LACTOSE INTOLERANCE AMONG MEXICAN-AMERICANS

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TABLE OF CONTENTS

LIST OF TABLES

LIST OF FIGURES

CHAPTER I

INTRODUCTION

Significance of the Study

Lactose is a major component of the mammalian milk, being only disaccharide, or sugar. Physicians have long recognized that some individuals cannot tolerate milk and become ill after drinking it, but recent attention has been focused on lactose in dairy products. Investigations show that certain populations have an unusually low tolerance to lactose. For instance, 1-24% intolerance among Caucasians, 54-100% intolerance among Asians, and 25-100% intolerance among Africans have been shown (5). Generally, populations that are non-milk drinking also are shown to have a high incidence of lactose intolerance. Investigations are being conducted to determine whether these populations are non-milk drinking because of illness induced by lactose, or whether these populations are intolerant because no milk was consumed (56).

If studies reveal that large numbers of the world population cannot effectively utilize lactose and suffer cramps, diarrhea, flatulence, and distention when it is consumed, the validity of food assistance programs stressing dairy foods may be questioned. Furthermore, other sources of calcium and protein must be stressed. In the United States, minority groups may be particularly affected by lactose intolerance.

Studies have indicated that Negroes, American Indians, Asians, and Mexican-Americans have limited capacity to metabolize lactose (5, 17).

Additional studies are needed to determine why populations become intolerant in adulthood, the age at which individuals become intolerant, the per cent of the population with adult intestinal lactase deficiency, and a simple screening method to determine lactose intolerance.

Statement of the Problem

Three million Mexican-Americans constitute the second largest ethnic group in the United States with significant population concentrations in the Southwest. Little is known of the incidence of lactose intolerance among Mexican-Americans, but three factors indicate high intolerance. A preliminary study by Dill and associates of 11 Mexican-American subjects indicated that 54% of the subjects were intolerant (17). Mexican-Americans are not noted for milk consumption, a frequent characteristic of the intolerant. Many Mexican-Americans have native American backgrounds, and native Americans are believed to be 60-80% intolerant (62). The first part of the problem will be a study of the incidence of lactose intolerance among Mexican-Americans.

The second part of the problem will be the determination of intolerance among families. A genetic development can be supported if predictable patterns of occurrence can be shown.

The following hypotheses were made in this study:

Mexican-Americans have a high incidence of lactose intolerance, with approximately 50% of the adult population intolerant.

The presence of lactose intolerance in a family member may be an indicator of lactose intolerance in other family members.

The Ames Dextrostix/Reflectance Meter may be an effective means of measuring lactose intolerance outside the laboratory.

The objectives of the study were:

1. To determine the incidence of lactose intolerance among a sample group of Mexican-Americans.

2. To determine the frequency of occurrence of lactose intolerance among Mexican-American families.

3. To determine the effectivensss of the Ames Dextrostix/Reflectance Meter in measuring lactose intolerance making the procedure applicable outside the laboratory.

Lactose intolerance was determined in a sample of 33 Mexican-American subjects. The sample included 16 children in four families and 17 persons unrelated by birth. Each subject was given an oral lactose load which was hydrolyzed to galactose and glucose if the necessary enzyme(s) were available. The change in blood glucose, an indicator of the ability to hydrolyze the lactose, was measured with an Ames Dextrostix/Reflectance Meter. The subjects were also interviewed for factors that might influence milk drinking habits.

CHAPTER II

REVIEW OF LITERATURE

Milk is considered one of the more perfect foods as it provides significant amounts of protein, carbohydrate, vitamins, and minerals. Milk is prominent in American diets as one of the major food groups of the Basic Four--A Guide to Good Eating. Milk is highly recommended for the undernourished or the rapidly growing since it is nearly a complete food. In powdered form, milk can be shipped and stored conveniently, making it available for use in national and international food programs (34).

Although milk is regarded as a nearly complete food, investigations show that some people cannot fully utilize the nutrients of milk. These individuals cannot totally digest milk because they have a limited quantity of lactase, the enzyme that cleaves lactose. (34) Lactose is one of the three major nutrients of milk, the others being protein and fat. Lactose, the only milk carbohydrate, is a disaccharide composed of the simple sugars, glucose and galactose. Lactose itself is not absorbed but is hydrolyzed by lactase to its constituent monosaccharides which are readily absorbed (47).

Most disturbances in disaccharide digestion and absorption are a result of limited quantities of the enzymes that normally hydrolyze the complex carbohydrates to their constituent monosaccharides. An individual can be deficient in one specific enzyme, and have difficulty

digesting and absorbing that carbohydrate for which the enzyme is specific. The undigested carbohydrate remains in the intestinal lumen and is eventually eliminated (3).

The disaccharides are hydrolyzed following contact with enzymes located in the brush border of the small intestinal villi (2). In humans, enzyme activity increases from the proximal to the distal duodenum with peak activity occurring in the jejunum since enzymes are not uniformly distributed along the small intestine. There is no appreciable enzyme activity in the normal stomach or colon (59).

Lactose Intolerance

When the individual develops symptoms following the ingestion of disaccharides, the individual is said to be disaccharide intolerant. When the individual is unable to digest the specific disaccharide, lactose, the individual is said to be lactose intolerant. Lactose intolerance may develop under a variety of conditions. Bayless (3) classifies these as follows:

a. Congenital, physiologic lactase deficiency in premature infants

b. Congenital lactose intolerance

c. Acquired lactose intolerance in older children and adults, probably genetically determined.

d. Acquired, secondary to diffuse mucosal damage and generalized disaccharide deficiency, associated with sprue, acute gastroenteritis, kwashiorkor, drug administration and parasites

e. Acquired, secondary to alteration in the intestinal transit, associated with small bowel resection or postgastrectomy.

This review will describe type c, the adult lactose intolerance. Adult lactose intolerance is present when the individual gradually loses his

ability to digest lactose following weaning, shows symptoms of digestive upset when large amounts of lactose are consumed, and shows limited enzyme activity in blood tests (34).

The appearance of lactose intolerance following weaning may be explained if there are at least two intestinal lactases in man. One of the lactases may be "infantile" and decrease after weaning, while the other lactase may be "adult" and persist for life (27). The concentration of enzyme(s) in the intestinal mucosa is the rate-limiting step in disaccharide digestion. Either one or both of the lactases may be deficient in quantity (3). Investigators have been unable to distinguish any qualitative biochemical or physical differences between the lactases isolated from the intestine of infants, tolerant adults, and intolerant adults. The difference is apparently quantitative; there is simply little lactase in the intestine of the intolerant individual (34).

The nature of the lactase(s) make them sensitive to adverse conditions. Fetal maltase, sucrase, and isomaltase reach the lower range of normal adult levels of activity following 28 to 32 weeks of gestation. In both premature and full-term infants, sucrase, maltase, and isomaltase levels are adequate for digestion. In contrast, the major digestive lactase(s) is present only at low levels of activity after 38 weeks of gestation. The lactase levels double and triple just at term to reach normal adult levels in Caucasian infants studied (3).

Hydrolysis rates of lactose are appreciably slower than those of the other disaccharides, maltose and sucrose. When expressed in terms of absorption of monosaccharide constituents, absorption rates for lactose are only half those of sucrose or maltose. In patients with

tropical sprue, lactose hydrolysis was more impaired than that of sucrose or maltose (24).

Manifestation of 'Adult' Lactose Intolerance

'Adult' lactose intolerance is found in healthy teenagers and adults with low levels of lactase. These individuals may experience abdominal bloating, cramps, or diarrhea following the consumption of excessive milk or dairy foods. These individuals were able to drink milk in infancy and early childhood, but in adulthood manifest symptoms of lactose intolerance. The intestinal mucosa is normal, other disaccharidases are normal, and there is no history of gastrointestinal illness leading to mucosal damage and a secondary enzyme defect (3).

Lactose intolerance may be suspected in: 1) abdominal distention or bloating; 2) unexplained cramps; 3) diarrhea; 4) irritable colon syndrome; 5) discomfort following a marked increase in milk consumption, such as pregnancy and peptic ulcer; 6) discomfort following the extended use of supplements containing milk; 7) populations with a high incidence of lactose intolerance; and 8) families with a history of milk or lactose intolerance (2).

Symptoms are experienced when the amount of lactose available to the intestinal cells exceeds the hydrolytic capacity of available lactase. The hydrolytic capacity may be limited by low lactase levels or an unusually large supply of lactose. Lactose is digested in proportion to the lactase available, with the remainder of the lactose passing into the large intestine. As the undigested sugar moves through the intestine, it increases the particle content of the intestinal fluid in relation to the fluid in tissues outside the intestine

(34). The high particle content induces an influx of fluid into the intestine by osmotic action. The fluid entering the bowel is low in electrolytes, and the intestine adds solute-containing fluid to attempt a sodium gradient balance. The increased intestinal volume accelerates transit of the remaining carbohydrate through the small intestine, limiting the hydrolytic capacity of digestive sites in the lower small intestine. The excess fluid induces cramps, distention, and diarrhea. In normal subjects, the hydrolysis of lactose would produce glucose and galactose which would increase sodium transport and overcome the adverse sodium gradient (12).

