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The Incidence of Lactose Malabsorption Among Cache Valley Young Adults

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THE INCIDENCE OF LACTOSE MALABSORPTION AMONG CACHE VALLEY YOUNG ADULTS

by

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Eileen Donna Cowles

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ABSTRACT

The Incidence of Lactose Malabsorption
among Cache Valley Young Adults

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Twenty-four young adults, ages 18 to 30, from Cache Valley were studied to determine the incidence of lactose malabsorption. Lactose malabsorption was determined by a two-hour oral lactose tolerance test. The criteria for a malabsorber was a flat glucose curve in which there was a rise in serum glucose less than 20 mg. per 100 ml. above the fasting level. Four of the subjects were found to be lactose malabsorbers. The incidence of lactose malabsorption was determined to be 16.67 per cent. A 95 per cent confidence interval for this study indicated that the actual percentage could be expected to be between 1.76 and 31.58 per cent with 75 per cent confidence. Two of

the subjects determined to be lactose malabsorbers developed gastrointestinal symptoms associated with lactose intolerance: abdominal cramping, flatulence and/or diarrhea. The daily milk intake varied greatly among both lactose absorbers and malabsorbers subjects.

(68 pages)

Chapter I: Introduction

Lactose intolerance is seldom considered in the treatment of northern European descendants complaining of gastrointestinal symptoms of abdominal cramping, flatulence, and diarrhea. The incidence of lactose intolerance among adult white Americans has been found to range from 5 to 10 per cent (1). Studies reviewed by Simoons (2) showed lactose malabsorption ranging from 6 to 25 per cent for white Americans. The overall prevalence of lactose malabsorption of Europeans and their overseas descendants was found to be 11 per cent (2).

Lactose intolerance is the result of a deficiency of enzyme activity preventing lactose from being hydrolyzed into glucose and galactose. The lack of enzyme, lactase, results in the inability of lactose to be split and absorbed in the intestinal lumen. Lactose remains in the intestine increasing the osmolality of the intestine. This results in fluid retention in the intestine and increased intestinal motility resulting in abdominal cramping and diarrhea.

Lactose intolerance has been observed in adults and children among much of the world population including Asians, Africans, Australian Aborigines, Israeli Jews,

South American Indians, and southern Europeans (3, 4). Populations with a low incidence of lactose intolerance are primarily Scandanavians and descendants of northern Europeans (5).

Populations with a high incidence of lactose intolerance use milk primarily during infancy. Lactose intolerance develops during later childhood and adult life. Populations that do not exhibit lactose intolerance are those that maintain a high ingestion of milk throughout life (5).

Cattle raising has also been correlated with lactose intolerance. Generally lactose intolerant populations traditionally do not raise cattle. Milk after weaning was, therefore, not readily available. Conversely, milk tolerant populations raise cattle allowing an availability of milk for consumption throughout life (5).

Two hypotheses are considered for the differences in lactose intolerance among differing populations. The genetic hypothesis suggests that the continued milk consumption through late childhood and adulthood has resulted in a continued lactase activity. Continued lactase activity has been considered to be a deviation from the normal disappearance of lactase activity following weaning (5).

The second hypothesis is termed the adaptive hypothesis. Changes in diet such as an increased lactose consumption result in increased dissacharidases including sucrase and isomaltase as well as lactase (5).

Secondary lactase deficiency has been induced by gastroenteritis and protein-calorie malnutrition. The age of onset of lactose intolerance may be correlated to gastroenteritis and malnutrition in populations where these disorders are frequent. Onset of all lactose intolerance can not be related to malnutrition and/or gastrointestinal diseases (5).

Cache Valley is known for its dairying and cheese production. Approximately 200,000,000 pounds of milk are produced yearly in Cache Valley (6). Approximately 75,000 pounds of cheese are produced daily (6). Cache Valley was originally settled by members of the Church of Jesus Christ of Latter-day Saints as directed by Brigham Young in 1856 in an effort to find more suitable lands for the increasing number of settlers arriving in the Salt Lake Valley (7). Many Cache Valley settlers were immigrants of northern Europe or their descendants. Immigrants came from the British Isles, Denmark, Switzerland, Norway, Sweden, and Italy (7).

The incidence of lactose malabsorption among the descendants of these settlers would be expected to range from 5 to 25 per cent. If the incidence of lactose malabsorption is 5 to 25 per cent, consideration should be given to general and therapeutic dietary counseling among white Americans. Lactose malabsorption should be considered among individuals seeking medical attention with complaints of gastrointestinal disorders such as abdominal cramping, flatulence, and/or diarrhea.

The purpose of this study was to determine the incidence of lactose malabsorption among young adult residents of Cache Valley. Current consumption of dairy products of subjects was obtained by questionnaire. Ancestry of subjects was obtained as possible in order to better define the population studied.

The incidence of lactose malabsorption was determined by an oral two-hour lactose tolerance test. The subjects studied consisted of twenty-four young adults ranging from ages 18 to 30. Subjects stated that they were residents of Cache Valley. Lactose tolerance tests were administered to subjects who appeared to be healthy with no current complaints of gastrointestinal disorders.

Chapter II. Review of literature

A review of current literature on lactose intolerance indicated concerns in six areas: (1) definition of lactase deficiency, (2) methodologies for the determination of lactase deficiency, (3) identification of lactose intolerant populations, (4) etiology of lactase deficiency, (5) milk consumption and lactose intolerance, and (6) practical concerns of lactose intolerance.

DEFINITION OF LACTASE DEFICIENCY. Hypolactasia or primary lactase deficiency has been defined as low levels of lactase found in the intestinal mucosa of the older child or adult (5). Lactase is necessary for the hydrolysis of the dissacharide lactose found in fluid milk and other milk products. Lactose is hydrolyzed into glucose and galactose by lactase prior to absorption through the intestinal mucosa.

With lactase deficiency, lactose can not be absorbed and remains in the intestine. This results in increasing osmolality causing fluids to diffuse into the intestine by osmosis leading to an increased gastrointestinal motility. Intestinal bacteria ferment the unhydrolyzed lactose. The increased motility and fermentation result

in the gastrointestinal symptoms of abdominal cramping, flatulence, and/or diarrhea.

Secondary lactase deficiency is a temporary decrease in lactase activity in the intestinal mucosa. This is a result of another condition such as protein-calorie malnutrition or gastroenteritis (5,8). The age of onset of lactose intolerance may be correlated to such disorders in populations where there is a high frequency of these disorders. However, the onset of all lactose intolerance can not be related to malnutrition and gastrointestinal diseases (5).

A third type of lactase deficiency occurs rarely in infants. It is termed congenital lactase deficiency. The absence of lactase in such infants is apparent within the first few weeks of life (5,8).

In the past, no distinction was made between lactose intolerance and lactose malabsorption. Current literature has made a distinction between these two terms. Lactose intolerance refers to the presence of gastrointestinal symptoms such as abdominal cramping, diarrhea, and flatulence. These symptoms occur after the ingestion of lactose or milk products containing lactose (5). Lactose malabsorption has been defined as a reduced

absorption of lactose as a result of lactase deficiency, usually determined by a lactose tolerance test (5).

Other dissacharidases are not affected in lactase deficiency. A decrease in lactase activity can occur at different ages: between three to five years of age, between ten to sixteen years of age, and during the late teens and early twenties (1,8).

Welsh et al. (9) investigated the relation of age and race to decline in lactase activity. The study consisted of 399 subjects ranging from ages one month to ninety-three years. Three different races were studied: 339 whites, fifty-three blacks, and seven American Indians. It was concluded that whites maintained high levels of lactase through the first five years of age. Low levels of lactase did not appear in blacks under three years of age.

