SOME NUTRITIONAL CONSIDERATIONS OF LACTOSE MALABSORPTION IN MEXICAN AMERICAN CHILDREN

by

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Dissertation submitted to the Graduate Faculty of the
Virginia Polytechnic Institute and State University
in partial fulfillment of the requirements for the degree of
DOCTOR OF PHILOSOPHY
in
Human Nutrition and Foods

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November, 1974

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ACKNOWLEDGMENTS

A research survey such as this involves the cooperation of many people. Each contributes special talents and resources invaluable to the success of the survey.

I wish to thank Dr. Eleanor A. Young, Dr. Elliot Weser, and Dr. S. J. Ritchey for their guidance and for reading and commenting on this manuscript. I would also like to thank the other members of my committee for their help -- Dr. Ryland Webb, Dr. Forrest Thye, and Mr. Rodney Young.

Dr. Thomas H. Woteki was most helpful in the statistical analysis of the collected data. Mr. Curtis Shoup adivsed on the preparation of the data for computer analysis.

Mr. Stephen Weiss collected the blood samples and performed the blood analyses. Mrs. Elizabeth Quintana assisted with dietary interviews.

Special recognition is due to Dr. Jerry Newton and to Dr. Cornelius Nau physicians for the San Antonio and Northside Independent School Districts and to the school nurses. Dr. Newton and Dr. Nau established our initial contact with the schools, while the school nurses made sure our visits ran smoothly.

Finally, I would like to recognize the 333 children who participated in the survey. Almost without exception they cooperated during both the blood test and the dietary interview. It was a pleasure to work with the children.

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INTRODUCTION

In recent years, reports have appeared in the medical literature on the occurrence of low intestinal lactase activity and lactose intolerance in apparently healthy populations in different parts of the world. These individuals have been demonstrated to have low lactase activity in the intestinal mucosa, and administration of an oral load of lactose brings about a low blood glucose response which may be accompanied by moderate to severe gastrointestinal disturbances. It has also been shown that milk and milk products (the only natural sources of lactose) consumed in quantity at one time may also induce the clinical symptoms of lactose intolerance.

Some controversy has arisen over the nutritional significance resulting from lactose intolerance in a growing child. Since some individuals with low lactase levels have abdominal cramping and diarrhea after consuming milk, they may voluntarily curtail milk intake to avoid the symptoms. Low intake of milk may cause a low intake of calories and the nutrients found in significant amounts in milk - calcium, riboflavin, protein, and vitamin A. Little work has been done to define the relationship between lactose intolerance and milk nutrient intakes, or lactose intolerance and growth in children.

The population groups in the Western hemisphere that have been examined include North and South American Indians, Peruvian Mestizo children, Colombian medical students, Bushnegroes and Creoles in Surinam, Mexican adolescents and adults, Mexican American adult males, and Negroes, Caucasians and Orientals in the United States. Since a high incidence of lactose intolerance was found in North and South American Indians, in

Mestizo children, and in Mexican American adult males, and since the Mexican American's ancestry is a mixture of European (Spanish) and Indian, it is hypothesized that Mexican American children also exhibit a high incidence of lactose intolerance. The present study was therefore undertaken to determine the incidence of lactose malabsorption in Mexican American children, to determine the effect of age on the incidence of lactose malabsorption, to explore the relationship between lactose malabsorption and milk intake, and lactose malabsorption and nutrient intakes, and to determine if growth is affected by lactose malabsorption.

REVIEW OF LITERATURE

Disaccharides comprise approximately 50% of the usual dietary carbohydrate intake in man and also are formed as the by-products of amylase digestion of polysaccharides (1). The primary dietary source of the disaccharide lactose is milk, each quart of milk containing approximately 50 g of lactose, and an 8 ounce glass of milk approximately 12 g (2).

Digestion of disaccharides, including lactose, occurs at the brush border in the small intestine of both man and animals (1). For lactose, the concentration of lactase in the intestinal mucosa is the rate-limiting step in digestion and absorption (1). For the other disaccharides hydrolysis is faster than absorption (3). The actual hydrolysis of the disaccharide occurs at the surface of the microvilli or just within its membrane since portions of hydrolysis products are released into the intestinal lumen (1). Wasserman and Belanger (4) demonstrated with radioactively labeled lactose that lactose accumulates in the striated border of the mucosa of the rat ileum. With a cytochemical technique, Miller and Crane (5) showed that hydrolysis of sucrose and maltose by hamster intestine rings occurs at a site from which the diffusion of monosaccharide products into the tissue occurs more rapidly than diffusion into the medium. They concluded that the site of hydrolysis was inside the intestinal mucosal cell, and later demonstrated that the major invertase and maltase activity are localized in the brush border region (6). Parsons and Prichard (7) proposed that the carrier system for the absorption of monosaccharides is closely related spatially to the disaccharidases, thus explaining the efficiency

of capture of hydrolysis products. Lactase is not present in the crypt cells, but appears in the brush border of differentiated intestinal epithelial cells (8).

Lactase activity in man and animals

Three intestinal disaccharidases exhibit lactase activity: brush border lactase, acid ß-galactosidase, and hetero-ß-galactosidase. Brush border lactase has a molecular weight of 280,000, pH optimum 6.0 (9), and appears to be the primary enzyme involved in hydrolysis of dietary lactose since there is a significant correlation between brush border lactase activity and the maximum rise in blood glucose after oral lactose (10). Neither acid ß-galactosidase or hetero-ß-galactosidase show a significant positive correlation with maximum blood glucose rise. Lysosomal acid ß-galactosidase hydrolyzes lactose and synthetic substrates and is relatively nonspecific. It is confined to the lysosomes and probably does not contribute to the digestion of dietary lactose *in vivo* (10). The molecular weights of its two species are 156,000 and 660,000 and the pH optimum 4.5 (9). Hetero-ß-galactosidase is present in soluble form in the cytoplasm and hydrolyzes several synthetic hetero-ß-galactoside substrates but not lactose (11), to any significant extent. Its molecular weight is 80,000 and its pH optimum is 6.0 (9).

There are no qualitative biochemical or physical differences among lactases isolated from the intestines of infants, lactose tolerant adults and intolerant adults. The major difference is one of quantity of enzyme (8). Cook *et al.* demonstrated in 12 Bantu (8), and in two groups of Zambian Africans (11,12) with low intestinal lactase activity that the mean acid ß-galactosidase and hetero-ß-glactosidase activities

were within the normal range in all, and brush border lactase activity exhibited a low residual activity in 24 of the Zambian subjects. Gray et al. (13) demonstrated the absence of brush border lactase in intestinal material obtained by biopsy from 5 lactase deficient patients; acid ß-galactosidase remained at normal levels.

Lactose intolerant patients do not lack absorptive capacity; they lack hydrolytic activity (14). In patients with normal lactase levels perfused with lactose, McMichael et al. (15) found the K_m to be 2.15 g/100 ml and the V_{max} to be 0.36 g/min./30 cm. Lactase deficient patients had absorption rates for lactose which were grossly reduced in comparison to normals and the amount of free glucose in the intestinal lumen never exceeded 1% of the total sugar concentration. In these patients glucose and galactose absorption were normal (15). The hydrolysis rate for lactose is significantly lower than for sucrose or maltose in normal adults, but in patients with an isolated lactase deficiency, lactose hydrolysis is depressed even more while sucrose and maltose are normal (16).

