

Sucrose, lactose, and glucose tolerance in northern Alaskan Eskimos^{1, 2}

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ABSTRACT Sucrose tolerance tests were performed on several adult Eskimos who reported a history of intolerance to sweets. Six experienced severe diarrhea and a rise in capillary blood glucose of less than 20 mg/100 ml after a 50-g oral dose of sucrose. The Eskimo apparently exhibits a higher incidence of sucrose intolerance than does any other population tested. This condition may be a significant public health problem among Eskimos, whose consumption of sucrose has increased markedly in recent years. Fasted adults were given 10, 20, or 30 g of lactose orally on consecutive days and evaluated for clinical signs of lactose malabsorption. Children, aged 7 to 14 years, were given a 50-g oral dose of lactose and the increase in capillary blood glucose was measured. Clinical symptoms of lactose malabsorption were present in 65% of the adults and 55% of the children receiving 30 and 50 g of lactose, respectively. Blood glucose levels rose less than 20 mg/100 ml in 70% of the children. Despite the high incidence of lactose malabsorption indicated by these tests, 95% of the adults could consume the lactose in 1 cup of milk without adverse effects, and 96% of the children routinely consumed at least 1 cup of milk a day. *Am. J. Clin. Nutr.* 26: 1185-1190, 1973.

For centuries the Eskimos of northern Alaska consumed a diet consisting almost entirely of foods of animal origin. Caribou, whale, and seal constituted the dietary staples, and walrus, fish, and birds represented lesser ingredients. Consequently, the diet was high in protein and fat and extremely low in carbohydrates. Under these circumstances, most of the glucose required as a metabolic fuel was derived from gluconeogenic amino acids. Whether the surfeit of dietary amino acids was sufficient to prevent the formation of ketone bodies and their utilization as an important energy source is unknown. In any event, the fact that Eskimos habitually obtained most of their blood glucose from biosynthetic, rather than dietary sources, plus the fact that until recently diabetes was virtually nonexistent among this population, has generated an interest in their metabolic response to the current influx of dietary carbohydrates.

There is conflicting evidence concerning the ability of Eskimos to metabolize glucose. Mouratoff and co-workers (1) observed that Eskimos from the Bethel region of southern Alaska exhibited a high tolerance for a glucose load, and they associated this adaptability with the rarity of diabetes. Schaefer et al. (2), on the other hand, reported that over one-half a sample of 76 adult Canadian Eskimos exhibited

an impaired tolerance to oral glucose that was associated with a delay in insulin release. For comparative purposes, we have conducted a series of oral glucose tolerance tests on adult Eskimos of the Inupik group of northern Alaska, who share linguistic and dietary similarities with Canadian Eskimos and differ significantly in these respects from the Upik group of southern Alaska.

Beyond weaning age, the traditional northern Eskimo diet was essentially devoid of milk and milk products, and the supply of sucrose from plant sources was negligible. Both lactose- and sucrose-containing foods are readily available to the Eskimo today. In recent years, an increasing proportion of the Eskimo diet has been derived from processed foods shipped into the villages, and much of this food is high in carbohydrates. We have performed lactose and sucrose toler-

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ance tests on northern Alaskan Eskimos as a means of evaluating their ability to adapt to the presence of these sugars in their diet.

The studies described here were carried out under the auspices of the International Biological Program. Most of the work was conducted in the Eskimo villages of Wainwright and Point Hope, which are situated on the northwest coast of Alaska.

Materials and methods

Oral glucose tolerance tests were performed on 17 healthy adult Eskimos including 13 males and 4 females, ranging from 18 to 60 years of age. All tests were done in the morning to avoid problems encountered with reported diurnal variations in glucose tolerance (3). After an overnight fast, each subject received 100 g glucose in water. Venous blood samples were taken at 0, 60, and 120 min after the glucose load. Plasma glucose was determined immediately by the glucose oxidase-peroxidase method using Glucostat Reagent (Worthington Biochemical Corp., Freehold, New Jersey). Plasma samples were frozen and immunoreactive insulin was subsequently determined by Bio-Science Laboratories, Van Nuys, California.

