

ARTICLE:
**STUDIES ON THE METABOLISM OF
ESKIMOS**

Peter Heinbecker
J. Biol. Chem. 1928, 80:461-475.

Access the most updated version of this article at
<http://www.jbc.org/content/80/2/461.citation>

Find articles, minireviews, Reflections and
Classics on similar topics on the [JBC Affinity
Sites](#) .

Alerts:

- [When this article is cited](#)
- [When a correction for this article is posted](#)

[Click here](#) to choose from all of JBC's e-mail
alerts

This article cites 0 references, 0 of which can be
accessed free at
[http://www.jbc.org/content/80/2/461.citation.full.ht
ml#ref-list-1](http://www.jbc.org/content/80/2/461.citation.full.html#ref-list-1)

STUDIES ON THE METABOLISM OF ESKIMOS.

By PETER HEINBECKER.*

(From the Departments of Biological Chemistry† and Physiology, Washington University School of Medicine, St. Louis.)

(Received for publication, July 9, 1928.)

It is well known that in most human subjects ketosis appears when the material metabolized becomes restricted to protein and fat. This condition is observed after several days of fasting, on diets deficient in carbohydrate, and in diabetes in consequence of the loss of ability to metabolize carbohydrate. These facts have led to the view that a certain minimum combustion of carbohydrate is necessary to avoid ketosis. The diet of the Eskimo has long been recognized as an apparent exception to this view. Being restricted almost wholly to animal tissues and containing therefore only a very small proportion of carbohydrate, such a diet might be expected to produce a ketosis. Similar diets fed to human subjects of temperate zones do so. Although the fact appears not to have been investigated, it has been assumed that Eskimos do not exhibit ketosis on their customary diets. To account for this assumed fact it has been suggested that the Eskimos must possess an adaptation to non-carbohydrate diet.

In his analysis of the ketogenic and antiketogenic factors, the balance between which seems to determine the appearance or non-appearance of ketosis, Shaffer (1) has considered the Eskimo dietary, using the data reported by Krogh (2). According to his analysis the metabolism of the foodstuffs contained in the Eskimo dietary would not be expected to cause ketosis, because the calculated antiketogenic effect of the large protein ingestion was

* National Research Council Fellow in Medicine.

† It was the interest of Dr. P. A. Shaffer in the problem of ketosis which was primarily responsible for the nature of the investigation. He contributed generously his time and knowledge of the subject in the preparation of the manuscript.

somewhat more than enough to offset the ketogenic effect of fat plus protein.

In an effort to obtain more definite information as to the carbohydrate metabolism of the Eskimos and their liability to ketosis, the writer undertook, at Dr. Shaffer's suggestion, the experiments here reported. The observations were made in July and August, 1927, upon Baffin Island Eskimos, the writer being a member of the Putnam Baffin Island Expedition.

Before presenting the results we may make a brief statement concerning the dietary habits of Eskimos. A fuller description has been written by Krogh and Krogh (2) in connection with their studies of South Greenland Eskimos at Godhavn in 1908. The statements here given apply to Greenland Polar Eskimos and to Baffin Island Eskimos, the peoples visited by the writer.

The Eskimo in his natural state eats practically only flesh. It is impossible for him to do otherwise, as plant life of a type yielding edible material does not exist. Very occasionally a bit of kelp or a few blueberries are eaten but they cannot be considered articles of diet. All animals found in the North are eaten by Eskimos but they now depend mostly on those found in the sea. Especially is this true of the Polar Eskimos, as caribou and musk-ox are practically extinct in the Thule district. The Central Eskimos can still secure both of these animals at certain seasons of the year. The seal is the animal most constantly and most universally depended upon. Not only is its flesh highly prized but the oil obtained from it furnishes heat and light. Other animals of importance are the walrus, the narwhal, the white whale, the polar bear, and the arctic hare. Dogs are eaten only in times of stress. Fish are not found to any extent where the Polar Eskimos dwell but become increasingly plentiful farther south. Everywhere water birds are found in large numbers and extensively hunted, especially eider ducks, auks, and murre. Eggs of all these birds are consumed in large numbers. Eskimos drink large amounts of water.