The developmental pattern and distribution of lactase is similar in most animals and man. In the rat, ß-galactosidase activity is detectable on the 18th day of gestation. Activity increases rapidly and attains a maximum towards the end of gestation or shortly after birth. Lactase activity then declines to a low plateau at about 4 weeks of age, and this low level of lactase is maintained throughout life (17,25,26). In the human, lactase and cellobiase, the ß-glycosidases, develop late in intrauterine life and reach maximum values at the end of the normal gestation period. In newborns lactase activity is of the same magnitude

as maltase, while in adults it is usually less (18). Premature infants have low lactase activity, but the level rapidly rises independent of the milk intake (18). It should be noted that the only carbohydrate present in significant quantity in human milk is lactose which contributes about 40% of the infant's ingested calories (19). This large lactose load probably exceeds the ability of the infant's small intestinal lactase to split lactose. Fermentation of non-digested lactose by the bacteria of the intestine would account for the normally low pH and high lactic acid concentration of stools of breast-fed infants (18).

In adults with normal lactase activity, lactase is low in the duodenum, reaches maximum activity in the jejunum or proximal ileum, and then rapidly decreases in the ileum (20). Lactase deficient adults have a similar distribution of lactase, but the amount of lactase is greatly reduced in comparison with normal adults (20).

Lactase activity, as stated, is at its maximum at birth, and remains high during infancy, falling to about one-tenth of this level following weaning. It appears that most humans have high levels of lactase in infancy and early childhood, and that these levels fall following the age of weaning, as happens in most other mammals. However, there are a few groups which maintain the infantile pattern of high lactase levels during adolescence and adulthood; this group is composed primarily of Caucasians of Northern European descent (21). Huang and Bayless (22) postulated two intestinal lactases: an infantile lactase and an adult lactase. The infantile form, present in infants and children at high levels, declines following weaning. In those persons who are lactose tolerant, an adult lactase takes the place of the infantile form; while

in those who are lactose intolerant, no adult lactase is present. This theory has been refuted by the work of Cook et al. (10,11) and Gray et al. (13) which show no physical differences among the lactases isolated from the intestine of lactose tolerant and intolerant subjects, only quantitative differences in the amount of enzyme.

Lactase activity, unlike the other disaccharidases, does not appear to be markedly influenced by diet in either animals or man (23), but this point remains controversial (24). Alvarez and Sas (25) demonstrated that adult rats fed a diet rich in lactose showed no induction of lactase, and that maintaining rat pups on a lactose-rich diet did not prevent the decrease in lactase at 21 to 22 days of life. Koldovsky and Chytil (26) demonstrated that feeding rats a diet rich in lactose from day 14 postnatally in the presence of the mother rat slowed down the decrease in β-galactosidase activity, but did not prevent it. While the β-galactosidase activity is elevated in suckling animals and decreases at about the time of weaning, the β-glucosidases (maltase, isomaltase, invertase, trehalase, and palatinase) are virtually absent in the suckling phase and increase in activity during weaning in the rat, dog, cow, rabbit, mouse and pig (27). Adult male rats fed either a sucrose or a casein diet ad libitum show no differences in lactase activity, although sucrase and maltase activities are increased over fasting levels (28). Bolin et al. (29) fed adult female rats 30% lactose and 30% glucose diets. After 5 to 8 weeks, the rats on both diets showed increased jejunal lactase activity. Caine et al. (30) fed weanling rats a diet containing increasing amounts of lactose. On 5% lactose the rats developed diarrhea, on 30% lactose blood rings were present around the eyes, on 50% lactose

they began losing weight. However, the group receiving lactose showed significantly higher lactase and cellobiase than a control group. Rats fed either an 8% dextrose or 8% lactose diet from weaning fail to reveal any significant differences in lactase, sucrase or maltase activity, regardless of whether the activities are expressed in terms of whole intestine, per g of intestine or per 100 g of body weight (31). Rats fed either a 30% lactose diet or a 15% glucose and 15% galactose diet from one day old exhibited high jejunal lactase activity up to between weeks 3 and 4 of life at which time lactase activity decreased to a low level and remained close to this value in both groups. There were no significant differences in lactase, sucrase or maltase activities between the groups (32).

Studies in nonhuman primates show similar results to those in rats and other animals. Lactase activity in adult bonnet, owl and rhesus monkeys is low, ranging from 0.07 to 0.74 units/g wet weight mucosa (33). The distribution of lactase in the adult Olive baboon is similar to the rat: low activity is present in duodenum and ileum, peak activity is present in the jejunum. The drop in lactase activity at weaning also occurs in the Olive baboon and does not appear to be just a response to lack of lactose in the diet, but rather an age-related phenomenon (34). The feeding of a diet containing 20% lactose to lactose intolerant galago monkeys for 4 months increased their lactose tolerance index and increased intestinal lactase activity, but had little effect on other disaccharidases (35).

Lactose deprivation in normal human subjects does not lower lactase levels. Knudsen et al. (36) demonstrated in 26 obese subjects fasted

for up to 42 days that complete lactose deprivation does not significantly change lactase activity or lactose tolerance. In a further study, 7 healthy male Caucasian volunteers consumed two lactose-free synthetic diets for 42 days, during which time intestinal lactase decreased in three subjects, increased in two and remained unchanged in one. Repeated lactose tolerance tests during the period revealed no clinical intolerance except in one subject who was already lactose intolerant before entering the study. Since the intestinal epithelial cell renewal rate is 2 to 3 days, during the diet period at least 10 generations of cells were exposed to a lactose-free environment (37). Knudsen concludes that dietary lactose is not an important factor in determining the level of intestinal lactase in humans (36). Rosensweig and Herman (38) fasted obese patients for 14 to 28 days and noted that the fall in sucrase and maltase activities was much greater than the fall in lactase which fell only slightly. However, Cuatrecasas et al. (39), using a lactose absorption ratio, found that after total abstinence from lactose for 5 months the lactose absorption ratio in two patients fell from 92.0% to 71.1% and 97.4% to 58.1%.

Feeding of lactose does not appear to raise lactase levels. Knudsen et al. (36) fed lactose to two obese patients after a 14-day fast, and noted that there was no selective increase in lactase activity, rather there was a general increase in enzyme activity and protein concentration in the mucosa of the jejunum. Rosensweig and Herman (38) demonstrated that after a 14-28 day fast, patients fed a 2000 calorie carbohydrate-free diet for one week showed no increase in disaccharidase activity. However, when fed a 2000 calorie 84% glucose diet for one more week, sucrase

and maltase activities increased, but lactase showed no change. Rosensweig (40) fed normal males a diet containing the lactose equivalent of 8 to 10 quarts of milk per day and did not increase lactase activity above the level seen with a baseline glucose diet. Lactose was fed for 10 to 14 days without effect, while in parallel sucrose and fructose-feeding studies an increase in sucrase and maltase activities occurred within 2 to 5 days. This time lag suggests that the effect of dietary sugars on disaccharidases occurs at the crypt cell level and is manifested as these cells mature and migrate from the crypt to the villus tip. Feeding of 50 g of lactose per day to 50 healthy young adult Thais for 4 weeks did not alter the tolerance test or enzyme assay for lactase, suggesting that in the Thai population lactase deficiency is not the result of adaptation to a milk-free diet (41). Reddy and Pershad (42) fed 6 Indian adults 60 g of reconstituted skim milk powder (30 g of lactose) daily for 4 weeks. There was no change in jejunal lactase activity, but the protein content of the mucosa increased and severity of symptoms decreased. Cuatrecasas et al. (39) failed to induce lactase activity in 11 subjects fed 150 g of lactose daily for up to 3 months. The 7 nonabsorbers had considerable diarrhea for 7 to 14 days after which it disappeared, the 4 tolerant subjects had no symptoms and lactase activity was unchanged in both groups.

Clinical conditions exhibiting low lactase activity

Of all enzyme deficiencies, probably the most common is lactase deficiency in adults (43). Lactase deficiency arises from several conditions, and consequently there are several classifications for lactase

deficiencies: congenital lactase deficiency, low lactase activity of secondary origin, and low lactase activity of primary origin.