Tests to evaluate lactose malabsorption were conducted on 11 male and 9 female subjects ranging in age from 25 to 59 years. Progressive oral doses of lactose (10, 20, and 30 g) were given in aqueous suspension after an overnight fast. The lactose was administered on consecutive days until the subject either completed the series uneventfully or experienced clinical symptoms of lactose intolerance (gastrointestinal distress, excessive flatulence, bloating, or diarrhea). The test levels were selected to approximate the amount of lactose present in 1, 2, or 3 cups of cow's milk. This test is similar to that of Desai et al. (4), which was designed to determine the minimum oral lactose dose that resulted in clinical symptoms of lactose malabsorption.

Lactose tolerance tests also were conducted on 27 healthy Eskimo children ranging in age from 7 to 14 years. After an overnight fast, each child was given 50 g lactose orally in aqueous suspension. Capillary blood samples were collected by finger prick, and blood glucose was measured by the glucose oxidase-peroxidase method. Determinations were made before and 30 and 60 min after the lactose load. Clinical symptoms of lactose malabsorption were noted, and each child was questioned concerning his normal, daily consumption of milk and milk products.

Oral sucrose tolerance tests were carried out on six adult Eskimos who reported that they experienced diarrhea or abdominal cramps after consuming sucrose-containing foods. Five were given 50 g sucrose orally in water after an overnight fast. Capillary blood samples were obtained by finger prick before and 30 and 60 min after the test dose. Blood glucose was measured by the glucose oxidase-peroxidase method after deproteinization of the sample. Another subject, who reported that he was acutely intolerant to sugar, was given a 17-g oral load and his response was evaluated symptomatically.

Neale et al. (5) reported that a sucrose-deficient, adult male excreted 2 g sucrose in a 6-hr urine collection following a 50-g sucrose load test. Measurements of total sucrose excretion during the sucrose tolerance tests on Eskimos therefore were made to see if urinary sucrose could be used as a criterion for evaluation of sucrose malabsorption. Urine was collected for 6 hr following administration of the sucrose load. Deionization was carried out by treating 2 ml urine with 3 g Amberlite IR-120 and IR-45 in a 1:1 ratio (6). Sucrose was separated from other urinary carbohydrates by thin-layer chromatography (7), eluted and quantitated by the phenol-sulfuric acid procedure of Dubois et al. (8).

Results

Glucose tolerance tests

The plasma glucose and insulin levels before and after glucose administration are illustrated in Fig. 1. Based on the glucose tolerance test standards proposed by Fitzgerald and Keen (9), none of the 17 adult Eskimos tested showed diabetic-type blood sugar curves after receiving a 100-g glucose load. Moreover, none of the subjects exhibited an excessively high or delayed plasma insulin response. Plasma insulin levels at fasting and at 30 and 60 min after the dose were well within the upper limits of the normal range used in the clinical evaluation of results of the radioimmunoassay for insulin (Bio-Science Laboratories).

Lactose malabsorption

The results of the lactose load tests done on adult Eskimos are given in Table 1. As the test dose was increased from 10 to 30 g, the number of subjects experiencing symptoms of lactose malabsorption also increased. Of the 20 adults tested, 13 (65%) incurred symptoms of malabsorption at or below the 30-g dose level. Only

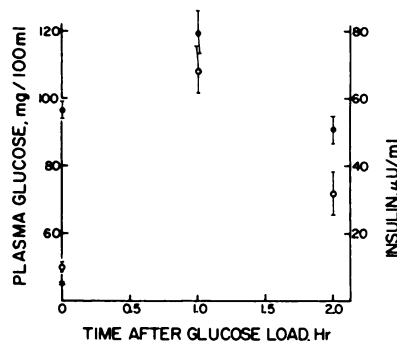


FIG. 1. Responses in blood plasma glucose (closed circles) and insulin (open circles) in 17 adult Eskimos following a 100-g oral glucose load (mean $\pm$ SE).