The preparation of Eskimo food is simple. Most meat is eaten partly boiled and the remainder raw. Frozen meat is generally eaten raw. When food of any type has been cached for some time and has become "high" or actually rotten, it is eaten raw. No salt is available.

The amount of meat eaten is very large. In times of plenty an average adult consumes 4 to 8 pounds in a day. Growing adolescents require even more. Babies are wholly breast-fed until the end of the 2nd year when meat is added to their diet. Children often nurse irregularly until the age of 4 or 6. The amount of fat eaten varies with the individual and the season of the year. In warm weather about one-seventh of the meat may be fat, in cold weather, especially when the Eskimos are traveling, one-third to one-half may be taken as fat. Krogh and Krogh have analyzed lean seal meat and found it to contain 6 to 10 per cent of fat. According to their data the average daily food partition is about 280 gm. of protein, 135 gm. of fat, and 54 gm. of carbohydrate of which the bulk is derived from the glycogen of the meat eaten.

The experiments of this report were made at Cape Dorset situated at the southwest end of Hudson Straits where a camp was established for a period of 6 weeks. The subjects utilized for the metabolism experiments were housed in a tent about 10 yards distant from the writer's, well separated from the tents of their fellow Eskimos, and were under almost constant supervision. Subjects were chosen whose physical characteristics were typical of pure blooded Eskimos. Except for short intervals they had always lived on the normal Eskimo diet. All subjects were found extremely cooperative and no difficulty was encountered in our efforts to collect desired specimens.

The main objects of the experiments were to learn whether detectable ketosis exists among Eskimos under natural dietary conditions; the extent to which ketosis develops in fasting and the rate at which it disappears on glucose ingestion; the "carbohydrate tolerance" as indicated by blood sugar curves; and to determine the respiratory metabolism during and after a ketosis-producing fast. Owing to difficulties imposed by the very limited facilities and low temperature, the respiratory data are doubtless less reliable than if obtained under laboratory conditions. But with this reservation the writer can find no reason to question the approximate correctness of the analytical results.

It may be said at once that the Eskimo on his usual dietary shows no ketosis and has high tolerance to ingested glucose. On

fasting he develops a ketosis, but only of mild degree compared to that observed with other human subjects.

Carbohydrate Tolerance as Indicated by Blood Sugar Curves.

With four subjects, three of whom were females, blood sugar was followed during $3\frac{1}{2}$ and 4 hours after administration of glucose in a postabsorptive state, about 12 hours after the previous

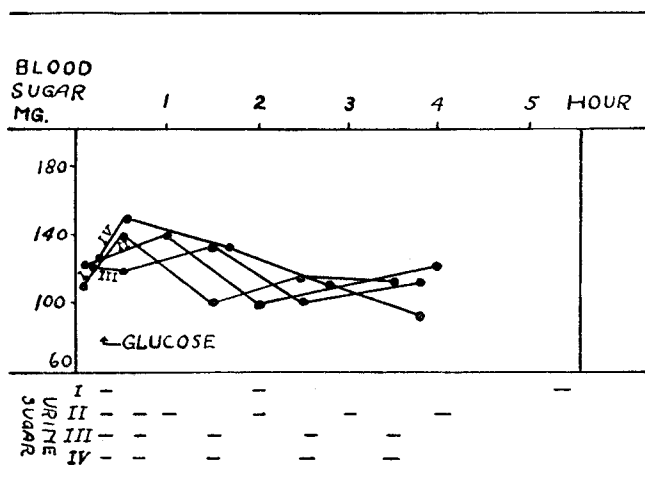


FIG. 1. Normal glucose tolerance curves. Subject I. Female, estimated age 23 yrs., weight 52 kilos; cerelose given 125 gm. Subject II. Male, estimated age 14 yrs., weight 52 kilos; cerelose given 110 gm. Subject III. Female, estimated age 13 yrs., weight 50 kilos; cerelose given 100 gm. Subject IV. Female, estimated age 12 yrs., weight 48 kilos; cerelose given 90 gm.

meal. After a preliminary blood sample was drawn, the subject drank the glucose solution, about 700 cc. containing amounts stated in legends to Figs. 1 to 3. Blood samples were drawn from an arm vein at half hour or hour intervals. In most instances urine samples were secured at the same times. The bloods were analyzed at once by the Shaffer-Hartmann method after precipitation with tungstic acid. The urines were tested qualitatively for sugar with the same copper reagent used for blood.