Congenital lactase deficiency with no lactase activity was first described by Holzel et al. (44), and is due to the absence of intestinal lactase at birth, the defect lasting throughout life. Diarrhea is the main symptom, appearing as soon as milk feeding has been well established, and is accompanied by failure to gain weight in spite of adequate food intake, flatulence, and colic. Removal of milk from the diet, and replacement by lactose-free formulas or other suitable foods results in immediate subsiding of symptoms and weight gain. Congenital lactase deficiency is believed to be hereditary and is relatively uncommon (21).

Congenital lactase deficiency should not be confused with the mild, transient lactase deficiency observed in premature infants (45). In studies on premature and term infants, Boellner et al. (19) found that lactose metabolism may be impaired significantly during the first three days of life in both uncomplicated premature and term infants. This impairment in hydrolysis of lactose may be the result of delayed maturation of the lactase enzyme system or from an anatomical or functional alteration of the intestinal epithelial cells at birth. However, lactase, sucrase and maltase activities reach normal infantile levels by 14 days of age in premature infants regardless of whether they are fed a lactose-containing formula or a sucrose formula (46).

Secondary lactose intolerance is frequently seen in infants with unexplained diarrhea. Removal of lactose from the diet results in rapid cessation of diarrhea and rapid weight gain (47,48). Examination of such patients several months after the diarrhea episode reveals normal

tolerance to lactose (47).

Reduced levels of intestinal lactase are frequently found secondary to mucosal damage associated with some disease states. Since lactase is located in the brush border of the villi, any anatomical or functional damage to the microvilli may result in depression of disaccharidase activity (36,49). Lactase activity is more depressed by mucosal damage than are the other disaccharidases (50), and lactase activity may remain low for many years following recovery from the original insult (51,52). Secondary lactase deficiency can be the result of inflammatory and degenerative diseases involving the small intestinal mucosa such as tropical sprue (51-54), gluten sensitive enteropathy (55,56), protein-calorie malnutrition (57-61), and following radical gastrointestinal surgery (62-64). Lactose intolerance may be associated with ulcerative colitis (65, 66), "irritable-colon syndrome" (67) and may accompany parasitic infestations (54,68). When the cause of the mucosal damage responds to treatment lactase activity usually improves slowly, but may remain abnormally low for several years after the other disaccharidases have returned to normal levels.

Low lactase activity of primary origin occurs on a world-wide basis, lactase levels being normal during infancy and early childhood, but falling during childhood and adolescence. By definition low lactase activity of primary origin is neither congenital nor secondary to mucosal damage (21).

Primary low lactase activity--definitions and diagnostic criteria

The procedure most commonly used to evaluate intestinal lactase activity in field studies is the standard lactose tolerance test. The

results of this test, however cannot be used as indicators of milk intolerance. Low lactase activity, lactose intolerance and milk intolerance are not synonymous.

Low lactase activity is defined as less than 2 units of lactase activity per g wet mucosa as demonstrated in either of two ways: directly by examination of intestinal biopsy material, or indirectly by the lactose tolerance test. In the second case, a rise of 25 mg percent or less following ingestion of a standard lactose dose of 50 g per square meter of body surface or 2 g per kg of body weight in children correlates well with intestinal lactase activity less than 2 units per g wet mucosa (69).

Lactose malabsorption is the reduced absorption of lactose as a consequence of low lactase activity as determined by the lactose tolerance test described above (21).

Lactose intolerance is defined as the clinical signs following ingestion of lactose mixed in water in a standard dose or less, in a person with proven lactose malabsorption (69).

Milk intolerance as defined here results from the lactose content of milk or milk products. Clinical signs consistently follow within a few hours of ingesting milk or milk-containing foods in usual amounts in a person with proven lactose malabsorption (69).

In the view of the Protein Advisory Group, intolerance to lactose in the large amounts commonly used in load tests (the equivalent of one quart of milk) gives no information on the existence of milk intolerance when milk is consumed in moderate quantities. The load tests measure the pressure of enzyme activity and not the capacity of the individual

to tolerate the quantity of milk which he normally consumes (69). However, Bedine and Bayless (70) have demonstrated that 15 of 20 lactose intolerant subjects became symptomatic with the amount of lactose found in 8 ounces of milk (12 grams of lactose). Ten of the 15 gave a history of milk intolerance. Based on this study, Bedine and Bayless estimate that at least 50% of lactose intolerant persons would be symptomatic after ingesting 8 ounces of milk.

Bayless et al. (71) use the following guidelines in evaluating the results of lactose tolerance tests in milk intolerant persons free from diabetes. If no symptoms are produced after a lactose load and blood sugar rises above 25 mg per 100 ml, then milk intolerance on the basis of lactose intolerance is unlikely. If symptoms are produced with the lactose tolerance test but not with a glucose-galactose tolerance test, and the peak blood sugar rise after lactose is less than 25 mg per 100 ml, one can be confident that the subject is milk intolerant, and lactase levels are usually found to be less than 2 units per g wet weight of mucosa. If symptoms are produced with lactose but not with glucose and galactose and the peak blood sugar rise is greater than 25 mg per 100 ml, the subject is probably milk intolerant and jejunal lactase levels are usually over 2 units. These persons comprise about 20% of milk intolerant persons, and they may also have other food intolerances. Finally, if no symptoms are produced by lactose but the blood sugar rise is less than 25 mg per 100 ml, the subject is probably not milk intolerant on a lactose maldigestion basis, and also not lactase deficient. Delayed gastric emptying caused by hypertonic doses of lactose or missing an early peak in blood sugar are possible explanations for this (72).

In a clinical situation, more rigorous criteria for diagnosing lactose intolerance are employed (73,74). These criteria include demonstration of:

1. diarrhea upon ingestion of lactose,
2. intestinal lactase activity less than 1 unit per g wet weight mucosa (biopsy from the area of the ligament of Treitz),
3. flat lactose tolerance test curve,
4. normal tolerance curves for glucose and galactose, and
5. disappearance of diarrhea upon removal of lactose from the diet.

The definitive test for primary low lactase activity is the actual measurement of enzyme activity in a specimen of mucosa obtained by intestinal biopsy. This technique is too difficult to be used in field work, and therefore the lactose tolerance test administered under specific conditions is used in field studies. Lactose loading followed by the measurement of blood glucose at 15, 30, and 60 minutes can be used to make a fairly accurate diagnosis of low lactase activity. A blood glucose rise of 25 mg per 100 ml or less after an oral lactose load correlates well with intestinal lactase activity below 2 units per g wet weight of mucosa (67,81,88,91,118).

Other tests have been recommended for determining lactose tolerance including breath analysis for H_2 or $^{14}CO_2$ after lactose loading (75,76), lactose loading with recording of symptoms only (77), and radiological diagnosis with lactose-barium meal (78).

Clinical signs and symptoms of lactose intolerance

The symptoms of primary, secondary, and congenital lactase deficiency are similar, although the congenital variety may be more severe

(21). The symptoms include any or all of the following: mild to moderate cramps, nausea, bloating, flatulence, borborygmi, one or more loose stools, and diarrhea. Symptoms may begin as early as 15 minutes after the lactose is consumed and may last up to 24 hours (79), and it is not unusual for symptoms to begin six to eight hours after ingestion of lactose (71). It should be noted that not all persons with low lactase levels have symptoms following milk ingestion or lactose loading in a lactose tolerance test, and some with normal lactase levels will develop symptoms after lactose loading (80,81). Sheehy and Anderson (80) estimate that one of every 6 persons with low lactase levels does not show intolerance to lactose.

