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Glucose as a Factor in Metabolism.

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While the question of the nature and rôle of the glycuresis is still open, it has been clearly shown that glycuresis may be interpreted as a stigma imperfectness in metabolic adjustment to the quantity and quality of food intake. It is plausible that this phenomenon of the metabolic mechanism should be more noticeable in individuals with an impaired metabolism. Peculiar metabolic features of mental defectives are commonly known. We shall mention only one phenomenon because it has close bearing to the subject of the paper. While the industrial efficiency of the feeble-minded is definitely lower than that of the mentally normal, the caloric requirements of the former is hardly lower than that of the latter. The demand of the feeble minded for carbohydrates, especially breads and sweets, is striking.

Substitution of a part for bread and cereals by meat, eggs and butter (see Table I), renders them dissatisfied, unhappy and depressed, and leads to gradual and permanent emaciation (see Table II). (In addition, green vegetables with some sugar and butter in them were given *ad libitum,* approximating the caloric intake to 3000 calories.) It seemed worth while, therefore, to investigate the inter-relationship of the glycuresis and the demand for high carbohydrate intake in mental defectives.

TABLE I.

Representing a special diet (per day) with reduced carbohydrate content.

		P	F	C	
Bread	450 gm.	41	7.2	240.0	
Sugar	30 gm.	—	—	30.0	
Butter	30 gm.	—	25.5	—	
Milk	250 cc.	8.5	0.7	12.5	
2 eggs		12.0	12.0	—	
Meat	250 gm.	50.0	50.0	—	
½ Orange		0.5	—	5.0	
Total		112.0	95.4	287.5	
Calories		448.0	858.6	1150.0	2456.6

In the tables, the glycuresis is represented in mg. of sugar as estimated by Folin and Berglund's method, *i. e.,* prior to and after hydrolysis of the urine. The figures following the plus or minus signs indicate the difference after hydrolysis. The decrease noted

TABLE II.

Showing changes in body-weight on reduced carbohydrate and on customary rich carbohydrate diets. Figures in kg.

	Persons								
	1	2	3	4	5	6	7	8	9
Prior to reduced CHO diet, Dec. 25, 1925	52.7	64.8	55.9	67.5	65.7	65.0	59.8	61.8	69.5
During Jan. 12, 1926	51.8	63.9	55.4	57.5	63.2	65.2	59.5	61.8	69.1
Special, Jan. 26	50.9	62.0	53.6	55.9	61.6	64.1	58.2	61.6	69.1
Reduced, March 13, CHO diet	47.7	56.9	52.3	53.7	58.4	59.1	55.0	55.2	68.2
Total loss	5.0	7.9	3.6	13.8	7.3	5.9	4.8	6.6	1.3
After 10 days of rich CHO diet	55.0	64.3	59.3	58.0	66.1	66.3	62.7	63.6	75.0
Gain during 10 days of customary diet	7.3	7.4	7.0	4.3	7.7	7.2	7.7	8.4	6.8

TABLE III.

Illustrating the peculiarities of preformed and hydrolyzable glycuresis (in mg. of sugar per 24 hrs.) of persons with impaired metabolic mechanism (A) while on reduced CHO diet, and (B) on rich carbohydrate diet. Data for Folin's normal are reproduced for comparison.

	Persons									Day of Experiment	Normal
					(A)						
March	1	2	3	4	5	6	7	8	9		
7	504+ 93	345+157	1205+1031	468+ 44	255+ 82	295+118	558+128	267+165	245+105	4	756+ 73
8	349+276	927— 20	406+111	663+106	331+192	350+171	682+ 96	382+195	472+169	5	652+201
9	361+255	462+597	338+325	554+183	325+372	319+550	374+248	366+281	336+315	6	543+101
10	335+147	440+ 88	602+ 9	1056+ 53	331+ 90	281+262	434+ 57	417+255	296+119	7	585+ 85
11	150± 0	420+ 30	1050+193	1013+334	160+ 70		318+ 45	532+ 2	142+ 90	8	624+ 52
Average	340+154	519+158	720+334	751+144	280+161	311+275	473+115	393+179	298+160		632+102
					(B)						
April											
21	799+305	1185—219	580+536	650—176	685+111	552+278	1344+776	863+104	867+173	9	1064+910
22	628+150	1493— 14	543+334	562+187	679+622	360+180	1559—228	832+176	634+182	10	1360+771
23	1034—167	781—452	408+153	434+319	486+260	489+298	926± 0	480— 88	420+322	11	1090+200
24	583+151	771+ 3	684— 61	264+187	934— 56	756—238	764+551	828—100	842—391	12	1650+485
25	594+258	950— 90	458+362	255+165	751+460	492+252	1139—122	798+ 42	632—212	13	1170+450
Average	728+139	1036—154	555+265	433+136	707+219	530+154	1146+195	760+ 27	679+100		1267+563

TABLE IV.