In the colon, the undigested lactose is fermented by intestinal bacteria to lactic acid and other short-chain organic acids, which, in turn, are broken down to carbon dioxide and hydrogen. The production of these acids raises osmolarity, lowers pH, and interferes with reabsorption of fluid (5). The resulting symptoms are those of a fermentative diarrhea including bloating, flatulence, belching, cramps, and an explosive diarrhea (34).

There is a variation in the amount of lactose required to induce symptoms in the intolerant individual. He may be able to continue drinking milk, although he will not realize its full nutritional value (2). The capacity to handle various levels of lactose may be related to the rate of gastric emptying, the speed of transit of material throughout the intestine, the net secretory response to the osmotic load, the intestinal motor response to the increased fluid, and the irritability of the colon (12).

The amount of lactose in dairy foods influences the individual's ability to tolerate that food. Lactose content in milk from the

world's major dairy animals is 4 to 5%; in ripened cheese 0.1%; in butter 0.40%; in alcoholic fermented milks 2.4%; in buttermilk 1.5%; in sweetened condensed milk 13 to 15%; and dried whole milk, 36 to 38% lactose. Usually the higher the fat content in a food, the lower the lactose content (56). The lower the lactose per cent composition, the greater the quantity of food which can be consumed without inducing symptoms. Ice cold milk seems to induce symptoms more readily than warm milk. Milk consumed with other food is less likely to cause symptoms. The other food may dilute the milk, help delay gastric emptying or release only small amounts of lactose in the intestine. The nutritional content of the food may aid in the intestinal fluid balance (2).

A powdered lactose-free product of milk, Lida-Lac by Lindano Co. of Denmark, has been developed and is satisfactorily tolerated by the lactose intolerant person. The product has been shown to be a useful milk substitute in the preparation of other foods; however, it has a strong taste of caesin which must be disguised (57). Foods such as yoghurt and cheese are usually well-tolerated because the lactose has been naturally fermented to lactic acid. To add variety to the diet of the lactose intolerant person, consideration is being given to using milk in which the lactose is hydrolyzed by added lactose, or to using soy milks. Usually in areas where lactose intolerance is common such as the Orient or Africa, high-lactose foods are rarely consumed. Dietary habits should not be altered to include large amounts of food containing lactose. Other foods should be emphasized that are rich sources of protein and calories, and if possible, native to the area (2).

Geographic Incidence of Lactose Intolerance

Current evidence indicates that low levels of lactase among adults is the norm in much of the world's population. The widespread prevalence of lactose intolerance may eventually make lactase deficiency an anthropological marker of races and nationalities (56). Table I summarizes the incidence of lactose intolerance in healthy subjects, who are adult unless otherwise stated.

Certain trends are notable in the prevalence of lactose intolerance. The studies, when grouped in populations, indicate definite racial patterns. Europeans and American Caucasians have the lowest incidence of intolerance with increasingly greater intolerance among the Mediterraneans, Africans, and Asians tested (23, 25, 32, 43). The percentage of intolerance is frequently characteristic of an ethnic group, and is not markedly influenced by the continent in which the groups reside. For example, tested European Caucasians are 1-24% intolerant, while North American Caucasians are 0-27% intolerant (25, 37, 53, 61). American Negro adults are 70-77% intolerant, while the African Yoruba adult is 84-99% intolerant (4, 35, 45, 62). The Yoruba tribe of Nigeria is believed to be the ancestral home of many American Negroes (35). American-born Chinese had an intolerance level similar to other tested Asians; however, Bolin found that Australian-born Chinese had significantly lower levels than other Asians (7, 28).

There are marked tribal differences in the incidence of intolerance among Africans. Kretchmer (35) tested for lactose intolerance among the four major tribes of Nigeria. The Yoruba and Ibo, living in an area where there was no cattle-raising, did not consume milk until

TABLE I

PREVALENCE OF LACTOSE INTOLERANCE

Population	Locale	No. Tested	% Intolerant	Date	Reference
EUROPEAN	Switzerland	17	6	1965	26
	Denmark	700	1-2	1969	25
	Dannish Eskimo	12	14	1969	5
	Finland	159	18	1970	31
	Slavic	38	24	1972	37
	Australian	12	16	1965	9
	Australian	10	20	1970	10
MEDITERRANEAN BASIN	Palestinean Arab	67	81	1971	22
	Greek Cypriot	17	88	1966	42
	European Jew	53	79	1970	23
	Israeli Jew	93	61	1968	52
	Israeli Jew	208	67	1970	23
	Mixed Jewish	63	60	1970	23
	Askenzai Jew	32	69	1971	36
ASIANS	Indian	22	54	1967	16
	Indian (adult)	12	100	1972	48
	Indian (children)	24	92	1972	48
	Chinese, Indian	29	85	1967	7
	Chinese, American-Born	3	100	1968	28
	Chinese, Australian-Born	34	56	1970	7
	Japanese	25	92	1970	53
	Formosan, Filipino	27	96	1968	28
	Thai	140	97	1969	32
	Thai	75	100	1969	20
	Mixed	11	100	1969	13
	Chinese, Australian-Born	10	80	1970	10
	Chinese	10	90	1970	10
AFRICAN	Nigerian Yoruba	48	84	1971	45
	Nigerian Yoruba	41	99	1971	35
	Nigerian Hausa and Fulani	15	60	1971	45
	Nigerian Hausa and Fulani	50	60	1971	35
	Nigerian Ibos	11	82	1971	45
	Nigerian Mixed	9	100	1971	45

TABLE I (Continued)

Population	Locale	No. Tested	% Intolerant	Date	Reference
AFRICAN (Continued)	Ugandan, Mixed	55	89	1966	14
	Ugandan Rwanda	27	27	1966	14
	Ugandan Ankole	24	25	1966	14
	Ugandan Bantu	22	95	1967	29
NORTH AND SOUTH AMERICA	Caucasian	11	0	1970	53
	Caucasian	50	6	1965	54
	Caucasian	20	10	1966	4
	Caucasian	100	10	1967	43
	Caucasian	145	19	1967	62
	Caucasian	19	19	1965	15
	Caucasian	93	19	1968	40
	Caucasian (Children)	20	10	1967	27
	Caucasian (Teenagers)	16	6	1971	38
	Caucasian (Family Study)*	11	27	1968	61
	American Native and Ethnic Groups				
	Eskimo	25	88	1969	5
	Indian-Eskimo	36	83	1972	18
	Canadian Indian (Teenagers)	30	63	1971	38
	North American Indian	3	67	1967	62
	South American Indian	24	58	1969	1
	South American Mestizo	16	25	1968	5
	South American Androqueno	29	38	1968	5
	Mexican-American	11	54	1972	17
	Negro	20	70	1966	4
	Negro	41	72	1965	15
	Negro	24	75	1968	40
	Negro	22	77	1967	62
	Negro (Family)*	11	100	1968	61
	Negro (Children)	20	35	1967	27

*A family study tests all available members of an individual's family.

recently. The Hausa and Fulani live in northern Nigeria where cattle-raising and ingestion of milk and milk products is traditional. The Yoruba and Ibos have a significantly higher incidence of intolerance than do the Hausa and Fulani.

Age appears to be a determinant in the level of lactose intolerance. In a study by Reddy (48), adult Indians tested had 100% intolerance, while the children were 92% intolerant. Likewise, Huang (27) found in a study of 20 Negro children, 35% were intolerant as compared with 70-77% intolerance among the tested adult population. Huang suggests that the incidence of lactase deficiency in healthy Negroes increases linearly with age. The following diagram indicates the influence of age upon the Nigerian Yoruba on the rise in glucose. (The greater the rise in glucose, the more lactose is being digested, an indication of high lactase levels.) (See Figure 1, page 14).

Some investigators have also described patterns of family tolerance or intolerance in which the incidence is different from that of the ethnic population. The incidence of lactose intolerance among Caucasian general populations is 0-19%, but in testing two Caucasian families where one member of each family was known to be intolerant, the incidence was 27% intolerant. Among American blacks, the incidence of intolerance ranges from 70-77% intolerant. In a study of two volunteer black families there was 100% intolerance (61).

