Human Subject							
.....	141	0.938	0.770	0.168	13.6	0.11	0.44
8 gm. taurin, 3.76 gm. benzoic acid.....	692	2.360	0.888	1.472	14.4	0.10	3.56
.....	163	1.220	1.002	0.218	15.3	0.09	0.38

TABLE II.

THE SIMULTANEOUS ADMINISTRATION OF SODIUM BENZOATE AND TAURIN.

Dose.	Free Benzoic Acid, Gm.	Conjugated Benzoic Acid, Gm.	"Neutral Sulfur," Gm.
Dog. No. 2.			
.....	0	0.15	0.009
3.76 gm., benzoic acid.....	0.19	3.36	0.032
3.76 gm. benzoic acid, 11.25 gm. taurin .	0.77	3.43	0.013
9.04 gm. benzoic acid, 27.0 gm. taurin ..	2.50	2.44	0.010
.....	1.61	2.32	0.007
Human Subject.			
.....	0.04	0.37	0.014
1.51 gm. benzoic acid.....	0.05	1.50	0.014
1.51 gm. benzoic acid, 4.5 gm. taurin ...	0.07	1.55	0.014

202 (1949)

Blood-sugar studies.

By G. L. FOSTER.

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In a recently reported study of certain blood-sugar phenomena McLean and de Wesselow[1] have attempted to explain the nature of the blood-sugar curve obtained after the ingestion of glucose by man. As a result of the comparison of the curves of a normal and of a diabetic individual they suggest that the existence of an alimentary hyperglycemia in the normal awakens and stimulates the glycogen-forming mechanism to such activity that not only is the rising hyperglycemia checked but that the blood-sugar concentration is rapidly brought down to normal or below, thus accounting for the rapid rise and fall of blood sugar which is the characteristic response of the normal to the ingestion of glucose.

[1] McLean, H. and de Wesselow, O. L. V., *Quart. Jour. Med.*, 1921, xiv, 103.

In the diabetic, on the other hand, the glycogenic function is impaired, it does not respond to the stimulus, and the blood sugar continues to rise as long as glucose is absorbed from the intestine.

Staub [1] in Spiro's laboratory, has offered a similar suggestion, the gist of which is that the presence of abundant carbohydrate food in the body stimulates the mechanism which is concerned with carbohydrate metabolism.

In a more recent paper Folin and Berglund[2] have found the hypothesis of McLean and de Wesselow to be superfluous. To them not glycogen formation but simply absorption into the tissues is entirely adequate to account for the fact that sugar does not accumulate in the blood after the ingestion of glucose or other sugars. In arriving at this conclusion they were apparently greatly influenced by their blood-sugar findings after feeding fructose and galactose to normal individuals. The latter is known to be a poor glycogen former, hence, if glycogen-formation is an important factor in reducing or preventing alimentary hyperglycemia, then galactose should be much more effective than glucose in raising the blood-sugar level. As a matter of fact, however, they found absolutely no rise after feeding galactose, nor after fructose and maltose. They make no mention of data in the literature contrary to their findings in this respect.[1, 2, 4, 5]

The purpose of this paper is to report in a very preliminary manner some data which favor the hypothesis of McLean. We think that our results with fructose, galactose and maltose (Tables I and II) remove the ground for Folin and Berglund's objection to McLean's interpretation. In four tests with Kahlbaum's purest galactose marked increases in blood-sugar concentration were found, in two cases extraordinary hyperglycemia developed, reaching a maximum in about two hours, which corresponds in time with the maximum glycosuria found by Folin and Berglund. Our maltose curves agree with the findings of Field,[3] McLean and de Wesselow[4] and Leire.[5] With fructose both Folin and Berglund and McLean and de Wesselow found no rise in blood-sugar level.

[1] Staub, H., *Biochem. Zeits.*, 1921, cxviii, 93.

[2] Folin, O. and Berglund, H., *Jour. Biol. Chem.*, 1922, li, 213.

[3] Field, C. W., PROC. SOC. EXP. BIOL. AND MED., 1919, xvii, 29.

[4] Loc. cit., p. 408.

[5] Bang, I., "Der Blutzucker," p. 63.

TABLE I.

EFFECT OF INGESTION OF GALACTOSE.

Time.	Blood Sugar. Mg. per 100 c.c. Whole Blood.			
	Exp. 8, 100 gm. Galactose.	Exp. 9, 100 gm. Galactose.	Exp. 17, 40 gm. Galactose.	Exp. 20, 60 gm. Galactose.
Before	82	86	84	96
15 min. after	139	101	154	147
30 " "	160	102	154	163
45 " "	170	123	117	160
75 " "	235	179	93	132
105 " "	263	206	82	101
135 " "	290	247	90	97
165 " "			88	104
180 " "	186	161	90	

TABLE II.