Barr et al. (10) studied the prevalence of lactose intolerance among children with recurrent abdominal pain. The study showed

> . . . that lactose intolerance was common in children of various ethnic backgrounds who present with recurrent abdominal pain. Twenty-seven per cent of a heterogeneous group of white children were affected (10).

Barr et al. further stated that

> . . . children without abdominal pain were not tested, we did observe evidence of lactose malabsorption more frequently in normal schoolchildren with a history of abdominal complaints than in those without such a history . . . both adults and children may lack awareness of intolerance to lactose, since most can tolerate some milk in the diet, symptoms are non-specific and may vary in frequency, and discomfort usually occurs more than one hour after ingestion of lactose. Thus, it would be surprising if children with recurrent abdominal pain and malabsorption of lactose were found to drink more milk than their nonaffected counterparts, they might be expected to continue to drink milk despite the pain (10).

Due to the prevalence of lactose malabsorption and its clinical indistinctness, Barr et al. (10) recommended that objective investigation should be done in children with recurrent abdominal pain "before other invasive diagnostic procedures and before assuming a psychogenic basis." It was also recommended that since lactase deficiency

> may be secondary or occur independently of an underlying disease, the patient's clinical course must be followed and, if indicated, further diagnostic tests performed (10).

Bayless et al. (1) reviewed a study that indicated that healthy adults who were able to consume milk without symptoms of intolerance as infants and children observed an onset of symptoms during their late teens and early twenties. Subjects who developed symptoms of intolerance were found to have a normal intestinal mucosa. Other

dissacharidase levels were determined to be normal. There was no history of gastrointestinal disease which could result in a secondary enzyme deficiency.

Adult lactase levels do not increase with increased lactose or milk consumption. Neither do lactase levels decrease with the restriction of lactose ingestion as in the treatment of galactosemia (1).

METHODOLOGIES FOR THE DETERMINATION OF LACTASE DEFICIENCY. Five different methodologies have been developed for determining lactase deficiency. The definitive diagnosis requires a biopsy specimen of the jejunal mucosa. Indirect methods have been developed due to the difficulty of obtaining biopsy specimens. Indirect methods include the oral lactose tolerance test and breath tests.

An intestinal biopsy has been used to estimate intestinal lactase activity. The procedure requires a specimen obtained from a site just distal of the dudenojejunal junction. Other sites may result in difficulties in the interpretation of lactase activity unless a sucrase-lactase ratio is obtained (11). A quantitative analysis of lactase activity is determined. The biopsy specimen should be examined for morphology. The morphology of the intestinal mucosa is normal in a primary

lactase deficient subject and abnormal in a subject experiencing a secondary lactase deficiency. These biopsies have been found to have possible hazards when performed on small children (11).

The oral lactose tolerance test has been the most frequently used method for determining lactose intolerance. Blood samples are obtained to determine changes in serum glucose as a result of lactose ingestion and digestion. Blood tests have been modified by sampling capillary blood rather than venous blood. Another modification is the administration of ethanol prior to the ingestion of lactose (12).

The purpose of the oral lactose tolerance test is to determine changes in serum glucose as a result of the hydrolysis of lactose into glucose and galactose. Blood samples are obtained at fasting and at intervals up to three hours following the ingestion of lactose. The samples are analyzed for serum glucose. A flat curve indicates a malabsorption of lactose (Figure 1).

The amount of lactose used for the lactose load varies. Studies suggested from 1 to 2 gm. lactose per kilogram body weight (5,8,9,12). Some studies however used 50 gm. lactose as an upper limit (5,11,12) while others recommended 100 gm. (12,14).

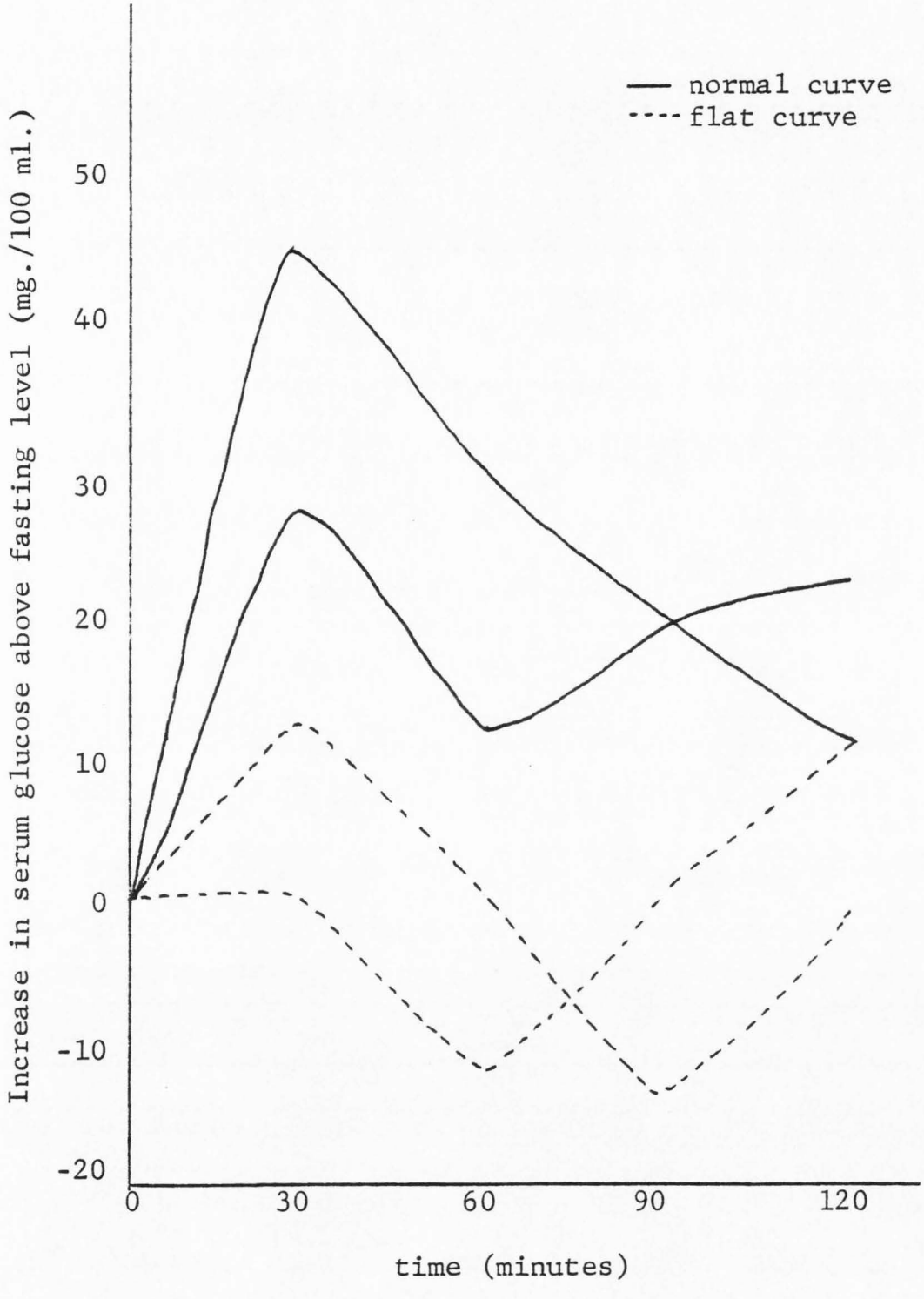

Fig. 1. Normal and flat glucose curves following a lactose load

The definition of a flat curve also varied among studies. Some researchers have used an increase in plasma glucose less than 20 mg. per 100 ml. above the fasting level (5,9,12,13). Other researchers allowed the increase to be less than 25 mg. per 100 ml. (1,8,14). A false positive test could occur in a lactose tolerance test for an individual with metabolic disorders such as functional hypoglycemia. In such an individual, an abnormally flat curve would result as the glucose is quickly absorbed from the blood.