Flatz (20) investigated the families of two Thai families in which there was one lactose tolerant individual per family. In each Thai family, at least one parent and one child were lactose tolerant. Of the 13 family members tested, 6 were tolerant and 7 were intolerant. Among other Thai population studies, the incidence was 97-100%

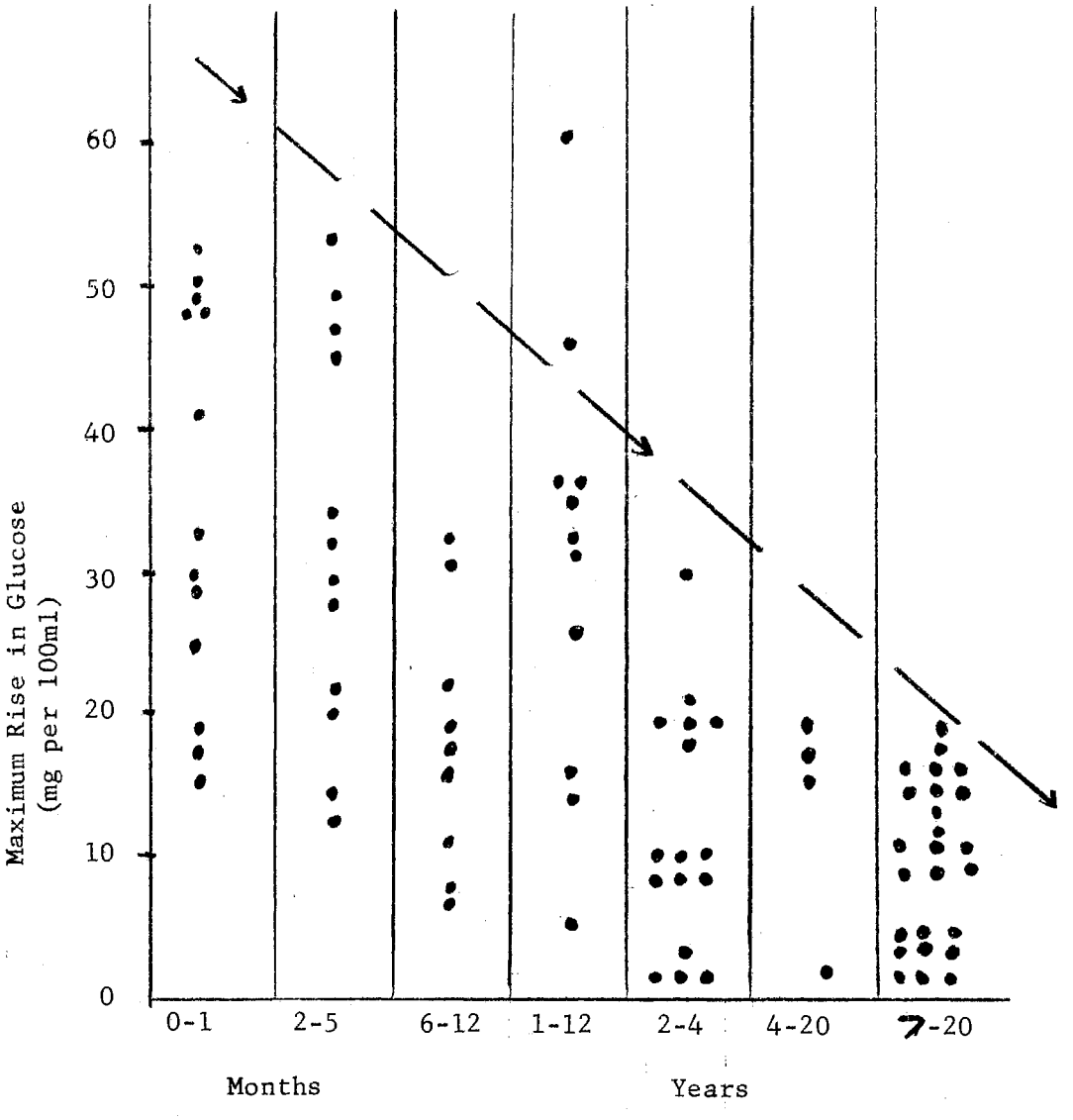

Figure 1. The Relationship of Age to Rise in Glucose, a Test for Lactose Intolerance, Among Pure Nigerian Yoruba (35)

intolerance. In these family studies, based on a known tolerance in one individual, the incidence was 46%.

Intermediate levels of intolerance have been found among the New World groups ranging from 25-83%, perhaps reflecting the variety of ethnic groups that have inhabited and intermarried in the New World. The Antiquenos are a mixture of European, Negro, and American Indian. The mestizo are a mixture of European Caucasian and American Indian (56).

Origins of Group Differences in Lactose Intolerance

Studies indicate that there are distinct regional and ethnic differences in the incidence of lactose intolerance. To explain these differences, four theories have been proposed. When high incidence of lactose intolerance appeared initially in the Near East, Africa, and Asia, it was thought that disease or parasites had damaged the intestinal mucosa, inhibiting lactase production even following recovery. Others suggested drugs or chemicals from food or other environmental sources had inhibited the production of lactase. As studies indicated a widespread incidence of intolerance, more generalized theories were proposed. Scientists related low levels of lactase to low consumption of foods containing lactose, providing inadequate substrate on which the enzyme lactase might act. Others proposed that the ability to digest lactose is genetic in origin (55).

Disease conditions such as intestinal infections, parasites, and protein-calorie malnutrition are known to damage the intestinal mucosa, altering its absorptive capacity. Even though the disease may be

remedied, the damage remains and continues to affect the ability to digest lactose (55). Investigators now believe that disease is not a direct cause of intolerance, but that it does hasten the appearance of lactose intolerance. Groups destined to manifest lactose intolerance as adults may be subject to environmental stresses as children which may hasten lactose intolerance. Better nourished groups may have the ability to delay rapid loss of enzyme activity and thus the onset of inadequate lactose digestion (46).

Lactose intolerance is thought to be related to diet. Some ethnic consumption patterns may include foods or substances that are lactase-inhibiting. Highly spiced foods and betel nut consumption are thought to alter the intestinal mucosa and contribute to malabsorption. It is known that the drug, colchicine, decreases an individual's lactase production, causing the person to become ill if milk is drunk (55). The widespread occurrence of intolerance among people with diverse dietary habits, such as the Eskimo and African Yoruba, has diminished the importance of this theory (18, 45).

Milk consumption may be related to lactase production. In Singapore, the onset of inadequate lactose digestion was apparently delayed for three years in a group of children who continued drinking milk after weaning; however, by the age of six, both milk drinkers and nondrinkers had similar lactase levels (2). Australian-born Chinese, who consumed 15 grams of lactose daily from infancy were 56% intolerant. The native population, who drink little milk, are 90-100% intolerant. The changes in lactase activity were attributed to induction of enzyme activity by the presence of the substrate, lactose (7).

Cuatrecases (15) showed a correlation between milk consumption and

lactose absorption in 60 patients, two-thirds of whom were Negro. While 86% of non-milk drinkers were unable to absorb lactose, only 13% of the milk drinkers could not absorb lactose. Paige (46) found similar results in a study of 521 children of whom 300 were Negro. Among all Caucasian children, 18% were lactose intolerant. Among Negro milk drinkers, 33% of the children were lactose intolerant, and among Negro non-milk drinking children, 77% were intolerant.

Bolin (9) demonstrated a significant fall in lactose absorption when two patients were deprived of milk for five months, suggesting that a decline in enzyme activity took place with no substrate challenge. Kogut (33) found, however, that galactosemic patients maintained on lactose- and galactose-free diets for many years, showed no evidence of lactase deficiency as measured by lactose tolerance tests. It is recognized that once low levels of lactase activity occur, enzyme activity is not influenced by short term, lactose-rich diets (49).

In explaining group differences, geographer Simoons postulates that before humans began consuming milk of animals, they--like other land mammals--experienced a decline in lactase activity after weaning, and these low levels prevailed into adulthood. With the development of milking about 10,000 B.C., most individuals could only consume small quantities of milk without symptoms; therefore, when milk became available in greater quantities, the individual limited his consumption. Milk may also have been limited by some peoples because of cultural and religious influences. Some regard milk as unclean, as food for children, or unfit because of spoilage. At the time of European contact, people living in the American hemisphere, the Far East, and tropical Africa did not drink milk, many of whom are thought to be intolerant.

Simoons (55) suggests that with the passage of time, adult lactose tolerance would not necessarily develop in a population because of its' use of milk or milk products. That development would occur only when genetic selection came into play. Then the aberrant individual, in whom high levels of lactase activity persisted into adulthood, would be favored in the struggle for survival. Under these conditions, the adult with a newly acquired ability to consume large quantities of milk, would enjoy health and vigor, would multiply better, and more successfully defend his family.

A genetic etiology is proposed since the incidence of lactose intolerance in races is reproducible in several parts of the world, regardless of the environmental conditions (2). The tribal differences among Africans also suggest a genetic etiology. Kretchmer (34) found that when a tolerant northern European marries a lactose intolerant Nigerian Yoruba, the offspring are most likely to be lactose tolerant. If a tolerant child resulting from such a marriage marries a pure Yoruba, the children are predominantly tolerant. There is no sex linkage of the genes involved. Kretchmer believes that lactose tolerance is transmitted genetically and dominant (the genes for tolerance from one parent is sufficient to make children tolerant). The children of two intolerant Yorubas are always intolerant, as are the children of a lactose-intolerant European female and Yoruba male. However, intolerance is also believed to be transmitted genetically, perhaps as a recessive trait. Both parents must be lactose-intolerant to produce intolerant children.