TABLE 1
Results of a series of lactose load tests
performed on 20 adult Eskimos

	Lactose load		
	10 g	20 g	30 g
No. of subjects first experiencing symptoms	1	8	4
No. of subjects with symptoms at this level or below	1	9	13
Percent of subjects with symptoms at this level or below	5%	45%	65%

one (5%) experienced symptoms after ingesting 10 g lactose. Only 2 of the 20 subjects reported that they neither drank milk nor used it in meal preparation, and both of these individuals showed evidence of lactose malabsorption. The remaining subjects used modest amounts of milk, mostly with other foods such as coffee or cereals, and none reported a history of milk intolerance. Hence, these tests showed that 95% of the adult Eskimos tested experienced no adverse effect after consuming an amount of lactose equivalent to that found in one cup of milk.

The results of the lactose tolerance tests performed on 27 Eskimo children, given in Table 2, indicate that 19 (70%) failed to show a blood glucose rise of 20 mg/100 ml or more within 1 hr after receiving a 50-g oral load. Fifteen (56%) reported clinical symptoms of lactose malabsorption. Characteristically, these subjects developed moderate or severe diarrhea of short duration beginning approximately 2 hr after receiving the lactose load. However, of the total sample of 27 children, 26 reported that they normally consumed one or more cups of milk a day without adverse effects. Most of the milk was consumed as a part of the school lunch program.

Sucrose tolerance tests

Figure 2 shows the blood glucose response curves for five adult Eskimos having a history of intolerance to sugar-containing foods who were given a 50-g oral sucrose load (subjects 1 to 5). All five had flat glucose curves and experienced diarrhea within 2 hr after sucrose administration. The remaining individual (subject 6) was the father of two of the subjects shown to be sucrose intolerant. He regularly consumed processed foods containing sucrose,

TABLE 2
Results of 50-g lactose load tests performed on
27 Eskimo children

Age, years	No. tested	No. with < 20 mg/100 ml increase in blood glucose	No. with clinical symptoms of intolerance	Milk consumption ^a
7	2	0	0	2
8	4	2	3	4
9	5	3	2	5
10	4	4	2	3
11	3	2	3	3
12	3	2	2	3
13	4	4	2	4
14	2	2	1	2
Total	27	19	15	26
Percent		70.4	55.6	96.7

^a Number of children who reported that they normally consumed one or more cups of milk per day without adverse effects.

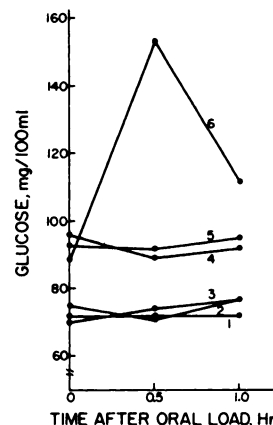


FIG. 2. Blood glucose response of five sucrose-intolerant and one tolerant subject given a 50-g oral sucrose load.

exhibited a normal blood glucose response, and experienced no diarrhea or gastrointestinal distress following the sucrose load. None of the sucrose-intolerant subjects reported that they were intolerant to milk.

An additional adult male (subject 7) who was given a 17-g sucrose load (the amount of sugar in a common brand of carbonated beverage to which he was intolerant) subsequently developed acute diarrhea. After a glucose tolerance test, he showed a normal blood glucose and insulin response. These findings indicate that his intolerance to sucrose was not due to

impaired monosaccharide absorption. This conclusion is supported by the fact that he consumed large quantities of raisins as a source of sweetness in the diet.