In order to learn the effect of fasting, the same glucose tolerance

test was performed with three subjects at the conclusion of an 82 hour fast, the glucose test dose being the first food given. The results are given in Figs. 1 to 3.

As indicated by Fig. 1, the blood sugar curves of these Eskimos, following a large dose (about 2 gm. per kilo) of glucose given 12

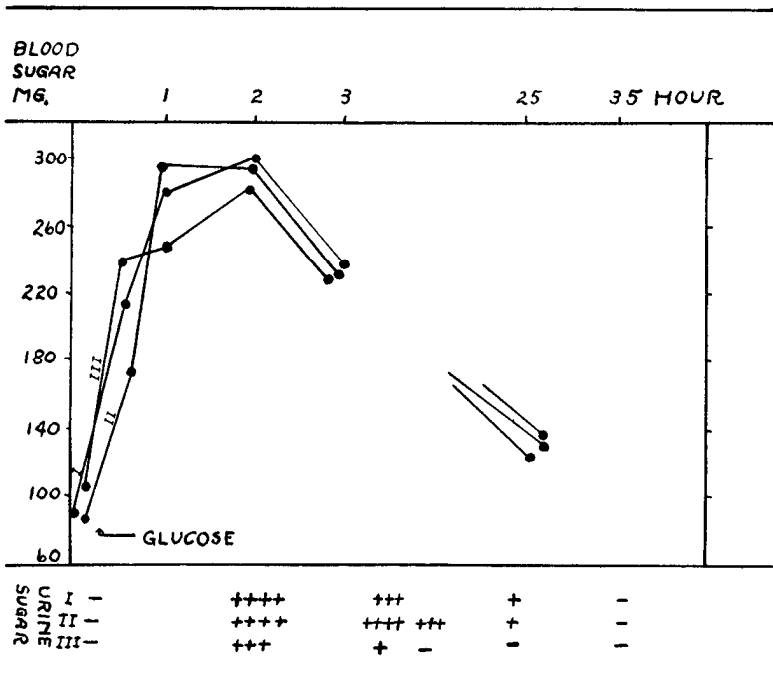


FIG. 2. Glucose tolerance curves after 82 hours of fasting. Subject I. Female, estimated age 26 yrs., weight 60 kilos; cerelose given 125 gm. Subject II. Female, estimated age 25 yrs., weight 63 kilos; cerelose given 125 gm. Subject III. Male, estimated age 28 yrs., weight 72 kilos; cerelose given 125 gm.

hours after food, show only slight rise, followed by the usual fall below the fasting value. The extent of the rise is less than commonly observed with normal subjects, and perhaps is to be interpreted as meaning exceptionally high carbohydrate tolerance. All of the urine specimens were free from sugar.

After a period of fasting, however, the behavior of the subjects

to similar doses of glucose was quite different, as shown in Fig. 2. In all three cases the blood sugar rose to maxima of 280 to 300 in 1 to 2 hours and after 3 hours was 230 to 240 mg. per cent. The first urine specimens voided 2 hours after glucose ingestion contained much sugar, and the glycosuria continued for at least 10 hours in 2 cases and for $4\frac{1}{2}$ hours in the other. 25 hours after