Family studies conducted by Welsh and Reddy (48, 61) show that family incidence of intolerance may differ from the norm incidence of

their population. Welsh graphs the occurrence of lactose intolerance among four families in Figure 2.

If lactose intolerance is assumed to be a genetic trait, the incidence of enzyme deficiency in Negroes is too high to be explained by recurrent mutation. It is possible that environmental or selective factors in Africa may have played a role in establishing and maintaining low lactase levels as a trait (2). Flatz (20) suggests that the gene responsible for the formation of intestinal lactase in most Caucasians is replaced in others by an allele governing the formation of a different gene product with some useful function. The enzyme has altered specificity. This allele may convey a selective advantage in an environment where milk is not used for human consumption.

Simoons (56) suggests that it is more promising to focus on the Western pattern of tolerance rather than the Oriental pattern of intolerance. The Oriental pattern is like that of almost all land mammals, with the exception of the guinea pig. The Western pattern is strikingly different with high levels of lactase and lactose tolerance throughout life. This pattern may be aberrant, and the pattern for which research must account.

Bayless and Huang (2, 28) suggest that lactase deficiency and lactose intolerance have a genetic etiology with delayed expression; however, milk consumption and other selective factors influence this expression.

Nutritional Implications of Lactose Intolerance

The high incidence of lactose intolerance among groups according to racial, ethnic, and age characteristics has a variety of nutritional

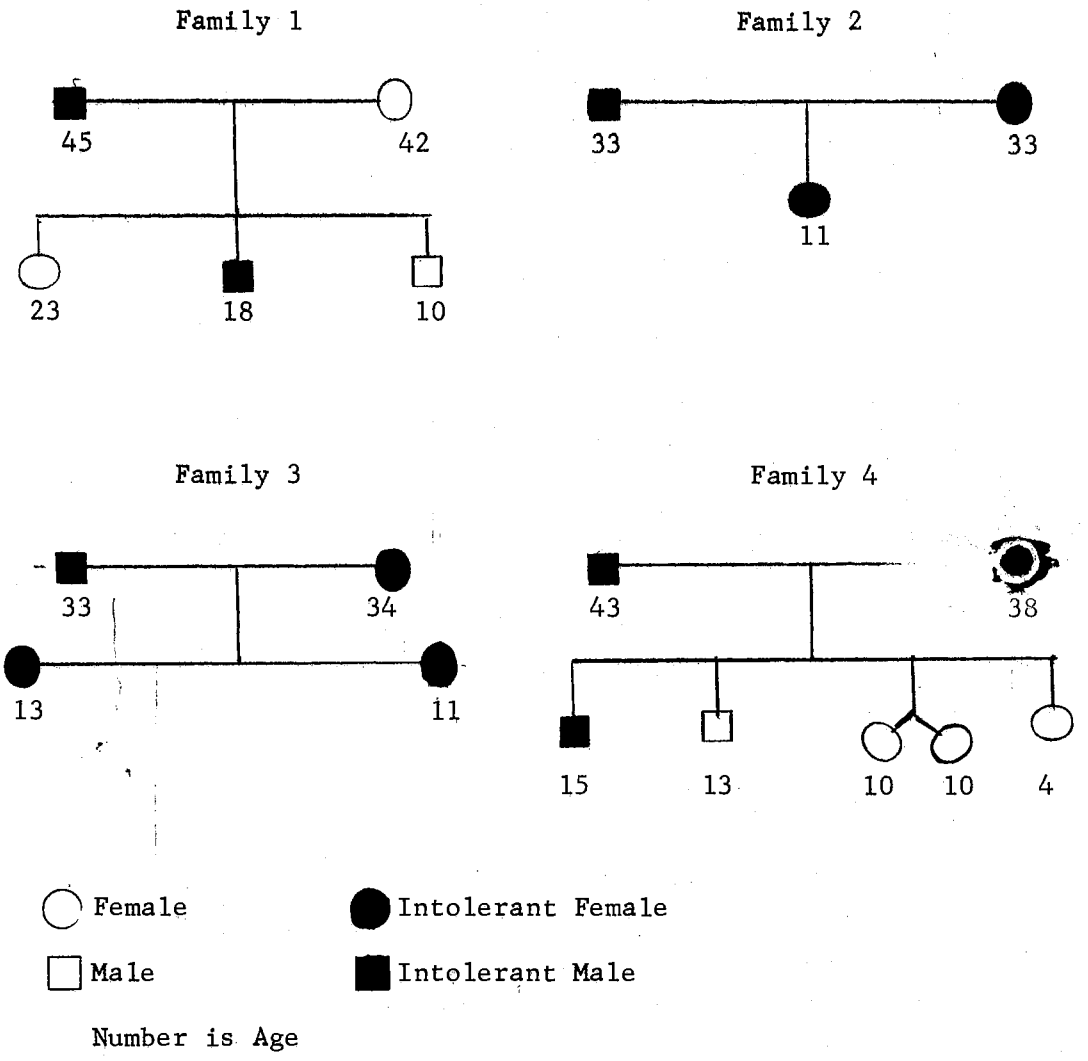

Figure 2. Graphic Description of Lactose Intolerance Appearing in Two Generations of Four Families (61)

implications. The use of milk in low income food programs in which many recipients are Negro may be deleterious and interfere with acceptance of milk (4). Furthermore, the large-scale use of milk in international food programs may not be as beneficial as previously thought. Lactose intolerance may also complicate meal planning and food service in institutions such as schools, prisons, and hospitals (2). Meal planning may be particularly complicated in medical situations involving peptic ulcers, pregnancy, or the use of high protein diets.

Removal of milk from the diet due to intolerance not only decreases the lactose, it also decreases calcium which must then be provided from an alternative source. Lactose may also have a secondary role in metabolism in that lactose may be utilized in the intestine to bind calcium in a form making it available for transport. Lactose may also be an active factor in promoting transport of calcium (53). The lactose-calcium interdependency may also be related to osteoporosis. In tests by Birge, all of a nonosteoporotic control group had normal results on the lactose tolerance tests, while lactose absorption was diminished in nine of 19 patients with osteoporosis (6).

Methods of Determining Lactose Intolerance

Lactose intolerance is directly related to intestinal lactase activity, so the capacity to digest lactose is measured directly or indirectly by the quantity of lactase available. Several methods of measurement are used to determine the lactase activity in the intestine:

a. Oral carbohydrate test followed by blood analysis

b. Oral carbohydrate test followed by measurement of respiratory hydrogen excretion

c. Oral labeled carbohydrate followed by breath analysis

d. Peroral jejunal mucosal biopsies

The most common test for lactose intolerance involves the administration of an oral lactose load and subsequent monitoring of the rise in blood glucose as the carbohydrate is being digested and absorbed. The carbohydrate load is based on the weight of the individual, and may be consumed with water or alcohol (60). After the consumption of lactose, blood glucose levels are taken at timed intervals with frequent determinations in the first hour after ingestion. The blood glucose level may be determined by the Nelson-Somogyi micromethod, by glucose oxidase, or by ferric auto-analyzer (49).

The interpretation of the lactose tolerance test is based on the difference between maximum blood sugar rise compared to the fasting blood glucose. In applying results, a rise in blood glucose of 20mg/100ml or below is abnormal; 20-25mg/100ml is suspect; and, a rise of 26mg/100ml or more is normal tolerance to lactose. The low rise in blood sugar in the intolerant as compared to higher rises in the tolerant can be graphed to show that "flat curve" characteristics of intolerance (60). Best results are obtained by using capillary instead of venous blood (41), and by taking frequent readings in the first hour following ingestion of the lactose (19).

Results of the blood analysis may be influenced by gastric emptying, hydrolysis, absorption, transport, utilization, and excretion. Slowed gastric emptying does not appear to be detrimental to the diagnostic accuracy of the study (59).

Respiratory hydrogen excretion can be measured 30 minutes after a lactose load is taken. Concentration of hydrogen rises rapidly in the breath of subjects from levels as low as 10-20 ppm to values as high as 230 ppm after carbohydrate ingestion. An average of 30 ppm or above indicates intolerance to lactose, while an average of 20 ppm or less is normal (11). The hydrogen measurement reflects the quantity of carbohydrate that is not absorbed whereas blood analysis reflects the quantity of carbohydrate absorbed. This eliminates error from factors such as gastric emptying that influences blood sugar levels after a carbohydrate load. Because of the lack of knowledge concerning bacteria responsible for hydrogen production, it is difficult to predict the factors which influence hydrogen production during carbohydrate fermentation (39). The method is simple to administer, causes no appreciable discomfort, requires no sterile procedure, is sensitive, and can be repeated as frequently as needed (11).

The C^{14}-lactose absorption test measures the amount of radioactive $^{14}CO_2$ in exhaled air following the ingestion of radioactively labeled lactose mixed with a lactose carrier. The labeled lactose is ingested with the carrier lactose, digested, and eventually reaches the lungs as labeled CO_2. The exhaled air is dried, neutralized, and marked with an indicator. The radioactivity is assayed in a liquid scintilation counter and compared with a known standard. The curve of $^{14}CO_2$ excretion in the breath provides an index of the patient's ability to use lactose (53).