The mechanism by which these symptoms are produced is not fully understood, but it appears that the unhydrolyzed nonabsorbable lactose remaining in the intestine causes an increase in the osmotic pressure of the gut contents. This in turn causes a net movement of water from the tissues into the gut lumen. The accumulated water causes distension of the bowel and provokes increased intestinal motility. When the undigested lactose reaches the colon it is fermented by colonic bacteria, which produce organic acids such as lactic acid and acetic acid. These acids act as irritants, causing increased peristalsis and resulting in diarrhea. Bacterial action on lactose in the colon could also produce gases and explain the frothy character of the diarrhea. Kern and Struthers (82) perfused human patients with isosmolar solutions of lactose or sucrose. Those with normal lactase and sucrase activities absorbed both sugars but sucrose was absorbed more completely. Patients with lactase deficiency absorbed sucrose normally and lactose poorly.

With lactose infusion, the lactose intolerant patients lost water into the jejunum at the rate of 5 ml per minute, showed no rise in blood glucose level, and experienced abdominal cramps, nausea and diarrhea. Laws and Neale (78) demonstrated that as little as 6 g of lactose added to a barium meal prior to X-ray examination of the bowel in patients with low jejunal lactase produced observable dilution and increased rate of transit in the small intestine. The small intestine was distended by dilute contrast medium, and active peristalsis was evident. By one hour following ingestion of the lactose, the contrast medium was usually in the transverse or descending colon. Using an intraluminal intubation technique, Christopher and Bayless (83) found that in lactase deficient subjects a test solution of lactose is diluted 5-fold in the proximal jejunum and ileum as compared to normals. The dilution observed with lactose was equivalent to the net secretory response of the small bowel to an osmotic load, and the resulting diarrhea was enhanced by interference with net fluid absorption in the colon. Rousseau and Sladen (84) have demonstrated in rats that a reduction of luminal pH inhibits the net absorption of water, Na^+, and Cl^- from rat ileum and colon. Such a mechanism may contribute to the interference with water reabsorption in the colon following lactose ingestion in lactose intolerant people.

Prevalence of primary low lactase activity

Carbohydrate intolerance was first described by Schmidt and Strasburger in 1901, and in 1935 Althausen *et al.* (85) published a study on 60 patients with carbohydrate intolerances. They recognized carbohydrate intolerance as a common condition and that milk was among the

most offending articles of food. Few references to lactose intolerance are found until 1963 when Auricchio et al. (86) described isolated lactase deficiency in 3 hospital patients. These were the first cases in which assays of intestinal enzyme activity were used to diagnose an isolated lactase deficiency. In the same year Dahlqvist et al. (87) demonstrated that lactose intolerance can be a cause of milk intolerance, and that impaired digestion and absorption of lactose was caused by reduced lactase activity in the intestinal mucosa of patients with lactose intolerance. They also demonstrated the usefulness of intestinal mucosa biopsy material in quantitative assay of disaccharidase activity. Since the populations these authors were investigating were northern European, it is not surprising that isolated lactase deficiency was considered a rare occurrence, even though thirty years earlier carbohydrate intolerance was considered a common occurrence. By 1965, however, Haemmerli et al. (88) recognized that lactose malabsorption in the adult was not a rare condition.

Many studies have been conducted on the prevalence of lactose intolerance in different parts of the world. These studies have usually been confined to adults, and there is not much information on the prevalence of lactose intolerance in children (21).

Sahi et al. (89) studied 130 Finnish children between the ages of 7 and 15 years. Fourteen children had a maximum blood glucose rise of less than 20 mg per 100 ml after lactose loading, and 8 of these also had low lactase activity as demonstrated by enzyme assay or a lactose tolerance test with ethanol. The prevalence of lactose malabsorption in boys and girls between 7 and 11 years was 4%, and in boys and girls 12 to 15

years 9%. Sahi et al. noted that children with lactose malabsorption are more often symptomless than adults probably because the colon of healthy children and young adults does not easily react with increased peristalsis to increased intraintestinal volume of fluid caused by unabsorbed lactose. Jussila (90) studied milk intolerance and lactose malabsorption in hospital patients and Finnish servicemen. The incidence of lactose malabsorption among 504 patients was 17%, and among 134 servicemen 18%. There was no significant correlation between lactose malabsorption and age nor lactose malabsorption and sex of the subjects.

In Switzerland, Haemmerli et al. (88) examined 24 adults: 17 normal controls, 4 with acquired milk intolerance, and 3 with idiopathic sprue. The mean blood glucose rise was 35.1 mg per 100 ml for the normals, 6.2 mg per 100 ml for the milk intolerants, and 9.0 mg per 100 ml for those with sprue. Those who were milk intolerant showed a decreased stool pH (5.3-6.0), and the presence of lactose, glucose and lactic acid in the stool.

The incidence of lactose intolerance varies in different segments of the Greek population. McMichael et al. (91) found 15 of 17 Greek Cypriots to have a blood glucose rise less than 20 mg per 100 ml following a lactose load. Spanidou and Petrakis (92) examined 16 Greek non-Cypriot students and compared them to 13 U.S. Americans of northern European background. Six of the 16 Greeks (38%) and none of the Americans were found to be intolerant to a lactose load of 50 g. Doxiadis and Papageorgiadis (93) studied 50 normal Greek infants and 24 normal Greek children between the ages of 7 and 13 years. Thirteen of the 50 infants and 13 of 24 children had a blood glucose rise less than 20 mg per 100 ml.

None of the infants had symptoms, and 6 of 13 children with flat curves developed abdominal pain, vomiting or diarrhea. Doxiadis and Papageorgiadis concluded that in the Greek population, lactase deficiency starts early in life, but the clinical symptoms arise later in childhood. Kattamis et al. (94) examined 82 healthy Greek children between the ages of 1 and 13 years with no clinical symptoms on ingestion of normal quantities of milk (200-500 ml daily). Fifty-five of these children, or 67% had a blood glucose rise of less than 20 mg per 100 ml after a lactose load of 2 g per kg of body weight. All but 8 of these had symptoms, while none of the 27 tolerant children had symptoms. Kattamis et al. concluded that milk intolerance is uncommon in Greek children and there is no correlation between milk intolerance and the results of the lactose tolerance test. Although the lactose tolerance test is abnormal in a high proportion of Greek children they have no symptoms after drinking milk. Zografos et al. (95) examined 250 Greek adults between the ages of 15 and 78 years, 24 of whom had spastic or "irritable colon syndrome." After a lactose load of 50 g, 58 (23%) showed a blood glucose rise of less than 20 mg per 100 ml. Forty-five with a flat curve and 18 normals reported symptoms after ingestion of milk, and all with a flat curve and 22 normals reported symptoms during the test. Of the 24 with irritable colon syndrome, 17 (71%) had a flat lactose tolerance curve.

In Africa the incidence of lactose intolerance has been studied in tribes in Uganda, Nigeria, and South Africa (96-102). Cooke and Kajubi (96) studied 135 healthy volunteers and patients in Uganda. The 20 healthy volunteers were from the areas of Baganda, Rwanda and Ankole. Ninety percent of them had a blood glucose rise of less than 20 mg per