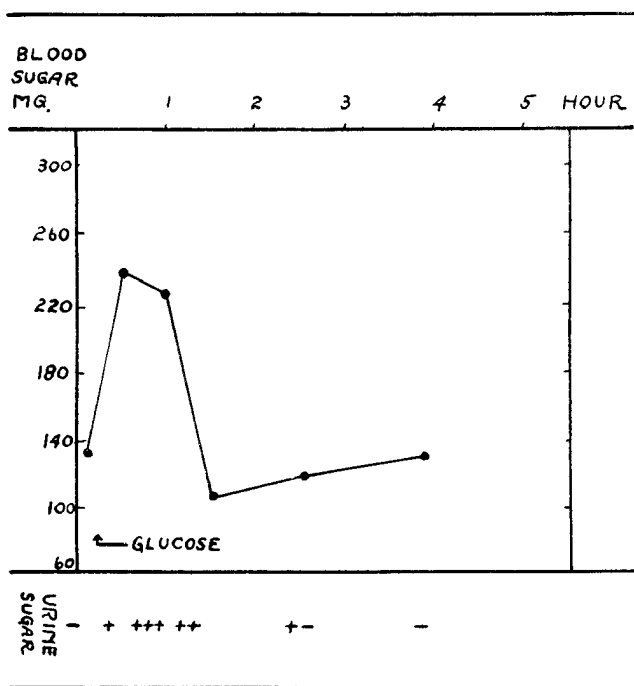


FIG. 3. Glucose tolerance curve 48 hours after Curve III, Fig. 2. Subject same as Curve III, Fig. 2.

glucose was taken the blood sugar was still somewhat higher than the fasting level.

Fig. 3 shows the blood sugar curve on Subject III after a second glucose test dose given 48 hours after the first test (Curve III, Fig. 2). During the interval he had eaten little except the 125 gm. of glucose of the first test, because his "stomach had shrunk so much during the fast that there was no room for food." In a half hour the blood sugar rose from 130 to 240 mg. and sugar appeared

in the urine. The glycosuria continued for 2.5 hours, though blood sugar dropped in 1.5 hours to below the initial value.

These results show in the first place, *that the low carbohydrate*

TABLE I.
Non-Protein Nitrogen of Eskimo Bloods.

Subject.	Date.	Description of blood filtrates.	Non-protein N, mg. per 100 cc. whole blood.
I. Aleuti.	<i>Aug.</i>		
	2	Fasting blood filtrate.	30
		Mixed blood filtrates (II, III, IV).	40
		Last blood filtrate of glucose tolerance curve.	112
	3	Fasting blood filtrate.	43
II. Martha.	2	Fasting blood filtrate.	32
		Mixed blood filtrates (II, III, IV).	33
		Last filtrate glucose tolerance curve.	30
	3	Fasting blood filtrate.	38
III. Poulussi.	2	Fasting blood filtrate (I).	31
		Mixed blood filtrates (II, III, IV).	33
		Last blood filtrate of glucose tolerance curve.	29
	3	Fasting blood filtrate.	43
	4	Mixed blood filtrates of glucose tolerance curve.	36
I. (Normal curve.)		Mixed blood filtrates of glucose tolerance curve.	30
II.		Mixed blood filtrates of glucose tolerance curve.	36
III.		Mixed blood filtrates of glucose tolerance curve.	29

content of the Eskimo diet does not result in a low carbohydrate tolerance. This is in agreement with the observation of Greenwald and coworkers (3) that high protein diets tend to prevent the fall of tolerance which accompanies fasting or low carbohydrate,

low protein diets. The sugar formed from protein is doubtless an efficient stimulant of the pancreas or other mechanisms. As a result of fasting the high natural tolerance of the subjects was temporarily much decreased, as demonstrated by the great and prolonged rise of blood sugar and simultaneous glycosuria which resulted from glucose ingestion. But in this respect also, the Eskimo is not exceptional, for similar loss of carbohydrate tolerance from fasting has been repeatedly observed with other human subjects and with animals by Greenwald (3) and Staub (4). The phenomenon is probably to be attributed to a quiescent state of the carbohydrate mechanism (perhaps the pancreas), resulting from disuse and requiring a new stimulus to arouse it to activity. The normal height of the 12 hour fasting blood sugar of the Eskimo subjects is slightly higher than usually observed, averaging about 120 mg. per cent. This value was lowered to about 100 mg. as a result of the fast.

Blood Non-Protein Nitrogen.

The excess blood filtrates from the blood sugar determinations were brought to St. Louis and analyzed for non-protein nitrogen by distillation and Nesslerization. On arrival a slight growth of mold was evident in all of the filtrates, but the error thus introduced is probably very small. The results are given in Table I. The last blood filtrate of the glucose tolerance test for Subject I yielded 112 mg. of non-protein nitrogen. This analysis was repeated with the same result and is perhaps due to faulty precipitation of the blood proteins. The other figures fall within the usual normal range and indicate no retention of nitrogenous products in the blood from the habitual high protein diets.