Intestinal mucosal specimens are obtained using a capsule to remove tissue from a point beyond the ligament of Trietz where lactase activity is reported to be higher. The tissue is given histologic

examination and enzymatic analysis. The results of the analysis are expressed as units of disaccharidase per gram of protein of the mucosa (19). When jejunal lactase levels less than 2U/g wet weight of mucosa are determined, most persons will develop symptoms with a large lactose load. The development of distention, cramps, loose stools, and a "flat" curve of less than 20mg/100ml rise with a lactose load almost invariably indicates that the person will have a jejunal lactase level of less than 2 units/g wet weight (3).

Quantity of the other disaccharidases can also be determined in biopsy, and a ratio developed expressing the relative enzyme activity. In a study by Figueroa (19) sucrase:lactase ratios were between 3:1 and 6:1 in the control tolerant group and 21:1 and 92:1 in the intolerant group.

CHAPTER III

METHODS AND PROCEDURES

The possible intolerance, or the inability to digest the carbohydrate, lactose, and the frequency with which it occurs in Mexican-American families was investigated in this study. It was postulated that lactose intolerance might exist among Mexican-Americans for the following reasons:

a. Limited previous studies have indicated a high incidence of intolerance among Mexican-Americans.

b. Mexican-Americans drink little milk.

c. Many Mexicans have an Indian heritage and other studies indicate Indians may be highly intolerant.

d. The investigator has observed extensive milk rejection among Mexican-American children.

This study was therefore designed to measure the incidence of intolerance in a sample group, and to test a sample with two family generations to investigate the possibility of genetic patterns.

Methods

Four general methods are available for determination of lactose intolerance: peroral jejunal mucosal biopsies, oral carbohydrate test followed by measurement of respiratory hydrogen excretion, oral labeled carbohydrate test followed by breath analysis, and oral carbohydrate

test followed by blood glucose analysis. The first three methods were rejected due to lack of equipment or lack of experience upon the part of the investigator. The method selected was the oral carbohydrate load followed by analysis of the blood glucose level.

The procedure for blood glucose determination was first investigated as to the most satisfactory method within the limitation of the study. A laboratory procedure, the Nelson-Somogyi micromethod, gives known accurate results but has disadvantages of requiring equipment in a laboratory setting, of requiring a greater quantity of blood from participants, and of requiring that blood be tested immediately for most consistent results. In contrast, a field method, the Ames Dextrostix/Reflectance Meter might give less accurate results but has advantages in being easily set up, in giving fast results, and in requiring less blood.

Each method was evaluated in tests in which a known tolerant and an intolerant person were given an oral carbohydrate load nine times each and the rise in blood glucose determined by both Ames Dextrostix/Reflectance Meter and the Nelson-Somogyi laboratory method. The Reflectance Meter readings were about 10% higher than the laboratory procedure. The readings were plotted for form curves which could be statistically evaluated. The curves from the two methods were not shaped to be statistically significant, due primarily to discrepancies in the fasting blood glucose readings; however, an interaction value indicated that curves from both methods were measuring the same phenomena of rise in blood glucose. Factors could be computed to explain 70% of the variablilty in the readings, and in doing so, produce similar curves from the two methods.

The Ames Dextrostix/Reflectance Meter was therefore selected as a result of these tests and the following further reasons:

1. Families would be tested for lactose intolerance in locations far removed from the laboratory. Blood samples would have to be stored up to 6 hours and transported 70 miles. These conditions would allow blood to deteriorate and less satisfactory readings would result from the laboratory procedure.

2. Subjects could be given a fasting blood glucose test with immediate results to prevent the giving of a carbohydrate load to an unsuspected diabetic.

3. The Ames Dextrostix/Reflectance Meter is a simple, portable device which can be used for tests in homes.

4. Evaluations of the Ames Dextrostix/Reflectance Meter by Joffe and Seftel had shown good correlations between the accuracy of the Meter and laboratory methods (30).

The Subjects

Thirty-three Mexican-American subjects between the ages of 6 and 60 were identified in four locations: Perry, Cushing, Stillwater, and Oklahoma City, Oklahoma. The 33 participants of the study included 16 children of four families and 17 persons unrelated by birth, i.e., singles and spouses. The subjects were identified with assistance from schools and church. Selection was based on convenience and availability, so the sample was not random.

The lactose tolerance tests were usually given early Saturday morning as participants were required to be in a fasting state and Saturday was a convenient day for school children and working parents.

At the time of the test, the purpose of the study was re-explained and participants urged to ask questions before signing the Consent and Release (see Appendix B). Each subject completed a Medical History form (see Appendix) for an indication of unstable intestinal conditions or diabetes. A fasting blood glucose test was given to screen for diabetes and to provide the basis of comparison for maximum blood glucose rise after administration of the lactose. The range of 70-110 mg/100ml was used as the safe level of fasting blood glucose allowing subjects to be tested further. Individuals with higher levels were advised to see a physician.

Procedure

An oral lactose load was given in the amount of 1 g/kg of body weight dissolved in 350-400 ml of water. The 350 ml was used for persons weighing less than 150 pounds, and the 400 ml was used for persons over 150 pounds.

Three fingerprick blood samples were taken at 20 minute intervals following lactose consumption and the results evaluated immediately in a calibrated Ames Dextrostix/Reflectance Meter. The Meter employs a stick impregnated with glucose oxidase which reacts with blood glucose to produce a measurable color change. When the maximum rise in blood glucose was greater than 25 mg/100 ml, the subject was considered tolerant. When the maximum rise in blood glucose was less than 25 mg/100 ml, the subject was considered intolerant (60). Subjects also indicated the presence of any symptoms such as cramps, gas, diarrhea, or distention.

During the course of the test, subjects were questioned about

heritage, milk drinking habits, and other pertinent information (see Appendix B). Forms were also developed in Spanish for those who felt more comfortable speaking Spanish. The Spanish translations were validated by 10 Spanish-speaking residents of Stillwater prior to use.

Several safety measures were built into the study for protection of the subjects as follows:

1. A physician or use of a hospital emergency room was available on standby basis for subjects during the test. The physician or hospital was notified as to whom was being tested and given copies of the Medical History form.

2. The investigator remained with the subject six hours following administration of the lactose, or until all possible adverse effects abated.

3. A diet high in protein and carbohydrate other than lactose was suggested for a period of 24 hours to counter the osmotic effects of the lactose.

4. The study and its procedures were reviewed and accepted by the Oklahoma State University Committee on Research and Experimentation Involving Human Subjects.

Statistical Analyses

The information concerning maximum rise in blood glucose and the dietary interview was placed on 80 column computer cards for a program written in Fortran computer language. The factors coded on the computer cards included the participant number, age, sex, average daily lactose eaten, rise in blood glucose above fasting level, the presence or absence of symptoms, and the readings of blood glucose at 20 minute

intervals from which the rise was computed. Other information gathered in the interview including place of birth, time lived in the United States, and prematurity were determined to be inconclusive and were therefore not analyzed.

A statistical analysis system for North Carolina State University[1] was used for computing the program. The statistical methods used include analysis of variance using the F distribution, plotting two factors against each other to develop a linear relationship, and hypothesis testing to determine the difference in results of this study and previous studies.

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[1] Statistical Analysis System (Copyright Institute of State, Raleigh Division, 1972).

CHAPTER IV

RESULTS AND DISCUSSION

Mexican-Americans between the ages of six and 60 were interviewed and tested for lactose intolerance, the inability to digest the carbohydrate of dairy products due to enzyme deficiency. The 33 participants of this study included 16 children of four families and 17 persons unrelated by birth, i.e., singles and spouses. The subjects were not randomly sampled. The intolerance to lactose was determined by administering an oral lactose load and measuring the maximum rise in blood glucose above the fasting level.

Incidence of Intolerance Among the Subjects

It was found that eight of the 17 Mexican-American subjects (47%) were intolerant to lactose. This incidence of Group I (47%) is statistically consistent with that of Dill (17) who found six of 11 Mexican-Americans intolerant. The incidence among Group II, the blood-related subjects, was found to be 50%. (See Table II, page 32).

Participants of this study were judged intolerant to lactose when maximum blood glucose rise was less than 25mg/100ml following administration of lactose orally. Those subjects with a maximum rise of more than 25mg/100ml above fasting levels were judged lactose tolerant. Based on this criteria, 47% of the non-related subjects tested were lactose intolerant, an incidence much higher than that found for

Caucasians (1-25%). Furthermore, when the number of subjects having similar maximum rises were grouped, it was found that lower glucose rises were more common than higher glucose rises (see Figure 3).

