will not reverse the inhibitory effect of azaserine on the reproduction of J-128 cells in tissue cultures. Cells exposed to azaserine increase markedly in size with a parallel increase of nuclei size and DNA content. The drug appears to act quite similarly to certain mustards by stopping cell division between S and G_2. A lesion appears in the central sphere of attraction in azaserine-treated cells, increases in size and apparently causes death of the cell.

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Effect of Diabetogenic Hormones on Transport of Glucose in Small Intestine *in vitro*. (31445)

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The intestinal absorption of glucose is diminished after surgical removal of the hypophysis(1) or the thyroid gland(2). In hyperthyroid patients the rise in blood sugar level in the glucose tolerance test is higher when glucose is fed than when it is injected (3). This indicates the facilitatory effect of the thyroid hormone on intestinal absorption of glucose. The absorption of glucose is greatly reduced in adrenalectomized animals(4). This observation, however, has been contradicted(2,5). Methods employed in the above studies have limitations. We, therefore, studied the effect of the diabetogenic hormones on absorption of glucose by the everted small intestinal sac technique of Wilson and Wiseman (6).

Materials and methods. Male albino rats weighing between 150 and 250 g were used. Different groups of rats were given treatments as follows:

Adrenocorticotrophic hormone (ACTH). One International Unit per 100 g body weight was injected intramuscularly daily for 10 days and the animal was sacrificed for the study of glucose absorption on the eleventh day.

Hydrocortisone and desoxycorticosterone. 2.5 mg hydrocortisone or 1 mg desoxycorticosterone was injected daily to each rat for 6 days and the animal was killed on the seventh day. *Thyroid gland.* 15 mg desiccated thyroid gland powder or 15 mg methyl thiouracil in a watery suspension was fed to each rat for 6 days and the animals killed on the seventh day.

All the animals were fasted for 24 hours before they were killed by stunning and decapitation. Everted sacs of the intestine were prepared(7), filled with Krebs-Ringer-bicarbonate saline with a 0.3% glucose concentration, incubated in a shaking incubator(8) and glucose in the serosal and muscosal fluids was estimated(9).

Results. Significantly increased transference of glucose was observed in the intestinal sacs of rats treated with ACTH, hydrocortisone or desoxycorticosterone. The transference did not change in the intestinal sacs of animals treated with thyroid or methyl thiouracil. The results are given in Table I.

Discussion. ACTH, hydrocortisone and desoxycorticosterone enhanced the intestinal transport of glucose. The effect might have been either a direct action of the hormones on the intestinal mucosal transport mechanism or their indirect effect *via* the adrenal cortex which is stimulated. In hypoadrenocorticism there is increased urinary excretion of sodium

TABLE I. Absorption of Glucose by the Everted Small Intestine of Rats Under Different Treatments.

Treatments	Transference of glucose, mg/g dry sac/1 hr of incubation at 37°C
After inj of ACTH, 1 unit/100 g /day for 10 days (11)	22.18 ± .77*
After inj of hydrocortisone, 2.5 mg/animal/day for 6 days (8)	42.86 ± 4.90
After inj of desoxycorticosterone, 1 mg/animal/day for 6 days (8)	14.42 ± 3.00
After feeding desiccated thyroid, 15 mg/animal/day for 6 days (8)	8.77 ± 1.35
After feeding methyl thiouracil, 15 mg/animal/day for 6 days (12)	8.74 ± 1.62
Normal rats (21)	7.54 ± .94

Figures in parentheses indicate number of samples in study.
* Mean ± S.E.

chloride and increased movement of potassium from cell to plasma(10). Movement of glucose inside the cell has been shown to be accompanied with the movement of potassium in the same direction(11). Stimulation of adrenal cortex by ACTH is, therefore, likely to cause increased transport of glucose in the small intestine. ACTH promotes the entry of glucose inside the cells of the adrenal cortex leading to the increased concentration of glucose-6-phosphate. As a result more TPNH would be formed due to the functioning of the pentose shunt mechanism(12). TPNH would furnish energy necessary for biosynthesis of the adrenal cortical hormones and as a consequence glucose absorption from the small intestine is enhanced.

Treatment with thyroid or methyl thiouracil for 6 days did not alter the intestinal absorption of glucose. This indicates that the thyroid hormone does not participate in the process of glucose absorption from the small intestine.

Summary. Intestinal transport of glucose *in vitro* was studied in rats treated with adrenocorticotropic hormone, hydrocortisone, desoxycorticosterone, thyroid powder and methyl thiouracil by use of everted small intestine sac technique. The concentration of glucose in the medium was 0.3%. ACTH, hydrocortisone and desoxycorticosterone treatments increased significantly the intestinal absorption of glucose. The thyroid hormone did not influence intestinal absorption of glucose in rats.

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