Newcomer et al. (12) recommended the collection of capillary blood rather than venous blood. This was to correct for the utilization of absorbed glucose by peripheral tissues.

Another modification of the lactose tolerance test is to administer ethanol at the time of lactose ingestion in order to inhibit hepatic metabolism of absorbed galactose. The ethanol modification of capillary plasma test requires the subject to drink 300 mg. ethanol per kilogram of body weight over a five to ten minute period. Fifteen minutes after the ethanol ingestion, a capillary blood sample is drawn for galactose determination. A solution of 50 gm. lactose in 500 ml. water is ingested.

Blood samples are obtained at 15, 30, 45, 60 and 120 minutes after the lactose consumption. Galactose is determined for each sample by a galactose oxidase method. An increase of less than 5 mg. per deciliter over the fasting state is considered lactase deficient (12).

Breath tests have more recently been developed for the determination of lactase deficiency. These tests measure the changes in breath concentration of either hydrogen or $^{14}CO_2$ (12).

The hydrogen breath test is conducted by determining breath hydrogen excretion at zero, one, two, three, and four hours after the oral lactose load. The breath hydrogen excretion is obtained by a rebreathing technique. A five-minute sample of expired breath is obtained with the subject breathing through a mouthpiece into a closed loop containing a flutter valve, a carbon dioxide absorbent, 30-liter neoprene bag, and a second flutter valve. The subject's nose is clamped. Ten liters of oxygen and 1.0 ml. helium is added to the bag before collection begins. The helium serves as an internal standard. Samples are withdrawn by a hypodermic needle into a glass vacutainer. Samples are analyzed for hydrogen by gas chromatography (12).

The $^{14}CO_2$ breath test utilizes labeled ^{14}C-lactose. It is given orally with 50 gm. lactose. Breath $^{14}CO_2$ samples are taken at zero, one-half, one, two, three, and four hours after lactose ingestion. Subjects are kept sedentary during the test. Breath is exhaled into a glass tube into a trapping solution containing 2 mEq. Hyamine hydroxide with thymolphthalein as an indicator. Liquid scintillation spectroscopy is used to determine the radioactivity of the trapped $^{14}CO_2$. Excretion of $^{14}CO_2$ is expressed as a rate (12).

Newcomer et al. (12) conducted a study to compare the indirect methods of determining lactase deficiency. The study was conducted on twenty-five subjects known to be lactase deficient (determined by a biopsy of the intestinal mucosa) and twenty-five subjects with normal lactase activity. Newcomer et al. stated that

> although all four of the indirect tests for detecting lactase deficiency possessed considerable sensitivity and specificity . . . , the standard plasma glucose test was clearly inferior to the plasma galactose . . . test, as well as to the two breath tests.

The accuracy of the four indirect tests studied by Newcomer et al. are shown in Table 1.

Table 1. Accuracy of indirect lactase deficiency tests

group	no. of subjects	results of tests	plasma glucose	plasma galactose	breath $^{14}CO_2$	breath hydrogen
			number of tests			
lactase deficient	25	false negative	6	1	2	0
lactase normal	25	false positive	1	0	0	0

Limitations exist for all the indirect tests. The ethanol modification requires the ingestion of alcohol which would not be acceptable in children. It may also be poorly tolerated by other subjects. The $^{14}CO_2$ breath test has three disadvantages: (1) the conversion of [^{14}C] lactose to $^{14}CO_2$ by bacterial enzymes, rapidly generating $^{14}CO_2$ which would be absorbed and expired in the breath, (2) the long physical half-life of ^{14}C, and (3) the association of metabolic disease states such as diabetes with a decreased rate of conversion of absorbed ^{14}C glucose to $^{14}CO_2$. The hydrogen breath test requires the presence of bacterial enzymes for the production of hydrogen from non-absorbed lactose or its digestion products. If the subject were to have a sterile bowel, the test would be invalid. This test also requires a bulky breath sampling system and the more complex analysis by gas chromatography (12).

Barr et al. (10) suggested

> that physicians cannot rely on differences in milk ingestion, pain frequency or presence of diarrhea to identify lactose malabsorbers, even when symptoms are recorded daily. Although symptom scores obtained after lactose ingestion have been recommended for use in epidemiological studies of lactose intolerance, they do not help to identify affected persons who present with abdominal pain as the chief

> complaint. In particular, abdominal pain was least helpful in distinguishing lactose malabsorbers from unaffected persons. This difficulty may relate to unreliable reporting by patients or parental concern about the cause of the symptom (or both) (10).

Different studies have shown variability in methods used for lactase deficiency determinations. The oral lactose tolerance test itself has a great amount of variability as to its administration. However, it appears that the oral lactose tolerance test can more easily be administered to a variety of subjects.

IDENTIFICATION OF LACTOSE INTOLERANT POPULATIONS. Lactose intolerance has been recognized for seventy years as gastrointestinal disturbances following the ingestion of lactose or lactose containing milk products (5). The focus of study has been on populations determined to have a high incidence (60 to 100 per cent) of intolerance. These populations include peoples from Asia, Africa, southern Europe, the Near East and their descendants such as the American black (1,2,5).

Populations considered to have a low incidence (0 to 30 per cent) of lactose intolerance are northern Europeans and their overseas descendants, a few African ethnic groups, Bedouin and other Saudi Arabs, and some groups

in northwest India and Pakistan. The low incidence populations make up a relatively small part of the world population (1,2,5).

With the increasing interest in lactose intolerance, there have been a greater number of studies conducted. The purpose of many of these studies have been to determine the incidence of lactose intolerance in different populations. Most populations studied have been those that would be expected to be lactase deficient.

ETIOLOGY OF LACTASE DEFICIENCY. Lactase deficiency among the total world population seems to be the rule rather than the exception. Because of this, the etiology of lactase deficiency has been investigated. Two hypotheses have developed for explaining lactase deficiency: genetic and adaptive.

The genetic hypothesis was based on the concept that adults with a persistent high lactase level resulted from selective mutation under certain ecological conditions which promoted survival under pastoral, milk-producing populations. This mutation resulting in a continued high lactase activity in late childhood and adult years would be a deviation from the usual disappearance of lactase

following weaning (5).

The genetic hypothesis was supported by Bayless et al. (1). Four factors have been recognized which have been supportive of the genetic hypothesis: (1) marked racial differences have been found reproduceable regardless of present environment in different locations, (2) tribal differences as found among Nigerians, (3) family history of deficiency in some white patients who have lactase deficiency, and (4) regular patterns of decreased enzyme activity with increasing age of Africans and their overseas descendants. A possible relationship has also been considered between the available milk consumption and the maintenance of a genetic trait for lactase deficiency (1).

Simoons (2) has developed a geographic extension of the genetic hypothesis which he has termed the geographic hypothesis. Simoons considered the geographic distribution of high and low incidence of lactase deficiency throughout the world. He explained the distributional patterns as affected by evolution and history.

The genetic hypothesis is supported by studies that conclude that lower incidences of lactose intolerance occurred as the percentage of European ancestry increased.

When native groups who were malabsorbers interbred with northwest European descendants who were mostly absorbers, a lower incidence of malabsorption was found than in the original native group (2).