100 ml after a lactose load of 50 g, and 16 (80%) had symptoms. A total of 95 adult patients were studied. Of the 35 Bantu, 31 (88%) had low lactase activity and 9 (26%) were lactose intolerant; of 12 Rwanda (Batutsi) 2 (17%) had low lactase activity and one was lactose intolerant; of 15 Rwanda (Bahutu) 5 (33%) had low lactase activity and none had symptoms; of 11 Ankole (Bahima) one had symptoms and none were intolerant; of 13 Ankole (Bairu) 5 (38%) had low lactase activity and one was lactose intolerant; and of 9 Nilotic 4 (44%) had low lactase activity and 2 (22%) were intolerant. Of the twenty children included in the study, the 16 Baganda (Bantu) had an incidence of 81% low lactase activity and 19% lactose intolerance, and 1 of a group of four Ankole and Nilotic children had low lactase activity. Cooke and Kajubi also performed jejunal biopsies on 54 of the adult patients. Eleven of these had grossly abnormal mucosa and were excluded, while 43 were comparatively normal with well-formed leaves, finger villi and short ridges. Lactase levels were below 2.5 units/g tissue wet weight in 24 Bantu, 1 Batutsi, 1 Bahutu, and 4 Nilotic. In all cases, those with low lactase levels as demonstrated by assay of jejunal biopsy were intolerant to an oral lactose load. In a further study of 72 infants and children of the Baganda tribe, Cook (97) found that most Baganda become lactase deficient between the ages of 3 and 4 years, and some have low lactase levels as early as 6 months. The mean rise in blood glucose after a lactose load of 2 g per kg body weight in newborns is 44.3 mg per 100 ml, and this increases during the first week to 63.1 mg per 100 ml. The mean blood glucose rise decreases with increasing age so that by 47 months 60% of the Baganda children had low lactase levels and by 60 months

all the children examined had low lactase levels. Assessment of symptoms following the lactose load was difficult because of poor reporting. Cook concluded that the fall in lactase levels with age is not due to the absence of milk from the Baganda diet, because some infants with flat curves were still breast fed, and half of the infants from a children's home who received 140 ml of milk per kg of body weight per day also had flat curves. Kretchmer et al. (98) examined several Nigerian ethnic groups and 11 Caucasians (including 3 Semites). The Yoruba do not raise cattle and consume no milk after weaning, and of the 41 Yoruba examined, 40 or 99% had lactose malabsorption. The Hausa and Fulani are cattle raisers and milk is eaten as whole milk and as nono, a type of yogurt. Nine "genetically pure" nomadic Fulani had an incidence of 22% lactose malabsorption, while 24 town Fulani who were inbred with Hausa had an incidence of 70% lactose malabsorption. Thirteen of the seventeen Hausa (76%) examined had lactose malabsorption. Of the 11 Caucasians included in the study, 4 (36%) had lactose malabsorption, including all 3 Semites. Kretchmer et al. (98) found a highly significant difference in the ability to tolerate lactose between Nigerian infants up to the age of one month and those aged 2 to 4 years and adults greater than 20 years. Olatunbosun and Adadevoh (99,100) examined 83 hospital patients all of whom were free from diabetes or gastrointestinal diseases. They included 48 Yoruba, 15 Hausa and Fulani, 11 Ibo, and 9 from other ethnic groups. Forty of the Yoruba (84%) were lactase deficient, 9 Hausa and Fulani (60%) were lactase deficient, 9 Ibo (82%) were lactase deficient, and all of the 9 from other groups were lactase deficient, for an overall prevalence of lactase deficiency of 81% in Nigerians. In a further study in Yoruban children,

Olatunbosun and Adadevoh (101) found that 79% of the weaned children were lactase deficient, while none of 28 unweaned children were lactase deficient. Of 23 children with protein calorie malnutrition, 7 (30%) were lactose intolerant. Jersky and Kinsley (102) found that 90% of 38 South African Bantus had flat curves during a lactose tolerance test, but none reported any abdominal symptoms or diarrhea during the test. Most of these subjects drank small amounts of milk every day usually with tea or coffee or with porridge (102).

Ethiopian children have a mean frequency of lactose malabsorption of 80% (103). The incidence is lowest in children under 1 year (60%), and increases with age to 90%.

In the Near East, Gilat et al. (104-106) have studied lactase deficiency in various Jewish and Arab communities. The incidence of lactose intolerance varied from group to group. Among Yemenite Jews it was 44.4%, Iraqi Jews 84.2%, Oriental Jews 85.0%, Ashkenazi 79.2%, Sephardi 62.5%, for an overall incidence in the Israeli population of 71.1%. Lactase deficiency appears in the Jewish population between the ages of 6 and 8 years. Among Palestinian Arabs, the incidence of lactase deficiency is 81%, and lactose intolerance 57%, with a greater incidence of lactase deficiency in females (87.5%) than in males (70.4%). Tandon et al. (63) speculated on the high incidence of lactase deficiency found in Jewish patients with ulcerative colitis, and concluded that segregation of the genes for ulcerative colitis as well as for lactase deficiency in Jews is a possible reason for the high incidence of lactase deficiency found in patients with ulcerative colitis. Rotthauwe et al. (107) gave lactose tolerance tests to 26 subjects from Syria, Jordan, Libya, Saudi-Arabia,

Egypt, Iraq and Tunesia, and 21 had a maximum blood glucose rise of less than 20 mg per 100 ml. Symptoms occurred in 20, and in order of frequency were borborygmi, flatulence and diarrhea.

Asian populations have also shown a high incidence of lactase deficiency (41,42,79,108-114). In 12 normal Indian adults analysis of intestinal biopsies revealed that all had low lactase levels but normal sucrase and maltase, and of 18 given a lactose tolerance test, 11 had flat curves and 9 (50%) had symptoms, while a glucose-galactose tolerance test was normal in all. Reddy and Pershad (42) performed a jejunal biopsy in 15 normal Indian children and a lactose tolerance test in 54 healthy children. Lactase activity decreased with age, and 20 of the 54 children were lactose intolerant. When the same lactose load (2 g per kg body weight) was administered as skim milk, only 4 children had intestinal symptoms and these had no symptoms when the dose was divided in half. Intestinal motility was normal when these children were fed a lactose load as skim milk, but was rapid when the load was lactose in water.

Keusch et al. (108) and Flatz et al. (109) also found a decrease in lactose tolerance with increasing age in Thai children. Institutionalized Thai children less than one year old had an incidence of lactose malabsorption of 40.8%; those 1 to 2 years 75%, and over 2 years 86.7%. In contrast, Thai village children in the same age groups had incidences of lactose malabsorption of 14.3%, 30.0% and 87.5%, respectively. All the children had normal maltase and sucrase activities. Since neither group had overt PCM, gluten-sensitive enteropathy or tropical sprue, Keusch et al. attribute the higher incidence of lactose malabsorption

in the institutionalized children below the age of 2 to recurrent episodes of acute enteritis leading to selective lactase deficiency. Keusch et al. (41) and Flatz et al. (109) have found the incidence of lactose intolerance to be 97% in healthy adult Thais.

In Singapore, Bolin and co-workers (110) found the incidence of lactose intolerance to be 94%. The population studied consisted of 73 Chinese, 15 Malays, and 10 Indians between the ages of 1 and 42 years who were hospitalized for reasons other than gastrointestinal disease. Diarrhea invariably occurred after a lactose load in those over 9 years, but only occurred 50% of the time in those under 9 years. The incidence of lactose intolerance increased with age, being 36% in the 1 to 3 year age group, 50% in the 5 to 7 year age group, and 100% in those over 15 years. The mean jejunal lactase activity decreased from 22.0 units per gram of protein at 1 to 3 years to 1.5 units per gram of protein for those over 15 years. Seventy-five percent of the lactose tolerant patients consumed at least a pint of milk a day, while only 39% of the lactose intolerant patients usually consumed this amount.