Average glycuresis per day on customary high CHO diet

Ratios ——————————————————————

Average glycuresis per day on reduced CHO diet

Persons																			
1		2		3		4		5		6		7		8		9		Normal	
1.1	0.9	1.7	1	0.7	1.4	1.3	0.9	1.0	1.5	1.6	0.6	1.9	0.1	1.7	0.3	1.7	1.1	2.0	5.5

TABLE V.

Glycuresis, 5 to 12 P. M. urine with impaired metabolic mechanism on rich carbohydrate diet.

Date of experiment	Persons								
May	1	2	3	4	5	6	7	8	9
5	516— 54	518—84	340+20	470—29	376—20	365—39	620—179	631—128	668± 0
6	418+ 37	260+41	402+31	483—46	418+11	540—98	682— 96	531— 13	459—59
7	319— 23	194+18	423—86	319± 0	230+30	303— 6	501— 72	423+ 5	310+30
9	462—109	329—75	496—71	452—68	367—27	457—83	440—206	420— 93	437—20
Average	429— 37	325—25	415—26	431—36	348— 2	416—56	561—138	501— 58	468—12

occasionally is explained by the fact that hydrolysis apparently destroys a small part of the reducing substances.

It has been shown that in case of an efficient metabolic mechanism certain rather small deviations of the glycuresis from one day to the next occur, and the smaller the carbohydrate intake, the narrower these deviations become; on a rich carbohydrate diet the glycuresis increases generally, but the largest part of this increase consists of post-hydrolysis products. This is illustrated by a set of data reproduced from Folin's observation on a normal person (see Table III).

Estimations of glycuresis on our patients were made during 4½ months of observation, almost daily. Table III represents the data only for 5 consecutive days of dieting, and 5 days during customary diet. The number of mg. of sugar for any person varies from day to day quite extensively, and is quite different from the average, irrespective of the diet. On the other hand, as in Folin's normal, the average of preformed glycuresis is generally decreased when the carbohydrate intake is reduced. But the post-hydrolysis glycuresis is relatively, and even absolutely in the majority of the cases, increased in quantity with diminished carbohydrate intake. It can be seen by comparing the averages for any patient during the high and reduced carbohydrate diets. This condition is represented still more clearly by the ratios between averages, calculated on the basis of all the data available (see Table IV).

The glycuresis of normals, on a rich carbohydrate diet, consist of 5.5 times as many complex products (post-hydrolysis figures) as were present on a reduced diet, while the preformed glycuresis (ante-hydrolysis figures) was increased only twice. For defectives, however, the reverse is found; the increase was made up mostly by the preformed glycuresis, and not the complex metabolites. Moreover, persons 1, 2, 4, 6, 7, 8, showed a decreased post-hydrolysis glycuresis on a high carbohydrate diet, which is shown by ratios being below 1.

We conclude that a reduced relative quantity of carbohydrate in the diet of individuals with impaired metabolism renders their metabolic mechanism inefficient. The tendency to reduce preformed glycuresis exists, as is shown by reduced preformed glycuresis, but it lacks due steadiness and regularity. The ability of assimilation of metabolites, shown as post-hydrolysis glycuresis, is decidedly diminished, and the loss of body weight fulfills the picture of inefficiency of the metabolism on such a diet, in spite of the fact that the caloric value of it corresponds to the theoretical standards.

Hence the impaired metabolic mechanism of defectives evidently requires a larger relative amount of carbohydrates in their food. Within the organism carbohydrates circulate in the form of glucose. Therefore, our inference is that the circulating glucose is a factor in the metabolic mechanism and its effect increases with the increase of its quantity.

There is probably an increase in the quantity of circulating glucose in our patients, when after their last meal they rest comfortably in the living room. Apparently during this period glucose is not used as a source for muscular energy and is available as a factor in metabolism. Therefore, we ought to expect that in the urine collected from 5:00 P. M. there will be decreased quantity of the hydrolyzable products. Table V records the glycuresis in the urine collected from 5:00 P. M. to midnight. There is none or very little of post-hydrolysis reducing substances, which is in full conformity with the idea that glucose is a factor in metabolism.

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[3] Folin, O., *J. Biol. Chem.*, 1915, xxii, 327.

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Protective Power of Serum of Animals Fed Pneumococci or Tissue of Animals Killed by Pneumococcus.

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In an earlier paper on the immunization of rats to pneumococcus, produced by feeding the tissues of animals killed by the same organism, brief mention was made of the fact that the blood of the immune animals contains protective antibodies.[1] The present article is a report of the experiments made with sera of rats and dogs. The rats were fed either the infected tissue, the living pneumococcus, or the acid-killed germ. Such animals have already been shown to possess immunity to intraperitoneal injection of virulent pneumococci.[2, 3] The dogs were fed either the living pneumococcus, or infected rabbit tissue. The object was to learn whether the serum