The geographic hypothesis considers three points: (1) the belief that present day group differences in lactose malabsorption were due to genetics, (2) adult lactase deficiency was the normal state of most of mankind, and (3) group differences of lactose malabsorption prevalence relate to patterns of milk consumption over a long historical period (2). Simoons (2) explained that the low prevalence of lactose malabsorption among some groups was the result of selective pressures over a long historical period that favored lactose absorbers under certain ecological conditions.

Simoons (2) cited that with the origins of dairying, an individual who maintained a high level of lactase throughout life would experience a selective advantage in a pastoral setting. As lactose-containing dairy products were consumed under otherwise marginal nutritional circumstances without adverse symptoms, more of these foods would be consumed. This resulted in a healthier, taller, and stronger individual who was better able to care for

the family. Lactose absorption eventually became characteristic of that particular population. "Human groups not subject to such selective pressures would continue to have high prevalences of lactose malabsorption (2)."

Groups exhibiting high lactase activity as adults "are thought to be inheritors of a dominant mutation of a regulatory gene controlling lactase synthesis (13)." This hypothesis would suggest the existence of a combined effect of food consumption and evolution to nutrition and health.

The adaptive hypothesis currently has received little support as lactose ingestion has been found not to prevent decreased lactase activity. Also, children on a lactose-free diet for the treatment of galactosemia do not have a loss of lactase activity (1,5).

Current opinion appears to support the genetic or geographic hypothesis on the evolution of high levels of lactase activity through the life cycle in a small portion of the world population. This hypothesis suggests a relationship of cultural and historical influences on the evolution of biological factors for nutrition and health.

MILK CONSUMPTION AND LACTOSE INTOLERANCE. Questions have been raised concerning how much milk can be consumed by a lactose malabsorber without developing symptoms of intolerance. This concern has been considered in reviewing such nutrition programs as the school lunch program and the use of nonfat dry milk sent to developing countries by governmental, religious, and private agencies. Many of the populations that have a high incidence of lactase deficiency use small amounts of milk such as in tea and coffee. Studies have been conducted to try to determine the amount of milk that can be consumed by lactose malabsorbers without developing gastrointestinal symptoms.

Garza et al. (15) studied ninety-nine children between the ages of four to nine years, free from any disease that might result in a secondary lactase deficiency. The children were fed different levels of lactose to determine tolerance level. It was found that none of the white children developed lactose intolerance symptoms. Intolerance among black children increased with an increased lactose intake. None of the children had difficulty tolerating 12 gm. lactose as would be found in eight ounces of milk. Milk intolerance developed with

an intake of 18 gm. lactose (equivalent to twelve ounces milk).

Paige et al. (3) studied eighty-nine black elementary school children to show differences in milk consumption by lactose absorbers and malabsorbers. It was found that 54 per cent of the black children were malabsorbers as determined by a lactose tolerance test. Only 58 per cent of this group were classified as nonmilk drinkers (drank less than half by weight of the eight ounces whole milk offered in the school lunch). The black nonmilk drinker had 77 per cent lactose malabsorption as determined by the lactose tolerance test. Of the black milk drinkers, there was only 35 per cent with flat lactose tolerance curves. It was concluded that the black nonmilk drinkers were biologically different than other children studied.

Gudmond-Hoyer et al. (14) studied twenty subjects known to have lactose malabsorption. There was a significant correlation between gastrointestinal symptoms and the amount of lactose ingested. Individual sensitivity varied. Seven patients experienced symptoms while on a lactose-free diet which was related to irritable bowel syndrome. Two of the thirteen patients who were relieved of symptoms on a lactose-free diet developed symptoms with a 5 gm.

lactose intake. The number of subjects developing symptoms on the different levels of lactose is shown in Table 2.

Table 2. Number of subjects developing symptoms at different lactose levels

lactose levels grams	number of subjects
10	3
15	5
20	8
25	9

Lisker et al. (16) conducted a double blind study on 150 subjects. On three consecutive days, 250 ml. milk drinks were given. Milk A contained no lactose. Milk B contained 12.5 gm. lactose. Milk C contained 37.5 gm. lactose. Lactose tolerance tests were administered to determine whether the subjects had sufficient or insufficient lactase activity. Results of the study are shown in Table 3. It was concluded that the frequency of symptoms increased with the lactose content of milk.

Another study conducted by Lisker et al. (17) again indicated an increase in symptoms with an increased lactose ingestion. Of the 200 adults studied, 68.5 per

cent were determined lactose intolerant. Subjects were asked to drink varying amounts of milk. Results are given in Table 4.

Table 3. Percentage of subjects with symptoms for three levels of lactose intake

milk	sufficient lactase activity	insufficient lactase activity
Milk A	no GI symptoms	no GI symptoms
Milk B	3.8%	37.1%
Milk C	7.6%	83.5%

Table 4. Percentage of severe gastrointestinal symptoms for different levels of milk consumption

milk consumption (ml.)	per cent severe GI symptoms
250	5.3
500	28.2
750	26.0
1000	15.3

Of the subjects who were able to consume 1000 ml. milk, 25.5 per cent did not develop symptoms.

Welsh et al. (9) studied gastric emptying of lactose and milk in lactose malabsorbers. Six lactose absorbers

and five lactose malabsorbers (determined by lactose tolerance test) were studied. Each subject ingested four 750 ml. test meals: glucose in water, lactose in water, two per cent milk, and chocolate milk. Glucose and lactose meals were found to have similar gastric emptying rates in both the absorbers and the malabsorbers. It was found that as the osmolarity of the drinks was doubled, gastric emptying decreased significantly. Comparable amounts of milk had similar gastric emptying rates for both absorbers and malabsorbers when the osmolarity was similar. Chocolate milk was found to have a slower gastric emptying rate than plain milk. This was attributed to the higher osmolarity of chocolate milk due to the added sucrose. The difference in the rate of gastric emptying was found to be significant in the malabsorbers.

Gudmond-Hoyer et al. (14) also discussed the effect of whole milk resulting in fewer symptoms than skim milk. Skim milk ingestion resulted in fewer symptoms than lactose dissolved in water. The decreased rate of gastric emptying of whole milk was attributed to the fat content.

The studies indicated that there are differences in lactose tolerance among individuals. Not only is the tolerance affected by the amount of lactose consumed but also the effect of osmolarity of the medium containing the lactose resulting in differing rates of gastric emptying.

PRACTICAL SIGNIFICANCE OF LACTOSE INTOLERANCE. Current literature discusses problems involved in the effects of low milk consumption due to lactose intolerance on health. Problems reviewed in the literature included nutrition programs in developing countries, nutrition programs in the United States, treatment of peptic ulcer disease, osteoporosis, and pregnancy.

General concern has been raised as to the effect of food supplementation programs in which milk is sent to developing countries. Many of the people in developing countries sustain protein-calorie malnutrition. Many of these populations are known to have a primary lactase deficiency. Other populations in developing countries are suspected to have primary lactase deficiency. Secondary lactase deficiency may also be prevalent as a result of protein-calorie malnutrition.

Dahlquist et al. (8) reported that lactase deficiency should be taken into account when milk is used in the treatment of protein-calorie malnutrition. Protein malnutrition itself may result in lactase deficiency. The protein can not be as efficiently digested and absorbed with the increased intestinal motility occurring in a lactose intolerant individual following the ingestion of lactose. Lactase deficiency has been shown to be a significant factor in causing diarrhea in kwashiorkor (8). Dahlqvist et al. (8) concluded that in spite of the symptoms of lactose intolerance, enough protein supplied by the milk can be absorbed to benefit an individual treated for kwashiorkor.

