrawal and extract treatment the animals were allowed to regain their initial weight. After the first experiment with fasting and extract, the effect of sodium succinate, food withdrawal and extract was examined. The succinate was given orally for 4 days before as well as during the fasting period. The dose of sodium succinate was 20 mg per 100 g of rat per day which corresponds on a body-weight basis with the dose used clinically by Koranyi and Szent-Györgyi.[3]

In the third series of experiments the same dose of glucose was used (20 mg per 100 g per day).

The results are summarized in Table I, and show the average acetone body excretion for 100 g of rat per day for the 2nd and 3rd

1 Deuel, M. J., Jr., Murray, S., and Hallman, L., PROC. SOC. EXP. BIOL. AND MED., 1937, **37**, 413.

2 Rietti, C. T., *J. Physiol.*, 1932, **77**, 92.

3 Koranyi, A., and Szent-Györgyi, A., *Deutsch. med. Wchnschr.*, 1937, **63**, 1029.

days of extract injection. The reduction of acetonuria associated with succinate and glucose administration is indicated. This diminution is statistically significant except for the 3rd day of succinate. The fact that glucose is more efficient and the fact that succinic acid may be converted to glucose in the body[3] suggest that sodium succinate may exert its effect because of its conversion to glucose.

TABLE I.

Series	No. of Rats	Avg wt	Wt loss, avg	Avg acetone body excretion in mg per 100 g body wt† day of fast		
				1	2*	3*
A	17	185.1	23.8	1.24	21.28 ± 1.80	23.41 ± 2.21
B	17	183.6	26.4	0.94	15.89 ± 0.93	20.95 ± 0.84
C	17	189.2	21.3	1.01	8.74 ± 0.47	10.46 ± 0.52

A = Fasting.
B = Fasting with Succinate
C = Fasting with Glucose.
* Given anterior pituitary extract 0.5 cc per 100 g body weight.
† With standard error of mean.

These results are comparable to the effects of succinic acid and glucose on the ketonuria produced by high fat diets.[1] It appears that experimental ketonuria produced in 2 ways, by high fat diets or by fasting and anterior pituitary extract, may be diminished by succinic acid, but that this substance acts much less effectively than glucose.

10182

Availability of Dibenzoylcystine for Growth of the Young White Rat.

PONG C. JEN AND HOWARD B. LEWIS.

From the Department of Biological Chemistry, University of Michigan, Ann Arbor.

If the α-amino group of cystine was blocked by replacement of a hydrogen atom with a benzoyl group, the sulfur of the resulting dibenzoylcystine was not oxidized readily to sulfate sulfur in the organism of the rabbit, but a large part of the compound was excreted by the kidneys, either unchanged or as the corresponding cysteine derivative.[1] Since the biological oxidation of this α-substi-

[3] Ringer, A. I., Frankel, E. M., and Jonas, L., *J. Biol. Chem.*, 1913, **14**, 539.

[1] Lewis, H. B., Updegraff, H., and McGinty, D. A., *J. Biol. Chem.*, 1924, **59**, 59.