Liver Glycogen in Potassium-Deficient Rats Following Carbohydrate and Alanine Administration, With and Without Potassium. (24057)

CHARLES L. DODGEN AND EDWARD MUNTWYLER
Department of Biochemistry, State University of New York, Downstate Medical Center, Brooklyn

Numerous reports involving both *in vivo* and *in vitro* studies have indicated a relation between carbohydrate and potassium metabolism(1,2). It has been appreciated for many years that glycogen deposited in liver is accompanied by water, and Fenn(3) demonstrated that there is also simultaneous deposition of potassium. Studies with potassium-deficient rats have revealed elevated fasting liver glycogen, normal fasting blood sugar and normal oral glucose tolerance curve (4,5). Intraperitoneal injection of potassium with glucose to normal rats resulted in lesser liver glycogen deposition than when glucose without potassium was injected(6). The present experiments were therefore undertaken to study further the influence of availability of potassium on extent of liver glycogen deposition. For this purpose fasting control and potassium-deficient rats were given by stomach tube glucose, fructose and alpha-alanine, with and without potassium, and liver glycogen determined 4 hours thereafter.

Methods. Young adult male rats of Wistar strain, of uniform age and weighing between 250 and 298 g, were placed on control and low-potassium diets (as in previous studies(7,8)) for 5 weeks. After withdrawing food 48 hours and allowing water *ad libitum*, members of both groups were sacrificed by removing the heart following sodium pentobarbital anesthesia (50 mg/kg body wt., subcutaneous). The liver was quickly excised, blotted free of adhering blood, weighed and sampled at once for chemical analysis. Other members of both groups of rats were simultaneously given, by stomach tube, either 5 ml of 40% glucose or 5 ml of 40% glucose containing 1.2 meq potassium. The animals were then sacrificed 4 hours later. The experiments involving administration of fructose and alanine, without and with potassium, were carried out in same manner. For fructose administration 5 ml of 40% fructose and 5 ml of 40% fructose containing 1.2 meq K were given, while for alpha-alanine administration 5 ml of 20% alpha-alanine and 5 ml of 20% alpha-alanine containing 1.2 meq potassium were given. Liver glycogen was determined by the anthrone reagent(9). All other chemical analyses were carried out in essentially the same manner as previously reported(7,8).

Results. Liver glycogen. In agreement with the findings of others(4,5) the liver glycogen content of fasting potassium-deficient rats was appreciably greater than that of fasting control rats (Table I). There was an unimpaired deposition of liver glycogen in potassium-deficient rats given either glucose, fructose or alpha-alanine. Further, simultaneous administration of potassium appeared to have no significant effect on corresponding levels of liver glycogen.

Liver water and potassium. Liver water/kg fat-free tissue remained relatively constant. As pointed out in earlier studies(3,10,11) a constant percentage of liver water with glycogen deposition indicates addition of water. Likewise, an absence of fall in liver potassium with glycogen deposition indicates addition of potassium(3). A comparison of changes in liver glycogen, water and potassium from corresponding control levels can be made, if it is assumed that during the 4 hours experimental period there was no significant change in total N. On the basis of constant N content, changes in liver water, glycogen and potassium were similar in the 2 groups of animals. Further, it made no difference whether or not potassium was simultaneously administered. In rats fed low-potassium diet with apparent marked skeletal muscle potassium deficiency(7) potassium was made available from some source, so that the ratios of changes in liver potassium to glycogen were in ranges found in normal animals. Although the values were approximately the same within corresponding groups of animals, the increment of liver water/g glycogen showed some

TABLE I. Liver Glycogen of Normal and Potassium-Deficient Rats.