In a review by Ransome-Kuti (11), it was recommended that if milk, especially skim milk, were to be used in the treatment of protein-calorie malnutrition, milk should be used with caution. Fluid and electrolyte balance would need to be carefully monitored if diarrhea were present. If the diarrhea were to become the greater threat, a lactose-free milk should be substituted.

Paige et al. (3) recommended that the milk program which is a part of the school lunch program in the United States be reevaluated. The study conducted by Paige et al.

among black elementary school children indicated that many of these children who did not drink their milk from the school lunch did not drink much milk at home. These same children had a high incidence of lactose intolerance.

If the correlation between low milk consumption and lactose intolerance found by Paige et al. (3) exists generally in American black school children, it is possible that this correlation also exists among other American school children. This could exist among children with Oriental ancestry, American Indians, and Mexican-Americans.

The American Academy of Pediatrics, Committee on Nutrition refutes this concern. The committee stated that lactose intolerance is rarely seen in preadolescents of any ethnic group following the consumption of 250 ml. milk which is the amount usually provided in a serving in the school milk programs. It was felt that it would be inappropriate to discourage the use of milk programs directed at children on the basis of primary lactase deficiency (18).

Ransome-Kuti (11) discouraged the use of a high milk diet in lactase deficient patients treated for peptic ulcer disease. One study cited by Ransome-Kuti showed

that seven out of eight patients with peptic ulcer disease who were tested were lactase deficient. These patients were being treated with a high milk diet for peptic ulcers. Gastrointestinal symptoms began after the institution of the high milk diet.

Newcomer et al. (19) studied the prevalence of osteoporosis and lactase deficiency. It was found that there was a greater prevalence of lactase deficiency in subjects with osteoporosis than the controls. The differences between the results of the two groups were not as a result of ancestry. Heritage was similar for all subjects. The authors concluded that lactase deficiency could predispose an individual to osteoporosis due to a reduced milk consumption as a result of lactase deficiency or impaired absorption of calcium. With decreased milk consumption, there was decreased calcium intake. Dairy products provide two thirds of the calcium intake of North Americans. Lactase deficient subjects consumed less calcium and lactose than the lactose absorbers, even though the lactase deficient subjects had not been overtly aware of the enzyme deficiency. The effect of lactose on calcium absorption has not been concluded as results of studies were found to be presently conflicting.

Newcomer et al. (19) suggested that the relationship of lactase deficiency to osteoporosis may have a genetic basis. Lactase deficiency is thought to be genetically determined. Osteoporosis has been determined to have a significant genetic component. Newcomer et al. concluded that lactase deficiency does not cause osteoporosis by itself. But lactase deficiency may be one of the predisposing factors leading to osteoporosis (19).

Bayless et al. (1) suggested that pregnancy would be complicated in the case of a lactase deficient woman. Milk is a primary source of protein and calcium in the diet during pregnancy. If the milk were not replaced by other foods high in protein and calcium, a low milk consumption could have an effect on the fetus and the outcome of the pregnancy.

Conclusions have not been derived regarding the effect of lactase deficiency on nutritional and health status. Therapeutic measures for such problems as discussed in the literature has not as yet been specifically defined.

Welsh (20) conducted a study to determine the present practice of diet therapy in adult lactose malabsorption. Questionnaires were sent to 473 hospital

dietitians in all fifty states and Puerto Rico. Of the 323 questionnaires returned, 42 per cent stated that the hospital had a lactose-free diet in the hospital diet manual. Less than 1 per cent had a lactose-free diet for peptic ulcer disease for lactose deficient patients. It was found that many of the lactose-free diets were unnecessarily restrictive making the diet difficult for patients to follow. Some hospitals unnecessarily used galactose-restricted diets for the treatment of lactase deficiency.

Welsh (20) recommended that all hospital diet manuals should include a lactose-restricted diet, especially in hospitals where a high percentage of the patients are of races known to have low lactase activity. The author stated that the title "lactose-restricted diet" would be the most appropriate for such diets in that the amount of lactose that an individual can tolerate varies. Lactose seldom needs to be completely eliminated from the diet for a lactase deficient individual. Therefore, no level for the intake of lactose should be specified for a lactose-restricted diet.

It was also recommended that a complete diet history be taken when treating a lactase deficient patient. It

is necessary to determine the amounts and kinds of dairy products consumed daily. The diet history should be utilized to determine what restrictions may be needed for the particular patient (20). This study indicated that there is a need for greater awareness of lactase deficiency and its treatment among hospital dietitians in the United States.

The purpose of this study was to determine the incidence of lactose malabsorption among the young adult population in Cache Valley. This population is primarily of northern European ancestry.

Chapter III: Methodology

The purpose of the study was to determine the incidence of lactose malabsorption among Cache Valley young adults. The incidence of lactose malabsorption was determined by conducting an oral two-hour lactose tolerance test on twenty-four subjects.

Twenty-four young adults were selected from a general nutrition class at Utah State University and a student church group. Volunteers were selected on the basis that they were residents of Cache Valley for a minimum of four years. Both males and females were included in the study.

Subjects signed informed consent forms (Appendix A) and were given written instructions (Appendix B) for preparation for the test. Subjects were scheduled for an oral lactose tolerance test over a five-day period. Subjects were instructed to fast the night prior to the scheduled test, beginning after the evening meal. Water alone was allowed during the fasting period which continued until the test was

completed. Subjects were asked to report to the Conference Room of the Nutrition and Food Sciences Building at 7:00 a.m. Subjects were contacted the evening before the scheduled test to remind them of the necessary procedures. Subjects were to be free of any current gastrointestinal disorders. This reaffirmed by the contact made with the subjects the evening preceding the test.

Upon arrival, each subject had a 3 ml. fasting blood sample drawn by venipuncture into a 3 ml. gray stoppered vacutainer containing NaF, an anticoagulant. Needles used were twenty-two gauge needles. All blood samples were drawn by a registered medical technologist.

After the fasting blood sample was obtained, each subject drank a solution containing 100 gm. lactose in 500 ml. water with 30 ml. bottled lemon juice. The lactose was obtained from Logan Hospital. The hydrous lactose was packaged by Mallinckrodt, Inc. The lactose was weighed on a Mettler balance the day prior to the test

and kept in covered paper cups overnight. The water and lemon juice were added to the lactose prior to the arrival of the subjects. Subjects were encouraged to drink the solution quickly and to swirl it while drinking as the powdered lactose typically does not dissolve completely. The procedure followed for the administration of the lactose tolerance was that currently used at Logan Hospital (21).

Additional 3 ml. blood samples were drawn at 30, 60, 90, and 120 minutes following the ingestion of the lactose. Blood samples were kept at room temperature. After all the samples were collected, they were centrifuged at 4000 rpm for ten minutes in a Beckman centrifuge (model J-21C).

The serum of each sample was tested for glucose using Worthington Statzyme reagent. The reagent consists of hexokinase, ATP, glucose-6-phosphate dehydrogenase, and $NADP^+$. The following chemical reactions occur:

$$\text{Glucose} + \text{ATP} \xrightarrow{\text{Hexokinase}} \text{ADP} + \text{Glucose-6-phosphate}$$

$$\text{Glucose-6-phosphate} + NADP^+ \xrightarrow[\text{dehydrogenase}]{\text{Glucose-6-phosphate}}$$

$$\text{6-phosphogluconate} + \text{NADPH} + H^+$$

Both reactions are essentially irreversible. The total amount of NADH formed in the second reaction, which can be measured spectrophotometrically at 340 nm., is a measure of the total glucose present. This method is specific for glucose. Other methods determine the amount of hexoses present which would not be acceptable as it is necessary to determine only the amount of glucose hydrolyzed and absorbed from the ingested lactose.