For the intolerant subjects, urinary sucrose excretion during the 6-hr period following administration of sucrose ranged from 2.8 to 191.4 mg. For subject 6, who had no symptoms of sucrose malabsorption, sucrose excretion was 28.5 mg. These amounts all fall within the reported range of sucrose concentrations in normal urine. Welsh and his co-workers have reported (10, 11) that the degree of lactosuria that occurs during a lactose tolerance test in adults is of little value in differentiating between subjects with isolated lactase deficiency and those with normal lactase activity. This would also seem to be the case with sucrosuria during a sucrose tolerance test.

A hereditary pattern of sucrose malabsorption is suggested by the familial occurrence of intolerance among the subjects tested. Of the six individuals tested and found to be intolerant, two were brothers (subjects 1 and 2) and two were brother-sister siblings (subjects 3 and 7). The 3-year-old daughter of subject 7 was reported by her parents to experience diarrhea after eating sweets. The mother was observed to be tolerant to sucrose-containing foods and beverages. Another of the intolerant subjects (subject 6) reported that his sister, who could not be tested at the time, also developed diarrhea after consuming sucrose-containing foods such as soft drinks, cake, and ice cream.

Discussion

Glucose tolerance

No evidence was obtained for any abnormality in glucose metabolism among the northern Alaskan Eskimos examined in this study. Their response to oral glucose appears to resemble that of the southern Alaskan Eskimos investigated by Mouratoff and co-workers (1). The findings of Schaefer et al. (2) on Canadian Eskimos, in whom a 54% incidence of abnormalities was reported, contrast sharply with those on both groups of Alaskan Eskimos. The blood glucose concentration observed at 1 and 2 hr after an oral glucose load was similar in the two groups of Alaskan subjects, but in each case was substantially lower than that recorded at analogous times in over one-half the Ca-

nadian cohort. The highest 1-hr plasma glucose level observed in the present study was 166 mg/100 ml, a value less than the average for the normals in the Canadian sample. In addition, the insulin titer appeared to decline more rapidly. The mean insulin level at 2 hr was only 47% of that at 1 hr in the northern Alaskan Eskimos, compared with approximately 92% in their Canadian counterparts. There appears to be a real difference therefore in the ability of Eskimos from the two regions to metabolize glucose, which may be related to the mechanism of insulin release (2).

Lactose tolerance

Failure of blood glucose to rise more than 20 mg/100 ml after a 50- or 100-g oral lactose load has been used extensively as a criterion of lactose intolerance in various population groups (10-15). Welsh et al. (10) had demonstrated a good correlation between failure of capillary blood glucose to rise more than 21 mg/100 ml following an oral lactose load and reduced intestinal lactase activity (lactase activity of less than 1 unit/g wet weight intestinal mucosa). By this criterion, 70% of the Eskimo children tested in this study were lactose intolerant. This value may be slightly high. Measurement of the blood glucose response at an additional time interval of 15 or 20 min after lactose administration might have increased the number of children showing a rise in blood glucose of 20 mg/100 ml or more (16).

A second useful criterion has been the development of clinical symptoms of lactose malabsorption such as gastrointestinal distress, flatulence, and diarrhea following a 50-g oral dose of lactose. Of the 27 children tested, 15 or 56% experienced one or more of these symptoms. Based on these two criteria, it can be estimated that between 56 and 70% of northern Alaskan Eskimo children over the age of 6 are intolerant to a 50-g oral dose of lactose. An incidence of 54 to 88% intolerance has been reported for Greenland Eskimos of various ages (17).

Despite these results, the children in this study reported that they habitually consumed one or more cups of milk per day without adverse effects. The supervisor of the school lunch program reported that each pupil drank at least one cup of milk daily, apparently without subsequent difficulties. Huang and

Bayless (18) have reported that nine lactose-intolerant schoolchildren (seven Negro and two white) drank at least one glass of milk a day without untoward effects. Leichter (12) reported that healthy adults of Slavic origin consumed at least one cup of milk per day without difficulty, even though they demonstrated a 23.9% rate of lactose intolerance based on standard lactose tolerance tests.