EFFECT OF INGESTION OF MALTOSE AND OF FRUCTOSE.

Time.	Blood Sugar. Mg. per 100 c.c. Whole Blood.			
	Exp. 22, 75 gm. Maltose.	Exp. 23, 100 gm. Maltose.	Exp. 25, 75 gm. Fructose.	Exp. 26, 90 gm. Fructose.
Before	100	95	84	95
8 min. after	—	—	101	125
15 " "	144	157	108	126
25 " "	—	—	—	128
30 " "	89	165	105	111
45 " "	91	118	—	101
55 " "	—	—	92	—
70 " "	—	—	—	104
85 " "	77	91	—	—
120 " "	104	101	—	—

It is well known that fructose is much more rapidly oxidized in the body than glucose[1, 2, 3] and there is some evidence that it is a better glycogen former.[4] Taking our blood samples at short and frequent intervals we could detect a rather small but unmistakable rise of blood sugar in two fructose tests.

In studying the effects of repeated doses of glucose we have obtained results quite different from those of McLean and de

[1] Johansson, J. E., *Skand. Arch. Physiol.*, 1908, xxi, 1.

[2] Lusk, G., *Jour. Biol. Chem.*, 1915, xx, 555.

[3] Bürger, M., *Biochem. Zeits.*, 1921, cxxiv, 1.

[4] Weinland, E., *Zeits. f. Biol.*, 1899, xxxviii, 16 and 607.

Wesselow. They gave fifty grams of glucose which caused the blood sugar to rise and fall sharply. Then, when the curve had returned to normal, they gave a second dose of fifty grams which produced a response almost identical with the first. We find that the second dose may cause little or no response, depending upon the time at which it is taken (Table III). If the second dose is

TABLE III.

EFFECT OF REPEATED DOSES OF GLUCOSE.

Two doses of 100 gm. each at interval as noted by asterisk.

Time.	Blood Sugar. Mg. per 100 c.c. Whole Blood.			
	Exp. 21.	Exp. 24.	Exp. 27.	Exp. 4.
Before first dose.....	95	99	100	109
15 min. after first dose ..	171	139	166	—
30 " " " " ..	140	81	115	150[1]
45 " " " " ..	102	—	—	—
60 " " " " ..	105[1]	62[1]	84[1]	156
75 " " " " ..	101	57	85	—
90 " " " " ..	95	63	88	99
105 " " " " ..	86	57	89	—
120 " " " " ..	—	—	—	78

[1] Second dose swallowed immediately after this blood sample was taken.

taken when the rise is near its peak it produces only a slight "bump" in the curve, which we interpret as meaning that the mechanism (glycogen formation?) has nearly attained the same speed as the inflow of sugar from the gut. However, if the second dose be taken soon after the curve has returned to normal or below, it has no detectable effect on the sugar content of the venous blood (*i.e.*, the mechanism is now at top speed and can handle any quantity of sugar pouring in from the intestine). McLean worked with "finger blood" (arterial and capillary) while we used venous. We cannot yet say whether or not this is the cause of the apparent discrepancy.

Finally, the epinephrine hyperglycemias are not to be reconciled with Folin and Berglund's hypothesis. Here absorption by the tissues is normal (Palmer)[1] but nevertheless the hyperglycemia is excessive and prolonged. We are led to the conclusion that the chief factor in preventing hyperglycemia is glycogen formation, since this is presumably the only carbohydrate function which is

[1] Palmer, W. W., *Jour. Biol. Chem.*, 1917, xxx, 79.

upset by epinephrine. May we not have a mechanism analogous to secretin which controls the glycogenic function?

The subjects for our sugar experiments were healthy students (men and women). All were familiar with the technique of venapuncture, both as operators and as subjects. The blood samples were taken from an arm vein by skilled operators, and there was not the slightest ground for suspecting psychic effects on blood sugar in any of the experiments reported here.

ABSTRACTS OF THE COMMUNICATIONS.

MINNESOTA BRANCH.

FIFTH MEETING.

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203 (1950)

The diagnostic value of phosphate metabolism in experimental rickets.

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These results are based on a study of 150 rats, taken from the mothers at the age of three weeks when they weighed about 30 gm. and placed on experimental diets in small separate cages shielded from ultraviolet rays of sunlight. They were not given cod-liver oil or butter fat, but they had sufficient vitamine-A for varying degrees of growth. All of them were x-rayed at the end of this period and turned over to Dr. C. M. Jackson for morphological study. His observations have confirmed my diagnoses.

In order to diagnose rickets in rats the average P metabolism and growth per day were determined (weights being expressed in milligrams) and the following empirical formula was used. Rachitic index = *RI*.

RI = increase in body weight $\times$ 0.0022 $-$ (P retention $-$ 2).