To the Worthington Reagent 80 vial, 100 ml. distilled water was added. This was swirled gently to dissolve the reagent and yet avoid denaturing the coupling enzymes which are present in the reagent as recommended by the directions from Logan Hospital.

Each day following the collection of samples, the test was set up as shown in Table 5. Two tests were determined for each blood sample drawn. Moni-Trol I and Moni-Trol II are chemistry controls supplied by DADE Division of the American Hospital Supply Corporation. A glucose clinical standard solution of 200 mg. glucose per deciliter was used for the standard. This was manufactured by Harleco, a division of the American Hospital Supply Corporation.

Table 5. Procedure for glucose determinations

constituents	reagent blank	control I	control II	standard	subject
reagent	1.4 ml.	1.4 ml.	1.4 ml.	1.4 ml.	1.4 ml.
distilled water	10 λ	-	-	-	-
Moni-Trol I	-	10 λ	-	-	-
Moni-Trol II	-	-	10 λ	-	-
glucose standard	-	-	-	10 λ	-
subject serum	-	-	-	-	10 λ

For each test, 1.4 ml. reagent was pipetted. Ten λ serum, control, standard, or distilled water was pipetted with a ten microliter glass disposable micro-sampling pipet. Reagent and sample were mixed in the test tube by vortexing. This was allowed to set at least three minutes at room temperature. Samples were read on a Gilford Spectrophotometer (model Stasar II). The spectrophotometer was set on Absorbance at a wavelength of 340 nm. Absorbance values were read and recorded. Absorbance was adjusted according to the glucose standard in order that glucose values could be read directly. The subject was determined to be a lactose malabsorber if the glucose level of any of the samples was less than 20 mg. per 100 ml. above the fasting sample.

Each subject was asked to fill out a questionnaire to determine his/her current consumption of milk and other dairy products (Appendix C). Subjects were also asked to give information regarding the origins of their ancestry, if known. These forms were mailed to the subjects in order that they would be able to refer to their records at home (Appendix D).

The percentage of lactose malabsorption was determined. A 95 per cent confidence interval was determined for the results of the study. Statistics were hand calculated.

Chapter IV: Results

The purpose of the study was to determine the incidence of lactose malabsorption among young adults from Cache Valley. The incidence of lactose malabsorption was determined to be 16.67 per cent of the subjects studied.

Of the twenty-four subjects studied, there were nine males and fifteen females ranging from ages eighteen to thirty (Table 6). Four of the twenty-four subjects (16.67 per cent) were determined to be lactose malabsorbers (an increase in serum glucose less than 20 mg. per 100 ml. above fasting level). Serum glucose values for each subject are given in Table 7.

Table 6. Breakdown of subjects by sex and age

age	number of subjects	male	female	number lactose intolerant	number lactose malabsorbers
18	2	-	2	-	-
19	5	-	5	1	-
20	3	-	3	-	-
21	3	1	2	1	-
22	1	1	-	-	-
23	2	1	1	-	1
24	4	3	1	-	-
25	1	1	-	-	-
26	1	-	1	-	-
27	-	-	-	-	-
28	1	1	-	-	1
29	-	-	-	-	-
30	1	1	-	-	-

Table 7. Subjects' glucose values

subject number	serum glucose values (mg./100 ml.)				
	fasting	30 minutes	60 minutes	90 minutes	120 minutes
1	99	123	92	97	100
2**	86	86	73	87	94
3	66	126	99	93	87
4	82	127	88	67	92
5*	86	97	85	70	86
6	85	102	104	99	107
7	86	134	126	119	111
8	78	102	90	96	95
9	84	140	93	78	73
10**	79	96	63	62	90
11	87	138	99	94	-
12	91	118	86	98	-
13	87	121	103	90	99
14	85	117	113	115	91
15	90	135	120	109	100
16	92	123	90	87	78
17	85	121	73	91	83
18	87	143	124	93	104
19*	95	107	99	111	111
20	93	120	136	100	106
21	99	148	109	89	85
22	86	127	104	85	96
23	96	123	107	89	89
24	86	116	113	96	86

*Lactose malabsorbers.

**Lactose intolerant.

Of the malabsorbers, three were females. Two of the female malabsorbers were also lactose intolerant as gastrointestinal symptoms were experienced following the lactose tolerance test. One of the lactose intolerant subjects complained of only mild symptoms (slight abdominal cramping). The other subject experiencing more severe intolerance stated she had abdominal cramping, flatulence, and diarrhea.

Information on ancestral origins were obtainable from fifteen of the subjects. Countries listed by the subjects are shown in Table 8. The table also indicates the number of subjects with ancestry from each of the listed countires. Subjects were primarily descendants of northern Europe, particularly the British Isles. One subject was one-fourth Italian. Another subject was one-fourth Lebanese. Both of these subjects were determined to be lactose absorbers.

About half of the fifteen subjects responding to the questionnaire on ancestry were third and fourth generation residents of Cache Valley. The remaining subjects were second generation residents. The lactose malabsorbers were second generation residents.

Table 8. Subjects' ancestral origins, by country

name of country	number of subjects
England	13
Wales	5
Scotland	5
Ireland	2
Sweden	2
Norway	1
Switzerland	6
Germany	3
Denmark	5
Netherlands	2
Italy	1
Lebanon	1

A 95 per cent confidence interval determined for this study was found to be as follows:

$$p = .1667$$

$$p \pm 1.96 \sqrt{\frac{p - (1-p)}{n}}$$

$$.1667 \pm 1.96 \sqrt{\frac{(.1667) - (.8333)}{24}}$$

$$.1667 \pm .1491$$

$$.0176 \leq p \leq .3158$$

A 95 per cent confidence interval for this study would indicate that the actual percentage could be expected to

be between 1.76 and 31.58 per cent with 95 per cent confidence.

A summary of the consumption of milk products obtained from the questionnaire is shown in Tables 9, 10, 11, 12, and 13. Milk product consumption is given for lactose intolerant subjects, lactose malabsorbers, and lactose absorbers. The consumption of milk products varied among all groups of subjects. No general correlation could be made regarding the type and amount of dairy products consumed to lactose tolerance.

The estimated daily consumption of milk products, by servings, is shown for each subject in Table 14. Also shown are the subjects that may be deficient in daily milk consumption and therefore may have low calcium intakes.

Table 9. Type of milk drunk by subjects

type of milk	lactose intolerant	lactose malabsorber	lactose absorber*	total
whole	-	-	8	8
lowfat	2	1	9	12
skim	-	-	4	4
none	-	1	3	4

*Some subjects regularly consumed more than one type of milk.