Our study indicated that although 65% of the adult Eskimos were intolerant to 30 g lactose and 45% to 20 g, only 5% were intolerant to a 10-g load. Of the 13 adult subjects showing evidence of lactose intolerance, 70% were intolerant to the 20-g dose. This indicates that the lactose threshold for most of the intolerant subjects is between 10 and 20 g when lactose is given alone.

These observations illustrate the practical limitations of the 50-g lactose load test as an indicator of tolerance for milk or milk products. The prime reason for using this quantity of lactose (equivalent to that present in 1 liter of milk) presumably is that any lesser amount leads to a rise in blood glucose that is too small to monitor reliably. When the lactose tolerance test is used to test for tolerance to dairy foods, the use of this unrealistically large dose of lactose leads to a spuriously high estimate of intolerance for such foods. An ability to tolerate 1 or 2 cups of milk at one time (10 or 20 g of lactose) is usually sufficient. Moreover, lactose in milk evidently is tolerated better than lactose alone (19, 20). A recent recommendation of the Protein Advisory Group of the United Nations (21) calls for continued use of milk in feeding programs in developing areas, even though these populations may have a high incidence of lactose malabsorption when evaluated by lactose tolerance tests.

Within recent years many researchers have reported a high prevalence of lactose intolerance in adults of various ethnic groups: American Negroes, 70% (16); Orientals, 87 to 100% (22, 23); Asian Indians, 55% (4); American Indians, 67% (10); and Mexican-Americans, 54% (24). Duncan and Scott (25) recently reported that 80% of a mixed sample of 36 adult Alaskan Eskimos and Indians were intolerant to a 50-g lactose load. Alaskan and Greenland Eskimos therefore can be classified with the preceding groups with respect to lactose tolerance. Caucasians of northern

European extraction appear to be one of the few populations with a low incidence of lactose intolerance (0 to 20%) (10, 15).

Sucrose malabsorption

Sucrase deficiency has been recognized as a clinical entity only in recent years and to date some 100 cases have been documented (17, 26). Unlike lactase deficiency, which occurs according to a geographic and ethnic pattern, sucrase deficiency generally occurs much less frequently and in a sporadic distribution. Eskimos represent the only ethnic group with a known susceptibility to sucrose malabsorption. In addition to the evidence for sucrose intolerance in Alaskan Eskimos presented here, McNair and co-workers (17) recently reported a 10.5% incidence in a selected sample of Greenland Eskimos. Although too few northern Alaskan Eskimos were tested in our study to determine the prevalence of sucrose intolerance in this population, it is apparent that there is a higher incidence than has been found in any non-Eskimo population.

Because sucrose presumably was absent from the traditional Eskimo diet for many centuries, sucrase activity was a negligible factor in natural selection. The recent introduction of sucrose and sucrose-containing foods into the Eskimo diet may present a significant public health problem. Sucrose malabsorption should be considered as a possible cause of the persistent diarrhea and gastroenteritis that occurs with considerable frequency among Eskimos, especially in infants and children in the postweaning period. Although sucrose intolerance occurs with less frequency than lactose intolerance, its consequences for the individuals affected are more serious. Sucrose intolerance imposes multiple restrictions on food selection from the modern dietary, whereas lactose intolerance usually can be managed by moderating the intake of a small number of foods. Typically, the sucrose-intolerant subjects complained of diarrhea following consumption of a piece of cake or a can of carbonated beverage. There was a tendency to consume limited portions of such foods and to risk the consequences. The prevalence of sucrose intolerance in Eskimos deserves further investigation. Intolerant subjects in this study were initially identified by dietary questioning, and numerous individuals were not tested

because they habitually consumed sucrose-containing foods. Judging from a dietary survey conducted in Wainwright, the prevalence of sucrose intolerance would appear to be less than that reported in Greenland Eskimos (17); however, additional intolerant individuals undoubtedly could be identified by systematic sucrose tolerance testing. All the affected subjects reported that their intolerance was long-standing.■

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