TABLE II

RESULTS OF LACTOSE TOLERANCE TESTS

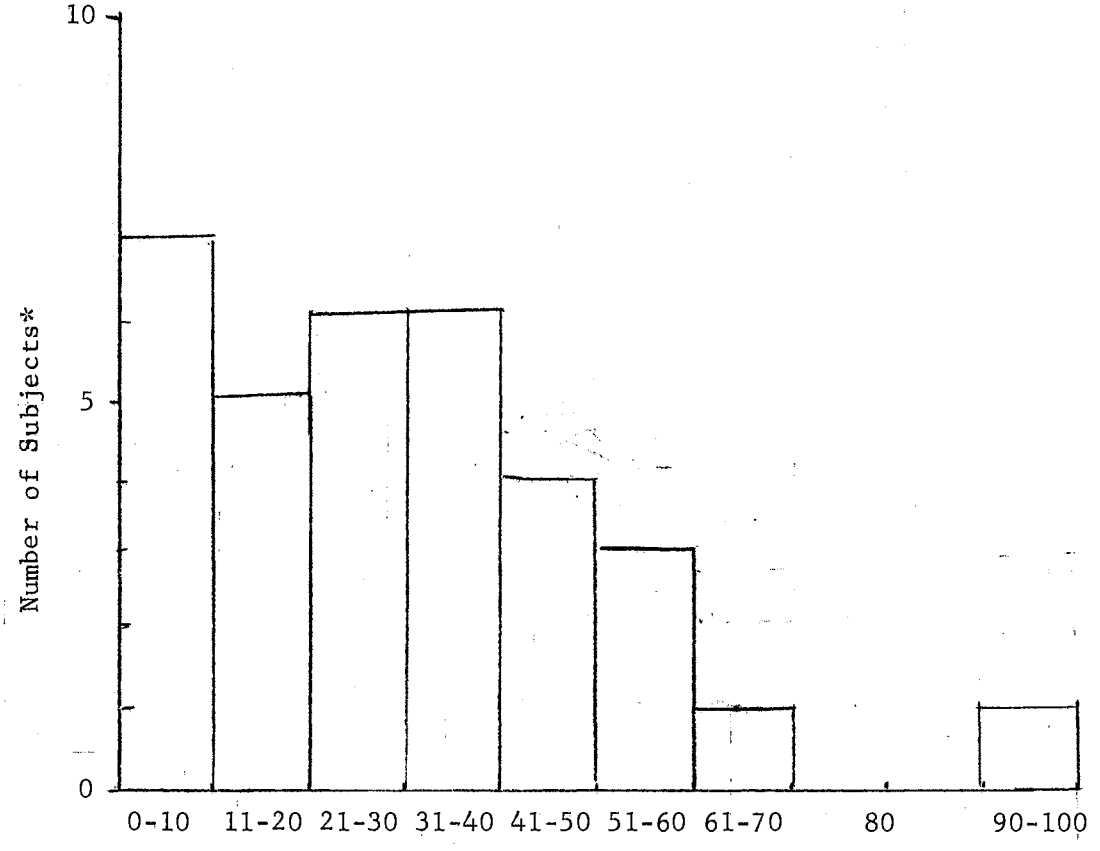
		Blood Glucose (<25mg/100ml)		Symptoms		Blood Glucose (>25mg/100ml)		Symptoms	
	Subjects	No.	%	No.	%	No.	%	No.	%
Group I*	17	8	47	8	47	9	53	9	53
Group II**	16	8	50	8	50	8	50	8	50

*Group I denotes subjects unrelated by blood, i.e., singles and spouses.

**Group II denotes subjects related by blood.

The mean rise in glucose was 31mg/100ml in the total sample of 33 subjects, and the mean rise in blood glucose was 33mg/100ml in the 17 subjects unrelated by blood.

These results show a significant incidence of lactose intolerance in the Mexican-American subjects studied. Lactose intolerance indicating lactase deficiency may be a significant health factor for Mexican-Americans. Since there are approximately three million Mexican-Americans in the United States, there is potential for great numbers of people to be intolerant to milk and certain dairy products. The potential would be particularly significant in the Southwest where the



*Denotes total sample of 33 subjects including blood relatives

Figure 3. Grouping of Subjects with Similar Rises in Maximum Blood Glucose

majority of the Mexican-American population reside.

The results of the lactose tolerance tests showed a correlation between rise in blood glucose and the presence or absence of symptoms. (See Table II). Subjects who had a rise in blood glucose of less than 25mg/100ml also had symptoms of lactose intolerance, i.e., diarrhea, nausea, flatulence, and distention. Those subjects with a rise greater than 25mg/100ml did not exhibit these symptoms. Thus, the eight subjects who had low rises in blood glucose also experienced symptoms.

A statistically significant relationship between the appearance of symptoms and rise in blood glucose was shown. The value observed (58.45) is significant at the 0.1% level (critical value = 13.29), so there is a marked relationship between rise in blood glucose and the presence or absence of symptoms.

TABLE III

ANALYSIS OF THE VARIABLE 'SYMPTOMS'

Sources	Degrees of Freedom	Sum Squares	Mean Squares	F Value
Symptoms	1	9658.5028	9658.5028	58.45
Residual	31	5122.4669	165.2408	

Effects of Sex, Age, and Lactose Eaten on Blood Glucose Rise

A low rise in blood glucose denoting lactose intolerance was found more frequently in females (50%) than in males (43%); however, the difference was not statistically significant. In this study there was no relationship between lactose intolerance and sex of the subjects. The subjects with no rise at all and who experienced severe symptoms were both female.

If more is to be learned about lactose intolerance, the age at which intolerance is most frequently manifested must be ascertained. In this study, there were no children under the age of 10 who were intolerant; however, one male child, age 11, was found to be intolerant. Of the 16 persons found to be intolerant (including blood relatives), 75% were intolerant after the age of 20 while 25% were intolerant before the age of 20. The relationship between age and intolerance is shown in Table IV.

TABLE IV

AGE AND LACTOSE INTOLERANCE

	Number of Subjects	
Age	Lactose Intolerant (16)	Lactose Tolerant (17)
6-10	0	1
11-15	1	1
16-20	3	3
20-30	7	9
➤30	5	3

Because of the size of the sample and lack of randomness, there is no discernible relationship between age and lactose intolerance in this study. Age was a factor found to be statistically insignificant.

Each of the 33 subjects tested for lactose intolerance was interviewed to determine the amount of lactose-containing food normally consumed, and the average daily amount of lactose consumed was computed. The following table shows the results.

TABLE V

LACTOSE CONSUMPTION AND INTOLERANCE

Daily Average Lactose Eaten	Number of Subjects	
	Lactose Intolerant (16)	Lactose Tolerant (17)
<15 grams	4	2
15-30 grams	6	7
>30 grams	6	8

Statistically there was no obvious relationship between the average daily lactose eaten and lactose intolerance. The investigator observed that the amount of milk consumed and lactose eaten from other dairy products was more a result of family eating habits than lactose intolerance. Several subjects realized that dairy products caused gastric upset, but still continued to consume large quantities of milk. Sports also influenced lactose consumption. Three of the six intolerant

subjects consuming more than 30 grams of lactose were varsity athletes who drank large quantities of milk as part of their training program knowing that the milk frequently resulted in gastric upset (the symptoms of lactose intolerance). It was also noted that those persons experiencing severe symptoms in the lactose tolerance test drank practically no milk.

Genetic Patterns

Lactose tolerance tests and dietary histories were obtained from 23 members of four Mexican-American families. On the basis of flat lactose tolerance tests, 12 of the 23 family members were judged to be lactose intolerant. The members of Family 1 showed rises in blood glucose over the 25mg/100ml level and showed no symptoms of lactose intolerance. Since neither parent was intolerant, neither of the children was intolerant, as would be expected with a genetic etiology. (See Figure 4).

Both parents of Family 2 exhibited low rise in blood glucose after taking the lactose and had severe symptoms. If a genetic etiology were proposed, one would expect each of the children to also show characteristics of lactose intolerance. The three older children ranging in age from 17 to 21 had flat lactose tolerance tests and marked symptoms; however, the two younger children, ages 9 and 11, were not lactose intolerant. If one considers the theory of an "infantile" lactase which loses potency and is supplanted by an "adult" lactase with advance in age, there is a plausible explanation for this discrepancy. Perhaps the "infantile" lactase is still effective in hydrolyzing the lactose; however, tests in two to three years might show intolerance as the