Table 10. Amount of milk drunk by subjects

amount of milk	lactose intolerant	lactose malabsorber	lactose absorber	total
none	-	1	3	4
$<$ 4 oz.	-	1	1	2
8 oz.	1	-	2	3
16 oz.	-	-	6	6
24 oz.	1	-	7	8
$\geq$ 32 oz.	-	-	1	1

Table 11. Cheese products

number of servings	lactose intolerant	lactose malabsorber	lactose absorber	total
two or more per day	-	1	1	2
one per day	1	-	10	11
two to three per week	1	1	4	6
once a week	-	-	3	3
less than once a week	-	-	2	2

Table 12. Yogurt

number of servings	lactose intolerant	lactose malabsorber	lactose absorber	
once a day	-	-	-	-
two to three per week	-	-	1	1
once a week	1	1	2	4
less than once a week	1	-	9	10
never	-	1	8	9

Table 13. Ice cream and/or sherbet

number of servings	lactose intolerant	lactose malabsorber	lactose absorber	total
once a day	-	-	1	1
two to three per seek	-	-	6	6
once a week	-	1	3	4
less than once a week	2	1	10	13

Table 14. Daily consumption of milk products per subject

subject number	age	sex	number of servings per day			
			milk	cheese	yogurt	ice cream/ sherbet
1	25	M	3	1	-	½
2*	21	F	1	½	-	-
3*	20	F	-	-	-	-
4*	19	F	1	-	½	-
5*	23	F	½	½	-	-
6	24	F	2	1	-	-
7	18	F	2	1	-	½
8*	18	F	-	½	-	-
9	19	F	2	1	-	-
10	19	F	3	1	-	-
11	21	M	4+	1	-	1+
12	23	M	3	½	-	½
13	19	F	3	1	-	-
14*	20	F	-	1	-	-
15	20	F	3	1	-	-
16	26	F	1	2+	½	½
17*	19	F	½	½	-	-
18	25	M	3	½	-	½
19	28	M	-	2	-	-
20	24	M	2	-	-	-
21	21	F	2	-	-	-
22	22	M	3	1	-	-
23	24	M	2	1	-	-
24	30	M	3	-	-	-

*Subjects with possible inadequate calcium intakes relative to RDA.

Chapter V: Discussion

The results of this study support the hypothesis that the incidence of lactose malabsorption among young adults from Cache Valley is similar to other studies of subjects who are of northern European ancestry.

The incidence of 16.67 per cent lactose malabsorption seen in this particular study coincides with the studies reviewed by Simoons (2) with an incidence of 0 to 30 per cent. The 95 per cent confidence interval for this study would indicate that the actual percentage of incidence could be between 1.76 and 31.58 per cent with 95 per cent confidence.

The milk consumption of the subjects indicated that the lactose malabsorbers generally have a low intake of fluid milk. The consumption of other milk products by lactose malabsorbers was not enough to compensate for the low fluid milk consumption. This may result in a possible inadequate nutrient intake. Lactose absorbers with low fluid milk intakes generally did not have adequate intakes of other milk products to assure adequate nutrition.

Of the lactose absorbers, 20 per cent had daily fluid milk intakes of less than four ounces. Of the

twenty-four subjects studied, 33 per cent did not consume at least sixteen ounces of milk or milk equivalent per day. Only two of the subjects with less than sixteen ounces of fluid milk per day indicated that they consumed enough other dairy products to meet the recommendation of the Basic Four Food Groups.

The information obtained from the subjects regarding consumption of dairy products suggests a need for better general nutrition education of the white population as well as other portions of the American population. It is of interest to note that the seven subjects identified for possible calcium deficiency were all female and primarily in their early twenties. The need for nutrients provided by dairy products should be included in nutrition education for the public. There is a need to stress alternatives to fluid milk consumption. The use of milk in soups, puddings, ice cream, sherbet, cheeses, and yogurt available to this population should be encouraged.

The concept of nutrient density could be used in order to educate the public about foods which adequately provide the required nutrients. An Index of Nutritional Quality (INQ) could be used to help identify those foods besides fluid milk which supply adequate calcium in the

diet. The INQ is based on a caloric level of 2000 calories and 900 mg. calcium. The suggested quantitative basis for nutritionally descriptive adjectives for the INQ is shown in Table 15 (22).

Table 15. Nutritionally descriptive adjectives for INQ*

	poor	fair	adequate	good	excellent
INQ for calcium	0.5	0.5-0.89	0.9-1.5	1.51-4.9	5.0

*Taken from Wittwer et al. (22).

A list of INQ values for calcium of different foods is given in Table 16.

There is also a need for awareness that lactase deficiency does exist among the Caucasian population as well as other populations. The prevalence among Caucasians is not as high as many other populations. However, with an incidence of one out of six individuals (and possibly as high as one out of three), nutrition educators should consider encouraging such dairy products as cheeses. Generally, lactose intolerance is not associated with Caucasians and therefore fluid milk consumption is often highly encouraged, especially for

Table 16. INQ for calcium in various foods*

food	serving size	kcal	calcium (mg.)	INQ
blue cheese	1 oz.	100	150	3.33
cheddar cheese	1 oz.	115	204	3.94
cottage cheese, 1% fat	1 cup	165	138	1.86
parmesan cheese, grated	1 tbsp.	25	69	6.13
swiss cheese	1 oz.	105	272	5.76
american pasteurized process cheese	1 oz.	105	174	3.68
american pasteurized process cheese food	1 oz.	95	163	3.81
american pasteurized process cheese spread	1 oz.	82	159	4.31
whole milk	1 cup	150	291	4.31
lowfat milk	1 cup	125	313	5.56
skim milk	1 cup	90	316	7.80
buttermilk	1 cup	100	285	6.33
chocolate milk, lowfat, 2%	1 cup	210	280	2.96
custard, baked	1 cup	305	297	2.16
vanilla pudding	1 cup	285	298	2.32
chocolate pudding	1 cup	320	265	1.84
yogurt, fruit flavored	8 oz.net wt.	230	343	3.31
macaroni and cheese	1 cup	430	362	1.87
cream of chicken soup with equal amount milk	1 cup	180	172	2.12
tomato soup with equal amount of milk	1 cup	175	168	2.13

*Taken from Hansen *et al.* (23).

individuals who are identified as having low milk intakes. It is possible that the individual who is not regularly consuming adequate dairy products may be lactase deficient.

Cheeses contain low levels of lactose. These foods do not require lactase for hydrolysis and absorption. The lactose contents of some dairy products are listed in Table 17. Values for the lactose, protein, and calcium content of some dairy products are given in Table 18.

With an incidence of one out of six individuals with lactose malabsorption, it would seem significant for nutritionists and dietitians to consider possible lactose intolerance in clinical settings. Such settings include pregnant and lactating women in WIC programs and hospitalized patients, particularly patients on a high milk diet for peptic ulcer disease and patients with osteoporosis. The clinical dietitian should be sure that an adequate intake of calcium is available in the hospital diet for patients who do not drink milk as the patient may possibly be an undiagnosed lactose malabsorber and should not be forced to drink milk.

The clinical dietitian should determine the consumption of milk products of a patient newly diagnosed as lactase deficient. The patient should be counseled

Table 17. Gm. lactose in dairy products*

food	amount		lactose		lactic acid	
	household measure	gms.	%	gms.	%	gms
whole milk	1 cup	244	4.8	11.71		
lowfat milk	1 cup	245				
skim milk	1 cup	245	4.9	12.05		
buttermilk cultured	1 cup	245	4.3	10.54	.8	1.96
canned, evaporated milk	1 cup	252	9.7	24.44		
canned, sweetened condensed	1 cup	306	11.4	34.88		
yogurt, partially skimmed	1 cup	226	4.3	10.54	.9	2.03
yogurt, whole milk	1 cup	226	4.1	9.27	.9	2.03
half and half	½ cup	121	4.5	5.45		
light cream	1 tbsp.	15	4.2	.63		
whipping cream, light	1 tbsp.	15	3.6	.54		
whipping cream, heavy	1 tbsp.	15	3.2	.48		
cottage cheese, creamed, low-fat, small curd	½ cup	105	3.3	3.47		
cheese cheddar	1 oz.	28	0-2.1	0-.59		
swiss	1 oz.	28	0-1.7	.48		
blue	1 oz.	28	0-2.0	0-.56		
parmesan, grated	1 tbsp.	5	0-2.9	0-.15		
cream	1 tbsp.	14	1.5-2.1	.21-.29		

Table 17. Continued

food	amount		lactose		lactic acid	
	household measure	gms.	%	gms.	%	gms.
process cheddar	1 oz.	28	0	0		
process cheese food	1 oz.	28	7.0	1.96		
process cheese spread	1 oz.	28	7.0	1.96		

*Compiled from information from Adams (24) and Webb et al. (25).