Several studies have been conducted on Orientals living in the United States (75,79,111). Huang and Bayless (111) tested 20 healthy physicians and medical personnel of whom 7 were natives of Taiwan, 3 were born in the U.S. to Chinese parents, and 10 were Filipinos. Twenty healthy Caucasians served as controls. Fourteen of the 20 Orientals reported that 1 to 2 glasses of milk or one serving of ice cream produced symptoms of lactose intolerance, and 13 of the 20 had a parent or sibling with a similar intolerance. In the Caucasian group, only 2 were milk intolerant. A lactose tolerance test produced symptoms in 19 of the 20

Orientals and in 2 of the Caucasians. Calloway et al. (75) used breath analysis to determine lactose tolerance in 5 native-born Chinese men. The subjects were given 48 g of lactose as pasteurized fluid whole milk, Swiss cheese, or lactose in water. Expired air was collected at 30-minute intervals for 5 hours. Breath hydrogen levels increased from 10 - 20 ppm to 230 ppm within 2 hours of ingestion of lactose in any form. This elevation was sustained for 1 to 2 hours after which it declined slowly. Breath hydrogen levels were slightly lower with milk than with lactose, and when the dose of lactose as milk was halved, the hydrogen values were also reduced about a half. Chung and McGill (79) performed jejunal biopsies and lactose tolerance tests on 11 Orientals (2 Japanese, 4 Korean, 5 Chinese) residing in the U.S. Biopsy samples revealed lactase activity of 0.3 and 0.4 units per gram wet weight mucosa in 2 of the subjects and essentially no activity in the other 9. Maltase and sucrase activities were normal in all 11, and the mucosal tissue appeared normal histologically. A lactose tolerance test produced loose stools within 4 hours in 9 subjects and mild to moderate cramps and gas in 10. One subject had no symptoms. Eight of the 11 drank between 1 and 3 glasses of milk per day (79).

Bolin et al. (112,113) examined groups of Southeast Asian students residing in Australia. In the first study, of 21 Chinese students 86% had a flat curve in the lactose tolerance test and all had diarrhea, 8 of 8 New Guinea natives had flat curves, and 4 of 5 Indian students had flat curves. In the second study, 15 Chinese students were given 80 g of lactose, and 14 of these had symptoms and a flat curve. Also included in the study were 5 Indians, 8 New Guinea natives and 23 Caucasians. Four

of the Indians had flat curves while none of the New Guinea natives and none of the Caucasians did. Jejunal biopsies in 6 of the Caucasian students revealed a mean jejunal lactase activity of 3.9 units per g wet weight mucosa, and in 7 of the Chinese students 0.4 units per g wet weight.

In Australian Aboriginal children between the ages of 0 to 15 years, Elliott et al. (114) found a high level of lactose malabsorption. In the Papunya tribe, 61% of the children had a rise in blood glucose of less than 10 mg per 100 ml, 29% had a rise of 10 - 20 mg per 100 ml, 5% had a rise of 20 - 30 mg per 100 ml, and 5% had a rise over 25 mg per 100 ml. In the Maningrida tribe 48% of the children had a rise in blood glucose of less than 10 mg per 100 ml, 21% had a rise of 10 - 20 mg per 100 ml, 5% had a rise between 20 and 30 mg per 100 ml, and 16% had a rise over 30 mg per 100 ml. The incidence of lactose maldigestion was related to age and inversely related to weight (114). In contrast, Bolin et al. (115) found the incidence of lactose intolerance to be 6% in a sample of 100 healthy Caucasian adults in Australia.

Prevalence of primary low lactase activity in the Western Hemisphere

In the Western Hemisphere, several studies have been performed on different ethnic and racial groups. As in Europe, the incidence of lactose malabsorption is low among Caucasians. Lactose malabsorption is common among Indians and Negroes, and is also common among Mulattoes and Mestizos.

The Caucasians of North America resemble the Northern Europeans in the incidence of lactose malabsorption. Cuatrecasas et al. (39) examined

19 adult Caucasian hospital patients (5 women and 14 men) for lactose tolerance and calculated the lactose absorption ratio for each. None of the women and 3 of the men were lactose nonabsorbers (absorption ratio less than 50%). There appeared to be no correlation between age and absorption. After an oral lactose load, lactose nonabsorbers had a more rapid intestinal transit time, an increase in stool lactose, and a decrease in stool pH.

Leichter (116) studied 38 healthy adults of Slavic origin residing in Canada. Six of 21 Poles (28.5%) and 3 of 17 Czechs (17.6%) were lactose intolerant on the basis of an oral lactose tolerance test.

Huang and Bayless (22) found that of 20 white children aged 11 months to 11 years, only 2 (10%) developed abdominal discomfort and bloating after an oral lactose load. However, 7 of 20 Negro children (35%) exhibited symptoms of lactose intolerance. All had consumed milk without ill effects during infancy and continued to consume 1 to 3 glasses of milk each day, although one child did report cramping after consuming more than one glass of milk.

Katz (117) found lactose intolerance in soldiers returning from Vietnam. Since fresh milk was unobtainable in Vietnam, the servicemen on returning home consumed large amounts of milk and milk products and some were troubled with gaseousness and diarrhea.

Welsh (118) compared data on 100 subjects with isolated lactase deficiency (ILD) to 150 control subjects with normal lactase activity (NLA). Fifty-five were healthy, but most of the subjects in both groups had various gastrointestinal and non-gastrointestinal conditions, including diabetes. The sample included 169 men and 81 women ranging from

2 to 80 years of age; the racial composition was 209 white, 39 Negro, and 2 American Indian. Of the 100 subjects with an ILD, 73 were men and 27 were women. The racial composition of the group with ILD was 67 whites (32%), 32 Negroes (82%), and one American Indian (50%). In an earlier study Welsh had found the incidence of ILD to be 19% in whites, 77% in Negroes, and 67% in American Indians. Fifteen of these subjects reported no history of milk intolerance, while the remaining 32 had histories of diarrhea after milk ingestion.

Paige et al. (120) compared 32 white and 32 Negro pregnant women as to the incidence of lactose intolerance. Of the 32 Negro women, 21 (66%) showed lactose malabsorption and 17 had symptoms; of the 32 white women, 3 (9%) had lactose malabsorption with 2 of the 3 exhibiting symptoms.

In Baltimore public schools, Paige et al. (121) found the incidence of lactose malabsorption to be 17% among white children who drank less than one quarter of the milk included in their school lunch (nonmilk drinkers), and 20% among white children who drank more than three quarters of the milk served with school lunch (milk drinkers). Among the Negro children at the same schools and of similar socioeconomic background, the incidence of lactose intolerance was 76.5% in the nonmilk drinkers and 35.7% in the milk drinking group. Paige et al. concluded that in white children lactose intolerance does not appear to be the main factor in milk rejection. However, in Negro children there was a definite association between poor milk consumption, lactose-induced symptoms and lactose malabsorption.

In Surinam, school children often suffer diarrhea after consuming school milk, and young children fed a milk diet have inexplicable cases of diarrhea. Luyken et al. (122) found the mean blood glucose rise after lactose loading to be 3 mg per 100 ml in Surinam Bushnegroes, 14 mg per 100 ml in urban Creoles, 12 mg per 100 ml in Creole soldiers, 16 mg per 100 ml in Creole children, and 12 mg per 100 ml in Hindustani children. In contrast, Dutch soldiers drank large quantities of milk and had a mean blood glucose rise after lactose loading of 33 mg per 100 ml. The authors feel that lactose intolerance may explain the local taboo against milk, the complaints after milk ingestion, and the frequent occurrence of diarrhea.

In a group of 94 rural Jamaican children aged 2 months to 4 years, Stoopler et al. (123) found 53 (56%) to have a blood glucose rise less than 26 mg per 100 ml during a lactose tolerance test. There was a significant increase in the percentage of lactose malabsorbers after the first year of life, but the capacity to absorb lactose did not correlate with sex, nutritional status, milk consumption, or duration of breast feeding. Nine (17%) of the 53 malabsorbers were still being breast fed. The parents of 34% of the malabsorbers and 32% of the absorbers reported abdominal pains, vomiting or diarrhea in their child after the lactose tolerance test. Six to 7 months after the initial test, 52 of 53 lactose malabsorbers were retested. The blood glucose response remained low in 41 (79%) children, but 11 (21%) had a normal response. Stoopler et al. also tested a group of 20 urban Jamaican children, 14 (70%) of whom had a flat lactose tolerance curve. As in the rural children, lactose tolerance did not correlate with sex, nutritional status, milk consumption,

or duration of breast feeding. In the rural children, lactose absorption was inversely correlated with age, but no correlation with age was present in the urban children. The pattern of lactose malabsorption was similar in urban and rural children in spite of different environmental conditions. Lactose malabsorption occurred even in the presence of successful breast feeding. The reversibility of lactose tolerance in the rural children suggests that transient environmental insults may temporarily suppress intestinal lactase activity.