Ketosis during Fasting.

Three of the subjects were fasted for 3 days, during which time the urine was collected, measured, and the specimens tested qualitatively for acetoacetic acid, both with ferric chloride and nitroprusside, NH_4SO_4 , and NH_4OH (Rothera's test).

About noon and in the evening of each day acetone was tested for in the breath of each subject. The air was exhaled for 2 or 3 minutes through a glass tube dipping into cold distilled water,

after which the addition of Scott-Wilson reagent caused opalescence if acetone were present. This test is very delicate.

Samples of the urine specimens were bottled with toluene and brought to St. Louis, where they were analyzed for acetone, diacetic, and β -hydroxybutyric acid by the Shaffer-Marriott method,¹ and for total nitrogen by the Kjeldahl method. Acetoacetic acid had disappeared from the urines on arrival in St. Louis, about 3 months after the experiments were conducted. There is, however no reason to doubt the reliability of the total acetone body deter-

TABLE II.
Qualitative and Quantitative Urine Analyses.
Qualitative Test for Acetone in Breath.

Subject.	Day of fast.	24 hour urine volume.	Total N.	Total acetone bodies in urine as β -hydroxybutyric acid.	Qualitative tests for acetone bodies in:	
					Breath.	Urine.
		cc.	gm.	gm.		
I. Aleuti.	1	1400	18.90	0.105	0	0
	2	2130	14.60	0.180	+	+
	3	1520	14.74	0.300	++	++
II. Martha.	1	1380	24.60	0.097	0	0
	2	1400	13.50	0.320	++	++
	3	1890	15.40	1.230	+++	+++
III. Poulussi.	1	1420	21.20	0.125	0	0
	2	1250	13.60	0.132	+	+
	3	1460	15.55	0.160	+	+

minations. The results obtained for the total acetone bodies show much less striking increase with the progress of the fast than indicated by the qualitative tests made at the time. In view of the fact that the total amount of acetone plus acetoacetic and β -hydroxybutyric acids in specimens of well preserved diabetic urines does not change significantly in periods of a week or two, we have assumed that no loss of total acetone occurred in these urines and that the analytical results represent the amounts originally present. As a check a diabetic urine under toluene was analyzed for total acetone bodies before and after 17 days of incubation at

¹ I am indebted to Dr. P. A. Shaffer for these analyses.

38°. There was no loss of total acetone bodies. Such incubation was considered more likely to produce decomposition than several months of preservation at ordinary temperature. The results of urine analyses and of the qualitative tests made at the time are shown in Tables II and III.

The qualitative tests of urine and breath show that the Eskimos did develop distinct ketosis on the 2nd day of fasting, which increased somewhat on the 3rd day. The tests were less marked with Subject III than with the others. Subject II, who showed the highest ketosis, was a nursing mother who nursed her year-old baby throughout the fast. The formation of lactose was presumably an additional drain on the glycogen reserves. Although the results of quantitative determinations in urine also rise during the fast,

TABLE III.

Rate of Disappearance of Acetone from Breath and Acetoacetic Acid from Urine after Glucose and Resumption of Food.

Hrs. after breaking fast with 125 gm. glucose.	Subject I.		Subject II.		Subject III.		
	Urine.	Breath.	Urine.	Breath.	Urine.	Breath.	
2	+	+	+	+	±	+	(Slight.)
4.5	+	+	+	+	±	+	“
6.5	—	+	+	+	—	+	“
10.0	—	—	±	+	—	—	
25.0	—	—	—	—	±	+	(Slight.)
35.0	—	—	—	—	—	—	

the total amounts are surprisingly small, the highest on the 3rd fast day of Subject II being equivalent to only 1.2 gm. of β -hydroxy-butyric acid. The maximum excretion by the others was only 0.3 gm. and 0.160 gm.

The amounts of acetone bodies excreted by different subjects during fasting vary greatly. Some may hesitate to grant that the small amounts excreted by these Eskimos indicate a relative non-susceptibility to ketosis. It is unfortunate that the fasts were not prolonged; but the positive qualitative tests in urine and breath on the 2nd day becoming more pronounced on the 3rd day, seemed to answer the main question whether a fasting ketosis would appear, and it was only when the urines were analyzed some months later that the slowness of the degree of ketosis was

appreciated. As examples of variation in the amounts of total acetone bodies excreted by other human subjects during short fasts the data in Table IV may be cited.

