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## Influence of Potassium on Blood Sugar and Tissue Glycogen.

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It has been suggested that most of the disturbances observed after adrenal removal are secondary to a primary defect in potassium metabolism.[1] For the specific case in which we are interested, this is held to mean that the low blood sugar and tissue glycogen of adrenalectomized animals result from a primary rise in serum potassium. Some of the experiments we have been carrying out recently bear on this hypothesis, but lend it no credence.

Normal white rats weighing between 80 and 100 gm. were fasted for 18 hours, given intraperitoneal injections of K-free or K-containing solutions, and then serially sacrificed. When the injected solution consisted of normal sodium chloride and 5% glucose solution (10 cc. per 100 gm. rat), the blood sugar and liver and muscle glycogen curves over 3.75 hours followed the course shown in Table I. Both muscle and (especially) liver glycogen were deposited in large amounts, while the blood sugar level remained relatively stationary. But when the injected fluid contained, in addition to so-

TABLE I.
Effect of Potassium Injections on Blood Sugar and Glycogen.

| Solution containing 0.9% NaCl, 5% glucose and | Animals killed after injection, hrs. | Blood sugar, mg. % | Liver glycogen, % | Muscle glycogen, % |
|---|---|---|---|---|
| No K | 0.75 | 123 | 3.40 | 0.44 |
| | 1.5 | 116 | 3.29 | 0.52 |
| | 2.25 | 132 | 3.38 | 0.59 |
| | 3. | 110 | 2.39 | 0.47 |
| | 3.75 | 117 | 3.64 | 0.44 |
| 0.2% K | 0.75 | 122 | 4.04 | 0.50 |
| | 1.5 | 117 | 2.65 | 0.46 |
| | 2.25 | 131 | 1.52 | 0.43 |
| | 3. | 105 | 2.37 | 0.40 |
| | 3.75 | 98 | 2.10 | 0.41 |
| 0.4% K | 0.75 | 144 | 0.37 | 0.35 |
| | 1.5 | 222 | 0.59 | 0.38 |
| | 2.25 | 141 | 0.88 | 0.34 |
| | 3. | 146 | 0.83 | 0.34 |
| | 3.75 | 102 | 0.74 | 0.39 |

Each figure is the average of 3 experiments.

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1 Truszkowski, R., and Zwemer, R. L., *Biochem. J.*, 1936, **30**, 1345.

dium chloride and glucose, 0.4% potassium (in the form of potassium acetate), the formation of both muscle and liver glycogen was completely suppressed, while the blood sugar level quickly rose over 100%. The administration of this amount of potassium (40 mg. per 100 gm. body weight) produced no visible toxic effects, and the rats when sacrificed were in as good condition as the controls. When half the quantity of potassium was injected (*i. e.,* 20 mg. per 100 gm.), the blood sugar and tissue glycogen values observed did not differ significantly from the control levels.

Other experiments performed on animals under somewhat different conditions (fully-fed; urea-injected, etc.) confirmed the conclusion that the administration of sub-toxic doses of potassium leads to a great increase in blood sugar and a diminution of the tissue glycogen levels; in other words, to glycogenolysis and hyperglycemia. It is pertinent in this connection to recall that MacQuarrie, *et al.,*[2] observed the administration of potassium salts to decrease the glucose tolerance of diabetic children. To contend, therefore, that the increased potassium level observed in adrenal insufficiency is the cause of the lowered blood-sugar levels characteristic of that condition is contrary to the above experimental facts. It is very significant that, in spite of the heightened serum potassium concentration, the blood sugar levels after adrenalectomy are found to be subnormal.

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## Effect of Prolonged Insulin Therapy on Glucose Tolerance in Schizophrenic Patients.

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The treatment of schizophrenic patients by the insulin hypoglycemia method of Sakel[1] has been carried on in this institution for the past year by Cameron and Hoskins.[2] In connection with this treatment a systematic investigation of the changes produced in

2 MacQuarrie, I., Thompson, W. H., and Anderson, J. A., *J. Nutrition,* 1936, **11**, 77.

1 Sakel, M., *Neue Behandlungsmethode der Schizophrenie,* Moritz Perles, Wien und Leipzig, 1935.

2 Cameron, D. E., and Hoskins, R. G., *J. A. M. A.,* in press.