Bose and Welsh (124) investigated lactose malabsorption in Oklahoma Indians. None of the 5 children examined (aged 3-22 months) showed evidence of lactose malabsorption; however, 29 of 36 adults (80%) had lactose malabsorption. Two-thirds of the nonabsorbers were full-blooded Indians and the remainder were at least 75% American Indian. Ten of the 24 adults who were symptomatic had previously recognized their intolerance to milk.

Among Canadian West Coast Indians aged 14-24 years, Leichter (125) found 63.3% had a blood sugar rise less than 20 mg per 100 ml, and 68.4% of these exhibited symptoms of lactose intolerance. None of the persons tested associated milk consumption with gastrointestinal complaints, but milk intake is low in this population.

Duncan and Scott (126) using a lactose tolerance test with ethanol in which the lower limit suggested for tolerance is 10 mg galactose per 100 ml blood, found all 36 Alaskan Indians and Eskimos tested to have blood galactose rises of less than 10 mg per 100 ml. Thirty of the 36 had severe diarrhea following the lactose loading for an incidence of lactose intolerance of over 80%. However, Bell et al. (127) estimate

the incidence of lactose intolerance to fall between 56 and 70% in northern Alaskan Eskimos. Greenland Eskimos have an incidence of lactose malabsorption of 72%, and if those with known Danish ancestors are excluded, the incidence of lactose malabsorption increases to 88% (128).

In Chami (Colombian) Indians, Alzate et al. (129) found none of the 24 adults tested to have a blood glucose rise over 16 mg per 100 ml, but only 14 (58.3%) had symptoms after a standard lactose load. The authors attribute the low blood sugar and lack of symptoms to either asymptomatic lactase deficiency, delayed gastric emptying, or lack of correlation in the sampling times and maximum lactose absorption. Nine of the 14 intolerants had a history of milk intolerance and the remaining 5 drank no milk at all.

The Peruvian, Mexican and Mexican American populations are similar in that they are primarily Mestizo populations. Such racially mixed groups generally show a level of lactose malabsorption intermediate between the low level of Caucasians (0-20%) and the high level for Indians (up to 100%). For instance, Dill et al. (130) found 54% of 11 Mexican American adult males to have low lactase activity while only 1 of 8 Anglos (12%) had low lactase activity. Lisker et al. (131) tested 401 Mexican adolescents and adults and found 296 (73.8%) to be intolerant to lactose. Symptoms appeared more frequently in intolerant subjects (64.9%) than in normal subjects (46.3%). In peruvian Mestizo children aged 10 months to 17 years, Paige et al. (132,133) found the incidence of lactose malabsorption to be 84%, and of these 75% were lactose intolerant. Breast feeding, age of weaning, and continued milk consumption did not appear to alter the ability to tolerate a lactose load. Age was the most pertinent variable

in the ability to hydrolyze and absorb lactose.

In Colombia, Alzate et al. (134) tested 54 medical students for lactose tolerance, 29 of whom were "antioquenos," an ethnogeographic grouping which includes a mixture of Indian, White and Negro. Fifteen of the antioquenos were considered Mestizos, and the rest of the group were racially heterogeneous. Eleven of 29 antioquenos (37.9%) and 4 Mestizos (25%) were intolerant to an oral lactose load.

Vasconcellos and Goncalves (135) found lactose intolerance to be the cause of chronic diarrhea in 16 (64%) Brazilian patients in a group of 25 with chronic diarrhea. Eleven of the 16 improved clinically with a milk-free diet.

Nutritional implications of lactose intolerance

There is some controversy over the influence of lactose intolerance on the absorption of nutrients from milk and on milk-drinking habits. If lactose intolerant individuals restrict milk intake, their consumption of calcium, protein, riboflavin, and other nutrients found in high concentrations in milk may be less than desirable. Since two-thirds of the RDA for calcium is obtained from milk and milk products by the peoples in Europe and North America, this nutrient may be expected to be the most compromised when dairy product consumption is restricted (136).

In several studies on Negro and Peruvian children, Paige et al. (121,133,137) have found a decrease in lactose tolerance is paralleled by a decreased consumption of milk. Lactose intolerance was associated with poor milk consumption in Negro school children in Baltimore, but not among their white schoolmates (121). Greater milk consumption was observed in Peruvian children with normal lactose tolerance than in their

intolerant siblings (133). However, the symptoms observed in Peruvian children after lactose ingestion may be modified by additional foods consumed along with milk (137).

Figueroa et al. (138) classified 50 Peruvian adults as milk tolerant or milk intolerant on the basis of past histories. Those in the milk tolerant group with normal lactase activity could drink one quart of milk with no GI symptoms. Those in the milk tolerant group with a flat lactose tolerance test curve were classified as "asymptomatic," developing symptoms only after 2-3 glasses of milk, but tolerating the small amounts of milk consumed daily in their diets. The milk intolerant group developed GI symptoms after consuming one glass of milk.

Nandi and Parham (139) found no correlation between milk consumption and lactose intolerance in Orientals and Caucasians living in the U.S. The authors concluded that lactose intolerant individuals can comfortably consume milk in limited quantities, however they caution that it should not be assumed "that all foods will be equally desirable for persons of different ethnic origin."

Stephenson and Latham (140) found that lactose intolerant adults can consume 15 to 30 g of lactose with only mild symptoms occurring. Bell et al. (127) found that Alaskan Eskimo children can consume one glass of milk without difficulty even though the incidence of lactose intolerance is between 56 and 70%. The threshold at which symptoms appeared after lactose was between 10 and 20 g in most of the children, the equivalent of one glass of milk. Similarly, Habte et al. (103) found that Ethiopian children could drink 250 ml of milk each day, and symptoms occurring initially rapidly abated over the 4 week test period.

Bayless et al. (141) and Desai et al. (54) feel that environmental factors such as malnutrition, parasitosis, infectious diarrhea and the resultant mucosal damage may hasten the appearance of lactose intolerance, while the maintenance of adequate nutritional status by continued milk ingestion and the avoidance of intestinal damage during early childhood may delay the onset of lactase deficiency. Some may interpret this as induction of enzyme activity by the presence of substrate, but it is not an induction in the strict sense. Jones and Latham (142) examined the lactose tolerance of young children and their parents, and found some of the children under 5 years to be intolerant while 72% of the parents were lactose intolerant. Since all the parents were born and raised in Asia, Africa, or Latin America it is speculated that despite genetic influence, the expected fall in intestinal lactase activity may be delayed in these children who have significantly higher levels of milk consumption than their parents did.

The Food and Nutrition Board of the National Research Council (143) is of the opinion that individuals with low intestinal lactase levels will spontaneously limit their milk consumption to amounts that cause them no difficulty. Jussila (90) found lactose malabsorption to be most common in patients with a history of milk intolerance, and relatively common in light milk drinkers. Among 26 Arabs tested for lactose tolerance, 16 could not tolerate large amounts of milk, and 14 of these consumed very little or no milk (107). However, several studies have found no significant relationship between milk intake and lactose intolerance (54,131,144). Those who have low lactase levels but are not milk intolerant probably do not suffer symptoms because they either do not consume

the 1 to 4 glasses of milk necessary to produce symptoms or they consume a fermented form of milk such as yogurt or cheese. Milk and lactose intolerance would not be a problem if these individuals are allowed to follow their natural dietary habits.