As a rule most subjects show a ketosis of a gm. or more of total acetone on the 3rd fast day, but it will be noted that after an interval following one or two fasting periods, the obese subjects of Folin-Denis showed slight or no ketosis on the 3rd fast day. Marked ketosis did appear in these subjects on the 4th or 5th fast days. However, the Eskimo subjects had not fasted previous to our tests.

Why the ketosis observed with the Eskimo subjects is so mild, it is perhaps impossible to decide with the evidence available.

TABLE IV.

		Excreted on 3rd fast day. Total acetone bodies as β -hydroxybutyric acid.
		gm.
Benedict (5).	Subject L., weight 60.1 kilos.	2.1
Folin-Denis (6).	Mrs. M. Obese, weight 108 kilos.	
	First fast.	20.1
	Second "	1.4
	Third "	0.55
Folin-Denis (6).	Mrs. B. Obese, weight 178 kilos.	
	First fast.	1.95
	Second "	0.0
	Third "	0.0

We may suppose either that marked ketosis was prevented by a continued combustion of small amounts of glycogen, thus providing a ketogenic balance as postulated in Shaffer's analysis, or we may assume that little or no glycogen was available and that the Eskimo actually does possess an adaptation permitting complete combustion of fat without any intervention of carbohydrate and in this respect differs from other human subjects. Before considering these two possibilities we shall present the data on respiratory metabolism, which were obtained in the hope of deciding between these two interpretations.

Respiratory Metabolism.

Daily during the fasts, determinations of the respiratory metabolism were made. The expired air was collected in 5 minute periods in a Douglas bag, after which the volume was measured by

TABLE V.
Respiratory Exchange during Fasting.

Day of fast.	Body temperature.	Pulse rate.	Respiratory rate.	Cc. O ₂ consumed per min.	R.Q.	Calories per hr.	Calories per sq. m. body surface per hr.	Du Bois standard.
Subject I. Female, age 26 yrs., weight 60 kilos, height 153 cm.; surface area 1.57 sq.m.								
1	98.0	76	20	224	0.982	67.80	50.7	37
2	98.2	71	10	303	0.806	87.60		
3*	98.0	73	18	282	0.640	79.20		
3.5	Basal metabolism not determined.				0.600			
Subject II. Nursing female, age 25 yrs., weight 63 kilos, height 159 cm.; surface area 1.65 sq.m.								
1	98.2	70	20	314	0.813	91.20	49.6	37
2	97.2	67	16	314	0.654	87.00		
3*	97.2	72	16	291	0.608	80.40		
3.5	Basal metabolism not determined.				0.454			
Subject III. Male, age 28 yrs., weight 72 kilos, height 160 cm.; surface area 1.75 sq.m.								
1	98.1	72	24	270	0.81	78.00	51.4	39.9
2	96.3	62	17	214	0.865	62.40		
3*	98.0	57	18	320	0.714	90.00		
3.5	Basal metabolism not determined.				0.676			

* Day on which checks were made.

water displacement in a glass cylinder, and samples analyzed for O₂ and CO₂ in a Henderson-Haldane gas burette. The subjects, dressed in their usual clothing, rested on a cot for 30 minutes before and during each respiration period. The temperature of

the tent varied from 10 to 14°. Body temperature (mouth), pulse, and respiration rates were recorded. The results of the respiratory exchange are given in Table V. The data are not as regular and consistent as would be desired, and may possibly be open to question. The conditions under which the work was done were not favorable. On the other hand, care was taken to detect and avoid errors of manipulation and analysis. The bag was tested for leaks. Atmospheric air and the writer's own alveolar air were repeatedly analyzed with satisfactory results as a check on the gas analysis. Duplicate determinations were possible only on the 3rd fast day because of a lack of assistants.