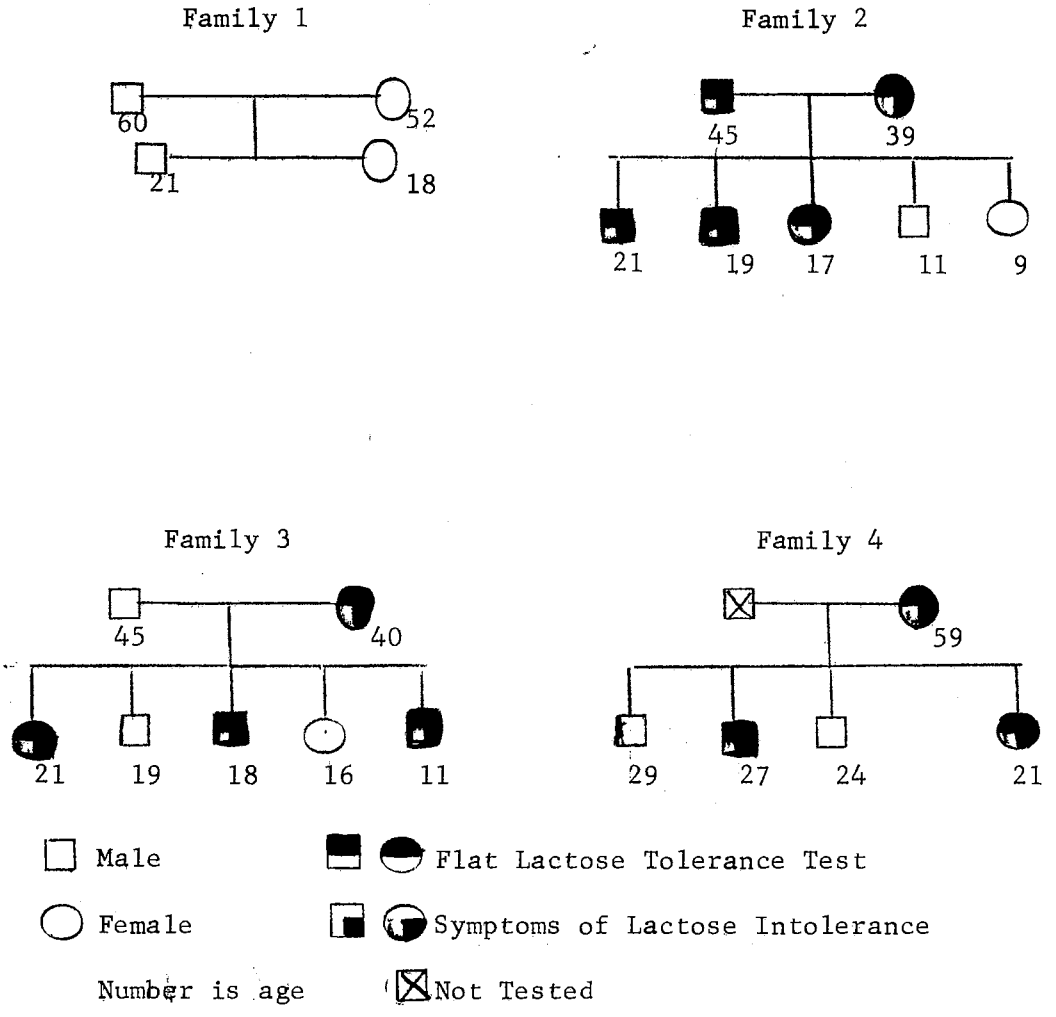


Figure 4. Graphic Documentation of Lactose Intolerance for Four Mexican-American Families

"infantile" lactase was not replaced with the "adult" lactase. (See Figure 4).

A single parent in Family 3 was intolerant, and as a result only part of the children showed symptoms or a limited rise in blood glucose. The intolerant parent had very severe symptoms and had no rise in blood glucose. The eldest daughter, who had very severe symptoms and no rise, had refused to drink milk since age five because of an adverse reaction. (See Figure 4).

Family 4 had only one parent available for testing, and this individual showed no rise in blood glucose and exhibited severe symptoms. Two of the four children were also intolerant. It is interesting to note the contrasts in the blood glucose readings of the children. The two intolerant children showed rises of 8mg/100ml and 4mg/100ml while the two tolerant children had rises of 65mg/100ml and 54mg/100ml. (See Figure 4).

A third generation would have been available in Families 2 and 3 except that members of the third generation were diabetic.

Family Studies of Lactose Intolerance

The supposition of a genetic basis of lactose intolerance has implications for application of nutritional counseling. In families where lactose intolerance is found in one or both parents there may be counseling with parents concerning the possibility of intolerance in their offspring and methods of minimizing any discomfort due to lactose intolerance. At the same time, parents and children need additional nutrition education for recognizing alternative sources of calcium and protein.

Effectiveness of the Ames Dextrostix/ Reflectance Meter

The Ames Dextrostix/Reflectance Meter was found to be an effective field method for evaluating lactose intolerance. There was a very high correlation between the occurrence of symptoms and low rises in blood glucose, both of which are indicative of lactose intolerance. The Meter provided an immediate determination of the fasting blood glucose which not only served as the basis for determining maximum rise, but also could be used as a screening method for unsuspected diabetes. The unsuspected diabetic can then be advised to see a physician and does not participate in the consumption of the carbohydrate load.

The Meter appeared to be sensitive to a wide range of blood glucose readings. This was observable when several subjects were being tested together and the group included tolerant and intolerant persons.

CHAPTER V

SUMMARY AND CONCLUSIONS

Thirty-three Mexican-Americans between the ages of six and 60 were interviewed and tested for lactose intolerance, the inability to digest the carbohydrate of dairy products due to an enzyme deficiency. The participants of this study included 16 children of four families and 17 persons not related by birth, i.e., singles and spouses. The subjects were selected on the basis of availability, therefore were not randomly selected.

Determination of the presence of lactose intolerance was based on a rise of less than 25mg/100 ml of blood glucose from the fasting level after the subjects consumed an oral lactose load based on the weight of the subject. The rise was measured by an Ames Dextrostix/Reflectance Meter. An interview was used to determine milk drinking habits.

It was found that 47% of the 17 non-related Mexican-Americans were intolerant to lactose, a level significantly higher than that of Caucasians. A marked relationship between low blood glucose rise and evidence of symptoms further verified a judgment of intolerance in the subject. There was no relationship between age, sex, or lactose consumed and lactose intolerance in this study.

The finding of intolerance in two successive generations of three families and in both sexes of the families adds support to the contention that lactose intolerance has a genetic basis, without sex

predilection. This would indicate implications for those agencies working in mass food service operations or agencies working with Mexican-American families and diet.

The Ames Dextrostix/Reflectance Meter was found to be an effective field method for evaluating lactose intolerance. It also can serve as a screening method to eliminate unsuspected diabetics.

From this study, it is suggested that the incidence of lactose intolerance is sufficient to warrant further investigation among Mexican-Americans. An ideal procedure would be that of including lactose studies in the multiphasic health screening conducted by the State Department of Health. The sample would be more random and could be studied in conjunction with diabetes screening.

It is also suggested that since a significant incidence of intolerance has been found, family studies should continue until it is learned whether lactose intolerance is a result of a dominant or recessive gene. These investigations might include other metabolic malfunctions as related to lactose intolerance.

Continued studies need to be made of ways to effectively test for lactose intolerance which would produce little subject discomfort and could be used with large populations. The Reflectance Meter is one type of procedure that could be further evaluated for use in additional studies.