Exp.*	No. of rats	Body wt, g	Liver wt, g	Liver fat, %	Liver/kg fat-free tissue: Water, g	Glycogen, g	K, meq	Na, meq	N, g	Content/g liver N: Water, g	Glycogen, g	K, meq	Change/g liver N: Water, g	Glycogen, g	K, meq	ΔH_2O / Δ Glycogen	ΔH_2O / ΔK	ΔK / Δ Glycogen
C	22	323	7.22	3.67	726.5	5.7	104.7	32.6	37.7	19.27	.151	2.777						
		31.6†	.674	1.049	3.67	4.02	3.76	2.27	.90									
C + G	5	314	7.71	2.68	724.9	38.8	106.4	30.4	33.6	21.57	1.155	3.167	2.30	1.004	.390	2.29	5.90	.39
		19.8	.776	1.027	5.76	18.20	1.97	1.48	1.08									
C + G + K	6	326	7.67	3.46	726.4	33.3	106.2	29.5	34.8	20.87	.954	3.052	1.60	.803	.275	1.99	5.82	.34
		24.3	.722	.563	3.37	5.71	2.76	1.92	1.66									
C + F	6	329	8.36	3.12	725.4	43.4	108.2	31.4	34.5	21.03	1.258	3.136	1.76	1.107	.359	1.59	4.90	.32
		23.4	.882	.580	2.48	7.94	2.18	1.39	1.37									
C + F + K	6	347	8.95	3.38	729.4	44.9	107.5	32.0	33.5	21.77	1.340	3.209	2.50	1.189	.432	2.10	5.79	.36
		30.6	.815	.679	6.78	12.31	4.12	2.86	.77									
C + Ala	6	333	7.98	3.84	733.8	19.6	104.2	32.1	36.3	20.21	.540	2.871	.94	.389	.094	2.42	10.00	.24
		27.7	.598	.586	5.74	8.00	3.49	1.41	.71									
C + Ala + K	6	348	8.13	4.03	730.7	14.7	105.6	32.6	37.0	19.75	.397	2.854	.48	.246	.077	1.95	6.23	.31
		14.0	.380	.797	6.66	6.03	3.35	1.02	1.18									
LK	22	250	6.76	2.04	727.2	13.8	102.0	39.0	36.7	19.81	.376	2.779						
		12.8	.584	.863	5.35	6.09	3.11	2.02	1.14									
LK + G	5	253	7.20	1.23	724.8	42.0	106.5	34.8	33.4	21.70	1.257	3.189	1.89	.881	.410	2.15	4.61	.47
		20.2	.784	.231	4.86	16.31	1.25	1.42	1.55									
LK + G + K	6	253	7.32	1.42	722.2	44.1	110.8	33.0	33.0	21.88	1.336	3.358	2.07	.960	.579	2.16	3.58	.60
		20.8	.990	.076	4.17	9.68	3.88	1.06	2.60									
LK + F	5	234	7.33	1.63	719.9	59.8	106.9	35.4	34.0	21.17	1.759	3.144	1.36	1.383	.365	.98	3.73	.26
		27.2	1.368	.307	5.34	20.29	8.03	.92	3.03									
LK + F + K	6	247	7.96	2.17	720.8	54.2	110.7	33.9	33.5	21.52	1.618	3.304	1.71	1.242	.525	1.38	3.26	.42
		17.9	1.000	.339	5.49	10.96	2.09	1.21	2.54									
LK + Ala	5	226	6.35	2.47	728.5	22.8	106.2	36.6	36.5	19.96	.625	2.910	.15	.249	.131	.60	1.15	.53
		19.2	.671	.249	5.19	9.28	4.23	1.71	1.09									
LK + Ala + K	6	251	6.77	2.62	729.9	21.3	106.3	36.9	36.6	19.94	.582	2.904	.13	.206	.125	.63	1.04	.61
		12.7	.673	.345	4.97	4.64	2.36	1.12	1.37									

* Control (C) or low-potassium (LK) diets for 5 wk, and fasted 48 hr. Glucose, 2 g (G); fructose, 2 g (F), or alanine, 1 g (Ala); with potassium, 1.2 meq (K), as KCl; or without potassium.

† Stand. dev. is listed below each avg value.

variation, and on the average is less than average values of 2.4 ml and 2.7 ml/g reported previously(10,11).