The results call for comment on several points. In each of the three subjects the first determinations, made in the postabsorptive state about 10 or 12 hours after the last food, give normal R.Q., but the total metabolism is much higher than the standards for subjects in the temperate zone. It is suggestive that the resting metabolism of these subjects living near the arctic is about 33 per cent greater than the values for the temperate zone; while according to a report by de Almeida (7) the metabolism is 24 per cent less for subjects in tropical regions. The total metabolism showed no tendency to fall except in Subject II, and in Subject III on the 2nd day when the drop from 78 to 62 calories per hour may perhaps be associated with the low body temperature at the time. The absence of a consistent fall of metabolism with fasting suggests that the high values are not merely the temporary immediate effects of the customary high protein ingestion. It would appear that the metabolism is adjusted to a higher level, an inference which may be related to the apparent high vascularity of these subjects. On the 3rd day of fast with each subject and on the 2nd with Subject II (this one developed the most marked ketosis), the R.Q. was at or below the values indicative of complete cessation of carbohydrate combustion. It will be noted that values of 0.64, 0.60, and 0.71 were observed on this day. With only one series of experiments one hesitates to state too positively that these represent true metabolic quotients. They are, however, the same on all subjects. No analytical defect could be detected and experimentally they are believed by the writer to be correct. They certainly leave no ground for supposing that the subjects were oxidizing carbohydrate, and some other explanation is necessary to account for the small amount of ketosis.

The conventional interpretation of such low quotients is of course that of carbohydrate formation (and storage) from fat. The acceptability of such interpretation will depend upon whether the reader favors or rejects the existence of this transformation. No other evidence is at hand to decide this controversial question; but if the hypothetical carbohydrate formed from fat, as suggested by the low R.Q., were stored as glycogen (only a part of it could have been burned), it seems surprising that the subjects should react as they did to glucose ingestion. The marked hyperglycemia and glycosuria certainly indicate that the subjects had a temporarily decreased power to store or oxidize ingested glucose.

In relation to the problem of accounting for the slightrness of the ketosis found in the Eskimo subjects, it is therefore apparent that they, like the obese subjects referred to above, require less carbohydrate to burn their fat than normal white subjects. Children on the other hand, as shown by Wilson *et al.* (8) require a larger proportion of carbohydrate to permit the complete combustion of fat. All these data are consistent with the hypothesis that there are two mechanisms possessed by the animal organism for the combustion of fat, one of which involves the coincident combustion of carbohydrate. The varying ketogenic-antiketogenic ratio might then be a function of the relative effectiveness of these two biological mechanisms, with the further possibility that adaptation can be brought about to a low carbohydrate diet by an increase in the effectiveness of the direct mechanism for the burning of fat.

SUMMARY.

A brief account of the diet of Polar and Baffin Island Eskimos is given.

By means of glucose tolerance curves it is shown that these people have a high tolerance for carbohydrate. Following a period of fasting this tolerance is markedly decreased.

The non-protein nitrogen of the blood of Eskimos is similar to that of other races. The results indicate no retention of nitrogenous products in the blood from the habitual high protein diets.

Eskimos show a remarkable power to oxidize fats completely, as evidenced by the small amount of acetone bodies excreted in the urine in fasting.

The basal metabolism of Eskimos is considerably higher than that of persons living in temperate zones.

During fasting the respiratory quotient falls to a level which may be interpreted as indicating a conversion of fat into carbohydrate.

BIBLIOGRAPHY.

1. Shaffer, P. A., *J. Biol. Chem.*, 1921, xlvii, 449.
2. Krogh, A., and Krogh, M., *Meddelelser om Grønland*, 1914-15, li, 1.
3. Greenwald, I., Gross, J., and Samet, J., *J. Biol. Chem.*, 1924-25, lxii, 401.
4. Staub, H., *Z. klin. Med.*, 1922, xciii, 89.
5. Benedict, F. G., A study of prolonged fasting, *Carnegie Institution of Washington, Pub. No. 203*, 1915.
6. Folin, O., and Denis, W., *J. Biol. Chem.*, 1915, xxi, 183.
7. de Almeida, A. O., *J. physiol. et path. gén.*, 1920, xviii, 713.
8. Wilson, J. R., Levine, S. Z., and Rivkin, H., *Am. J. Dis. Child.*, 1926, xxxi, 335.