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APPENDIX A

Number	Ages	Sex	Weight	Place of Birth	Time in the United States	Premature	Continued Milk Drinking	Amount of lactose eaten	Rise	Symptoms	Readings	Relationships
1.	45	M	145	L.A., California	All life	No	Yes	24 grams daily	36 mg/100	None	104-120-140-90	Father
2.	40	F	135	Perry, Ok.	All life	No	No	4 grams daily	Flat curve	Yes, diarrhea, cramps	135-135-115-100	Mother
3.	19	M	183	Perry, Ok.	All life	No	Yes	26 grams daily	40 mg/100	None	80-84-120-84	Son of 1 and 2
4.	18	M	136	Perry, Ok.	All life	No	Yes	38 grams daily*	11 mg/100	Yes, cramps, diarrhea	87-96-86-82	Son of 1 and 2
5.	16	F	115	Perry, Ok.	All life	No	Yes	24 grams daily	64 mg/100	None	76-84-120-84	Daughter of 1 and 2
6.	11	M	85	Perry, Ok.	All life	No	Yes	30 grams daily	24 mg/100	Diarrhea	92-116-106-110	Son of 1 and 2
7.	21	F	125	Perry, Ok.	All life	No	No	2 grams daily	Flat Curve	Diarrhea, cramps, nausea	98-98-92-96	Daughter of 1 and 2
8.	25	M	180	Culver City, Cal.	All life	No	Yes	62 grams daily	50 mg/100	None	70-120-115-100	Husband to 9
9.	24	F	130	Centralia, Ill.	All life	No	Yes	68 grams daily	40 mg/100	None	70-84-110-106	Wife to 8
10.	25	M	202	Chicago, Ill.	All life	No	Yes	55 grams daily	24 mg/100	Diarrhea, nausea	76-86-100-96	Husband to 11
11.	21	F	115	Shadduck, Ok.	All life	No	Yes	61 grams daily	50 mg/100	None	70-92-120-114	Wife to 10
12.	25	M	140	Independence, Ks.	All life	No	Quit age 10	8 grams daily	45 mg/100	None	85-135-130-110	Husband to 13
13.	23	F	150	Chanute, Ks.	All life	Yes	Yes	11 grams daily	39 mg/100	None	96-122-135-110	Wife to 12
14.	60	M	148	Zamra, Mexico	30 years	Unknown	Unknown	17 grams daily	43 mg/100	None	92-90-135-95	Husband to 15, Father
15.	52	F	132	Almonte, Cal.	All life	No	Yes	27 grams daily	39 mg/100	None	96-98-135-117	Wife to 14, Mother
16.	21	M	126	Omaha, Neb.	All life	No	Yes	40 grams daily	28 mg/100	None	82-110-110-108	Son to 14 and 15
17.	18	F	126	Omaha, Neb.	All life	No	Yes	44 grams daily	56 mg/100	None	74-104-130-120	Daughter to 14 and 15
18.	21	M	122	Oklahoma City	All life	No	Yes	85 grams daily*	18 mg/100	Gas, bloated feeling	80-88-98-90	Son to 19 and 20
19.	45	M	170	Oklahoma City	30 years	No	Yes	27 grams daily	8 mg/100	Diarrhea, bloated	92-100-108-100	Father
20.	39	F	100	Washington, D. C.	All life	No	No	20 grams daily	20 mg/100	Diarrhea, bloated	74-82-94-78	Mother
21.	19	M	125	Oklahoma City	All life	No	No	45 grams daily	6 mg/100	Bad diarrhea	80-82-82-86	Son to 19 and 20
22.	17	F	115	Oklahoma City	All life	No	No	7.5 grams daily	18 mg/100	Diarrhea, upset	74-75-92-78	Daughter to 19 and 20
23.	9	F	49	Oklahoma City	All life	No	Yes	33 grams daily	40 mg/100	None	70-75-110-75	Daughter of 19 and 20
24.	11	M	62	Oklahoma City	All life	No	Yes	48 grams daily*	32 mg/100	None	74-100-106-88	Son of 19 and 20
25.	24	F	128	Oklahoma City	All life	No	Yes	52 grams daily	22 mg/100	Gas, cramps, diarrhea	84-96-106-106	Unrelated
26.	24	M	164	Pharr, Texas	All life	No	Yes	22 grams daily	52 mg/100	None	70-122-118-108	Son of 28
27.	20	F	112	Tulsa, Ok.	All life	No	Yes	36 grams daily	18 mg/100	Diarrhea, stomach upset	86-100-104-86	Unrelated
28.	59	F	112	Pharr, Texas	All life	Unknown	No	18 grams daily	2 mg/100	Diarrhea, stomach distress	96-98-90-88	Mother
29.	29	M	180	Pharr, Texas	All life	No	Yes, goat milk	24 grams daily	54 mg/100	None	76-108-118-130	Son of 28
30.	21	F	105	Pharr, Texas	All life	No	Yes, goat milk	30 grams daily	8 mg/100	Diarrhea	86-94-94-86	Daughter of 28
31.	27	M	175	Pharr, Texas	All life	No	Yes, goat milk	18 grams daily	4 mg/100	Diarrhea	86-90-90-86	Son of 28
32.	23	M	150	Gove, Ok.	All life	No	Yes	36 grams daily	96 mg/100	None	84-130-180-170	Unrelated
33.	22	F	100	Anadarko, Ok.	All life	No	No	5 grams daily	24 mg/100	Diarrhea	86-88-110-88	Unrelated

APPENDIX B

Name_____ Date_____

Address_____ Age_____

Weight_____ Height_____ Sex_____ Dosage_____

Place of Birth_____

1. How long have you lived in the United States?_____

2. Were you premature?_____

3. Did you continue drinking milk after the age of two?_____

4. What amounts of the following foods do you eat?

	Daily	Weekly	Monthly
Whole milk			
Skim milk			
Powdered milk			
Canned milk			
Chocolate milk			
Malts and Shakes			
Ice Cream			
Cheese			
Yoghurt			
Gravy made with milk			
Cream soups made with milk			
Custard			
Sherbet			
Butter or margarine			
Puddings made with milk			
Cream pies			

5. What foods cause gas, distension, bloated feeling, diarrhea, vomiting, or cramps (not during menustration)?_____

_____

6. Do other family members have these feelings, which one?_____

_____

7. Are you going to the doctor, for what?_____

8. Do you have food allergies?_____

9. Name of Father_____

Mother_____

Grandfather_____

Grandmother_____

"Identification of Lactose Intolerance"

Name_____ Date_____

Address_____ Phone_____

Age_____Other_____ Weight_____

SYMPTOMS

Yes No

___ ___ High Fasting Blood Sugar

___ ___ Family History of Diabetes

___ ___ Blood in the Stool

___ ___ Tarry Stool

___ ___ Stomach or intestinal ulcers

___ ___ Continuous or frequent diarrhea

___ ___ Stomach or intestinal surgery

___ ___ Food Allergies

___ ___ Limited Milk intake by doctor

___ ___ Milk restriction as an infant

___ ___ Inability to swallow

Lactose Dose: 1 gram of lactose per kilogram of body weight. Given orally in distilled water. Diluted to 350 ml for the person less than 150 lb., and to 400 ml for the person more than 150 lb.

Investigator: Mary Frances Sowers, 377-0674
4100 W. 19th, E 101
Stillwater, Ok. 74074

STATE OF OKLAHOMA)
) SS
COUNTY OF_____)

CONSENT AND RELEASE

I, _____, hereby consent to participate in a study of "Identification of Lactose Intolerance Among Mexican-Americans" conducted by Mary Frances Sowers under the supervision of Esther Winterfeldt, and acknowledge that I have been informed and fully understand the following, to wit:

1. I have had the study explained to me and have been given the opportunity to ask questions and receive answers to questions about the procedures and the possible physical effects of participating in such a study.

2. I have been informed that I am free to withdraw from participation in the study at any time.

3. I have been given the name and telephone number of a doctor to contact with respect to my participation in this project if the need arises.

In consideration of the benefits I shall receive and the benefits to the Mexican-American population that will be made possible through information received from this study, I hereby release and forever discharge the Board of Regents for the Oklahoma Agricultural and Mechanical Colleges and Oklahoma State University of Agriculture and Applied Science, its agents, employees and assigns, and Mary Frances Sowers from all claims, demands, damages, actions or causes of action arising from or connected with my participation in the study of "Identification of Lactose Intolerance Among Mexican-Americans".

Witness my hand this _____ day of _____, 19___.

_____
Signature of Participant

Witness:

_____
Name

_____
Address

_____
Signature of Parent or Guardian of Participant

APPENDIX C

Mary Frances Sowers
4100 W. 19th, E 101
Stillwater, Oklahoma 74074
405-377-0674

Mr. Gilespie, Administrator
South Community Hospital
1001 SW 44th
Oklahoma City, Oklahoma 73109

Dear Sir:

This letter is to confirm our telephone conversation of March 20, in which it was agreed that the emergency room of South Community Hospital would be available to handle emergencies, if needed, for nutritional research related to lactose intolerance. The participants in the study would be given the telephone number of South Community Hospital for referral as required.

This study involves the administration of lactose, the carbohydrate of milk, and the taking of 4 fingerstick blood tests. The first blood test is taken in the fasting period, and the remaining three tests are taken at 20 minute intervals following the ingestion of the lactose solution. Blood glucose will be measured using an Ames Dextrostix/ Reflectance Meter.

It is expected that persons having adequate levels of the enzyme lactase will easily digest the lactose, and show a rise in blood glucose of 40-60mg/100ml above the fasting level. Those having inadequate levels will show a rise of less than 25mg/100ml above fasting levels.

We anticipate no difficulty; however, to protect the participants against some kind of unsuspected diabetic reaction or continued diarrhea due to some intestinal malfunction, we want the participant to recognize that medical assistance is available.

I am enclosing a copy of the medical checklist used in this study. Please note that the dosage determination is on the sheet along with my name, address, and phone number. I hope we will not have to avail ourselves of your service, but I do thank you for your assistance.

Yours truly,

Mary Frances Sowers

VITA

Mary Frances Sowers

Candidate for the Degree of

Master of Science

Thesis: LACTOSE INTOLERANCE AMONG MEXICAN-AMERICANS

Major Field: Food, Nutrition and Institution Administration

Biographical:

Personal Data: Born in Wichita, Kansas, May 30, 1947, the daughter of Mr. and Mrs. Robert Roy.

Education: Graduated from Frederic Remington High School, Whitewater, Kansas, in May, 1965; received Associate of Arts degree from Butler County Junior College, El Dorado, Kansas, May, 1967; graduated magna cum laude with a Bachelor of Science in Education from Kansas State Teachers College, Emporia, Kansas, January, 1969; completed requirements for the Master of Science degree at Oklahoma State University, July, 1973.

Experience: Extension Home Economist, Kingman County, Kansas, 1968-1970; Supervising Extension Home Economist, Oklahoma County, Oklahoma, 1971-1972; Television graduate assistant, Oklahoma State University, Division of Home Economics, 1973.

Professional Organizations and Honors: American Home Economics Association, Oklahoma Home Economics Association, public relations chairman; member of Phi Kappa Phi, Theta Epsilon, and Omicron Nu Honor Society; Who's Who.