Summary. Changes in liver glycogen, water and potassium were compared in normal and potassium-deficient rats following administration of glucose, fructose and alpha-alanine, with and without potassium. Potassium-deficient rats deposited liver glycogen equally as well as normal rats. The simultaneous increases in liver water and potassium were likewise similar in the 2 groups of animals. No significant differences in changes in liver glycogen, water or potassium were noted with simultaneous administration of potassium.

1. Fenn, W. O., *Physiol. Rev.*, 1940, v20, 377.
2. Hastings, A. B., Renold, A. E., Teng, C. T., *Rec. Prog. Hormone Res.*, 1955, v11, 381.
3. Fenn, W. O., *J. Biol. Chem.*, 1939, v128, 297.
4. Gardner, L. I., Talbot, N. B., Cook, C. D., Berman, H., Uribe, R. C., *J. Lab. and Clin. Med.*, 1950, v35, 592.
5. Fuhrman, F. A., *Am. J. Physiol.*, 1951, v167, 314.
6. Silvette, H., Britton, S. W., Kline, R., *ibid.*, 1938, v122, 524.
7. Muntwyler, E., Griffin, G. E., Samuelsen, G. S., Griffith, L. G., *J. Biol. Chem.*, 1950, v185, 525.
8. Muntwyler, E., Griffin, G. E., PROC. SOC. EXP. BIOL. AND MED., 1955, v89, 349.
9. Seifter, S., Dayton, S., Novic, B., Muntwyler, E., *Arch. Biochem.*, 1950, v25, 191.
10. Puckett, H. L., Wiley, F. H., *J. Biol. Chem.*, 1932, v96, 367.
11. MacKay, E. M., Bergman, H. C., *ibid.*, 1932, v96, 373.

Received April 18, 1958. P.S.E.B.M., 1958, v98.

Serum Calcium Levels in Thyroid-Ablated Rats.* (24058)

MARSHALL W. PARROTT, PATRICIA W. DURBIN, AND JUSTINE J. BURG
(Introduced by C. Willet Asling)
University of California Radiation Laboratory and Division of Medical Physics, Berkeley

Radioiodine is used extensively to destroy the thyroid gland. Complete and permanent thyroid ablation can be achieved in rats with an I^{131} dosage of 3 to 4 μC/g body weight (1). Microscopic examination of peritracheal remnants from rats injected with I^{131} at this dosage revealed only superficial alteration of the intimately associated parathyroid glands, and for this reason the parathyroids have been presumed to be functional. At very high levels of I^{131} administration, however, enough of energetic beta radiation penetrates the parathyroid glands to effect an almost complete destruction(2). In a study of comparative reproductive capacities of female rats, whose thyroid glands had been destroyed with 5 μC/g of I^{131}, and rats whose thyroid and parathyroid glands had been removed surgically(3), it was brought to our attention that parathyroid deficiency might seriously affect experimental results(4,5). McLean and Urist(6) point out that parathyroid glands are of major importance in maintenance of normal blood calcium level. In the absence of parathyroid glands, the plasma calcium may fall 30% or more below normal. The calcium level in plasma should therefore be a good measure of the functional status of parathyroid glands. These studies compare the serum calcium levels of rats whose thyroids had been destroyed by low and high dosages of I^{131} beta radiation, with those in surgically thyroparathyroidectomized rats.

Methods. The animals used were female Sprague-Dawley rats obtained from the original colony. They were housed in metal cages on wood shavings and were fed Purina Laboratory Chow and tap water *ad lib.* When the animals were 60 to 80 days old, 12 were surgically thyroparathyroidectomized, 6 were given I^{131}† intramuscularly 5 μC I^{131}/gram body weight(1,3), 16 rats were given I^{131} in-

* This work was performed under auspices of U. S. Atomic Energy Com.

† I^{131} was obtained from Oak Ridge Nat. Lab. and was administered in basic solution of Na_2SO_3.