Table 18. Lactose, protein, and calcium content of some dairy products*

food	amount	lactose grams	protein grams	calcium mg
whole milk	1 cup	11.71	8.5	288
lowfat milk	1 cup		10.3	352
skim milk	1 cup	12.05	8.8	296
buttermilk, cultured	1 cup	10.54	8.8	296
canned, evaporated milk	1 cup	24.44	17.6	635
canned, sweetened condensed	1 cup	34.88	24.8	802
yogurt, partially skimmed	1 cup	10.54	7.7	271
yogurt, whole milk	1 cup	9.27	6.8	251
half and half	½ cup	5.45	3.9	131
light cream	1 tbsp.	.63	.5	15
whipping cream, light	1 tbsp.	.54	.4	13
whipping cream, heavy	1 tbsp.	.48	.3	11
cottage cheese, creamed, low-fat, small curd	½ cup	3.47	14.3	99
cheese				
cheddar	1 oz.	0-.59	7	213
swiss	1 oz.	0-.48	7.8	262
blue	1 oz.	0-.56	6.1	89
parmesan, grated	1 tbsp.	0-.15	2.1	69
cream	1 tbsp.	.21-.29	1.1	9
process cheddar	1 oz.	0	6.6	198

Table 18. Continued

food	amount	lactose grams	protein grams	calcium mg
process cheese food	1 oz.	7.0	5.6	162
process cheese spread	1 oz.	7.0	4.5	160

*Compiled from information from Adams (34) and Webb et al. (25).

depending on the information obtained from the diet history. A patient may find it helpful to drink whole or chocolate milk which has a slower rate of gastric emptying than lowfat or skim milk. The amount of lactose tolerated is highly variable so individual counseling is crucial. Generally a patient will be able to obtain adequate nutrition by the incorporation of small amounts of fluid milk and cheese in the daily diet. Fermented milk is also available should the patient wish to drink more fluid milk than the patient is able to tolerate.

It would be beneficial to include lactose intolerance in the curriculum of dietetic students. Not only should the topic be included in the study of various cultural dietary habits, tube feedings, and disease states in which a secondary lactase deficiency may develop, but it should also be included in the study of general and therapeutic nutrition. It seems that even in a population in which there is a low incidence of lactase deficiency, it is significant to realize that one out of six may have some lactase deficiency. Lactase deficiency among individuals exhibits different levels of intolerance. Some individuals may require a severe

lactose restriction while others may require only a milk restriction.

Twenty-four young adults, age 18 to 30, from Cache Valley were studied to determine the incidence of lactose malabsorption. Lactose malabsorption was determined by a two-hour oral lactose tolerance test. The criteria for a malabsorber was a flat glucose curve in which there was a rise in serum glucose less than 20 mg. per 100 ml. above the fasting level. Four of the subjects were found to be lactose malabsorbers. The incidence of lactose malabsorption for this sample of the population was determined to be 16.67 per cent. Two of the subjects determined to be lactose malabsorbers developed gastro-intestinal symptoms associated with lactose intolerance: abdominal cramping, flatulence, and/or diarrhea. The daily milk intake varied greatly among subjects, both lactose absorbers and malabsorbers.

Populations of northern European descent should be further studied for lactose malabsorption. Studies with a larger sample would help to determine the incidence of lactose malabsorption by decreasing the experimental error. Also it would be beneficial to study the various age groups such as teenagers and the elderly in this same

valley to determine the relationship of age on lactase deficiency.

Lactose malabsorption should be considered in developing general nutrition education programs for the public. Nutritionists and dietitians should definitely consider the incidence of lactase deficiency in the clinical setting when working with the Caucasian population as well as Orientals, American blacks, Mexican-Americans, and American Indians.

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APPENDIXES

Appendix A: Informed consent

Project Title: Incidence of Lactose Intolerance among Young Adults from Cache Valley

PI: Eileen D. Cowles under the direction of Bonita W. Wyse, R.D., Ph.D., Gaurth Hansen, Ph.D.; LeGrande Ellis, Ph.D.; and John Carlisle, M.D.

Procedure: Each subject will be required to fast the night before the test. The subject will ingest lactose in water. 3 ml blood samples will be drawn 0, 30, 60, 90, and 120 minutes following ingestion of the lactose. Blood samples will be drawn by registered medical technologists or phlebotomists.

Possible risks include hematoma (bruising from blood drawing), infection, and/or discomfort as a result of collecting the blood samples. Diarrhea, abdominal cramping, and/or flatulence (intestinal gas) may occur following the ingestion of lactose if the subject is lactose intolerant.

Participants in this study will be informed of a positive lactose intolerance test and receive suggestions for appropriate dietary alternatives for avoiding lactose.

The subject may withdraw consent and terminate participation in this study at any time. The subject may make any inquiries concerning the project procedures.

If medical treatment should be required, the subject will be referred to the Student Health Services.

_____ _____

Subject Date

_____ _____

Primary Investigator Date
Eileen D. Cowles
372 West 200 North #7
Logan, Utah 84321
752-4201

Appendix B: Procedure for subjects participating in lactose tolerance test

You will need to fast the night before the test. Fasting should begin after your evening meal. No food or drink should be consumed except water during the fasting period. You must continue fasting until the test is completed. Cookies and punch will be provided for you after completion of the test.

Please report to the Conference Room (NFS 211) in the Nutrition and Food Science Building at 7:00 a.m. You will be finished by 9:30 a.m.

Date Scheduled_____

Appendix C

NAME_____DATE_____

ADDRESS_____

_____

PHONE NO._____AGE_____SEX_____

SOCIAL SECURITY NUMBER_____

1. Do you drink milk?
   If yes, about how much milk do you drink each day?
   ____½ cup (4 oz.) ____2 cups ____4 or more cups
   ____1 cup (8 oz.) ____3 cups

   What kind of milk do you drink?
   ____Whole milk ____2% milk ____skim milk

2. Do you eat cheese products (cheese, cottage cheese, cream cheese, etc.)? If yes, how frequently?
   ____2 or more servings per day
   ____1 serving per day
   ____2-3 times per week
   ____Once a week
   ____Less than once a week

3. How frequently do you eat yogurt?
   ____1 or more servings per day
   ____2-3 times per week
   ____Once a week
   ____Less than once a week
   ____Never

4. How often do you eat ice cream or sherbet?
   ____1 or more servings per day
   ____2-3 times per week
   ____Once a week
   ____Less than once a week

Appendix D

1. How long have you lived in Cache Valley?

2. Are you the first generation to live in Cache Valley? If not, how many generations of your family have lived in this valley?

3. From what country (or countries) did your ancestors immigrate to the American continent?

If you do not have this information, please give all the following information that you can.

	Name	Birthdate	Place of Birth
Paternal Great-Grandparents			
1.			
2.			
3.			
4.			
Maternal Great-Grandparents			
1.			
2.			
3.			
4.			

If information on great-grandparents is unavailable, please give the information for your grandparents (see next page).

Name	Birthdate	Place of Birth
Paternal Grandparents		
1.		
2.		
Maternal Grandparents		
1.		
2.		