The Protein Advisory Group of the United Nations (145) states that the most important question in the nutritional implications of lactose intolerance is whether those with low levels of lactase activity, particularly young children, will adapt so that they will tolerate milk consumption even if their levels of lactase activity are low. Development of tolerance to milk may be the result of small quantities of lactase dealing satisfactorily with lactose ingested without causing a normal rise in blood sugar, or it may be due to the development of a tolerance to unabsorbed lactose and the products of its breakdown.

Another important consideration is whether milk feeding in lactose intolerant individuals will affect the absorption of protein, calcium, and other nutrients present in milk. By definition, lactose intolerance implies a loss of lactose in the stool and thereby a loss of calories (145,146). Paige and Graham (146) reported increased stool water, fat and nitrogen in lactose intolerant children as compared to lactose tolerant children on a lactose-casein diet. Calloway and Chenoweth (147) reported two lactose intolerant adults showed no evidence of malabsorption on a diet containing 48 g of lactose per day while two other lactose intolerant adults had elevated levels of breath hydrogen and increased fecal losses of moisture, dry solids and energy. However, there were no significant differences in nitrogen balance among subjects on diets containing 48, 24 or 0 g of lactose. Condon et al. (148) performed a balance

study with one lactose intolerant and four tolerant volunteers. Administration of 60 g of lactose per day resulted in a fall in fecal and urinary calcium excretion, a striking improvement in calcium balance, and a mild improvement in phosphorus balance in the four controls. The lactose intolerant subject was in mildly negative calcium and phosphorus balance during the lactose-free diet period, but when changed to a diet containing 50 g of lactose per day there occurred an increase in frequency of bowel actions, increased mean stool weight, increased fecal fat, and calcium balance became more negative. Since glucose and galactose stimulate intestinal absorption of calcium, the beneficial effect of lactose on intestinal calcium absorption is probably dependent on its being hydrolyzed by lactase. Condon et al. (148) theorize that if lactase activity is low, lactose may not stimulate calcium absorption from the gut. Kocian et al. (149) found that in healthy subjects calcium absorption is higher from milk with normal lactose content than from lactose-free milk. Lactose intolerant subjects absorbed more calcium from lactose-free milk than from regular milk. Calcium absorption from milk in the lactose intolerant subjects was poor, however a balance study using labeled calcium revealed that calcium utilization and retention were practically equal, regardless of whether calcium was given in milk or lactose-free milk and whether it was given to healthy subjects or lactose intolerant patients.

Lactose intolerance has been reported to be associated with growth retardation in one case (150) and with osteoporosis in Caucasians but not in Negroes (151).

MATERIALS AND METHODS

The study population included 282 Mexican American children between the ages of 2 and 14 years, and 51 Anglo American children between the ages of 4 and 14 years. The Mexican American children were drawn from 10 schools in the San Antonio Independent School District, the Kenwood, Stonewall, Santa Cruz, and Pan-American EODC Head Start Centers, and Santa Rosa Children's Hospital in San Antonio, Texas. Criteria for selecting the Mexican American children stated that:

1. both parents be of Mexican or Mexican American descent;
2. children should be described by their school nurse as being "normal" and "healthy;"
3. children drawn from Santa Rosa Hospital should be free from gastrointestinal disorders and diseases involving the liver or pancreas, and should be tested as close to discharge as possible; and
4. no diabetic children should be included in the sample. In practice, the first selection guideline was modified so that Spanish surname of both parents was used in selecting children to participate in the study. Since 98% of the children attending school in the San Antonio Independent School District are Mexican American, this modification introduced little if any error.

The 51 Anglo American children were drawn from the Northside Independent School District and served as a control group. As used here, Anglo American refers to children whose parents are of northern European ancestry as determined by family surname. The Anglo American children also had to meet criteria numbers 2-4 listed above for the

Mexican American children in order to be included in the study population.

Parents of school children were advised of the testing by a letter sent to them through their child's school. The letter was accompanied by a consent form that had to be signed and returned before the testing could be done. Letters and consent forms were written in both English and Spanish, for there are many adults in the community who speak only Spanish. Hospitalized children were referred by their physicians and consent forms were signed by their parents on the day of testing. Copies of the letter to parents explaining the nature and purpose of the lactose tolerance testing and the consent form are found in Appendix A.

In the Mexican American group, the initial intent was to have at least 50 children in each of 5 age groups:

2 to 3 years
4 to 5 years
6 to 7 years
8 to 9 years
10 to 14 years.

In the Anglo American group the goal was to have 10 children in each of the above age groups. Table 1 shows the actual distribution of Mexican American and Anglo American children included in the study. The goal of 50 children in the 2-3 year and 6-7 year age groups was not reached in the Mexican American group. For the Anglo children, the goal of 10 children per age group was not reached for the 2-3 and 4-5 year age groups. We encountered difficulty in filling the younger age groups for several reasons. Although the Head Start Centers do accept some 2-year old children, most children do not enter the program until they are 3

TABLE 1

Distribution of Children by Age, Sex and Ethnic Background

	Mexican American			Anglo American		
Age	Males	Females	Total	Males	Females	Total
2-3	12	22	34	0	0	0
4-5	26	28	54	3	0	3
6-7	24	20	44	3	7	10
8-9	36	39	75	10	11	21
10-14	40	35	75	6	11	17
Total	138	144	282	22	29	51

or more years old. Since our study was conducted from December until June, many 3-year olds who had entered the program in September had already turned 4. A similar situation existed with the 6-year olds, who have to be 6 before they enter first grade. As seen in Table 1, approximately equal numbers of males and females are represented in the sample.

The information gathered on these children falls under three categories: the lactose tolerance test, anthropometric measurements, and dietary. Each of these areas will be discussed separately below.

Lactose tolerance test

The lactose tolerance test (LTT) was performed in the morning after an overnight fast. Parents were instructed in the original contact letter and again in a reminder from the school nurse to fast children after dinner on the night preceding the test, and to send them to school without breakfast. The LTT was performed in the first hour of school which usually began at 8:00 A.M. Between 4 and 7 children could be tested in this time, depending on their age and thereby on the extent of their cooperation. Finger prick, capillary blood samples were taken before the lactose load (0 time), and at 15, 30, and 60 minutes after the lactose load. Lactose was administered as a 20% solution in tap water flavored with 5 ml of lemon juice. Size of the dose was 2 g of lactose per kg of body weight in children weighing up to 25 kg. Those over 25 kg body weight were given a standard lactose load of 50 g. The lactose solution was prepared daily from glucose-free α-lactose hydrate (Sigma Chemical Company) and allowed to equilibrate for 24 hours prior to use. Each blood sample was collected in a heparinized 0.1 ml

capillary tube and analyzed for glucose by the semi-micro glucose oxidase method (144,145). On the day following the LTT the children were questioned on the occurrence, severity and duration of gastrointestinal symptoms in the intervening 24 hours. The questions asked were standardized and asked in the same order to each child.

A blood glucose rise of 25 mg per 100 ml or more indicates efficient intestinal hydrolysis and absorption of lactose (67,72,78,85,88, 114), and this was used to define the lactose absorber group. A blood glucose rise of less than 25 mg per 100 ml correlates well with intestinal lactase activity below 1 unit per g wet weight mucosa (67,72,78, 85,88, 114), and this was used to define the lactose malabsorbers.

Anthropometric measurements

Five anthropometric measurements were performed on each child: height, weight, head circumference, mid-arm circumference and triceps skinfold. Children removed their shoes, sweaters and jackets before measurements were taken. Weight was recorded to the nearest quarter pound. Height was measured against a vertical tape with a sliding horizontal bar. The children were urged to stand up straight and look straight ahead with heels against the base of the tape while the height measurement was taken. The horizontal bar was lowered to rest against the top of the head and the height was read and recorded to the nearest quarter inch. The contribution of socks to height was considered negligible.

Triceps skinfold thickness and mid-arm circumference were measured at a point on the left arm halfway between the tip of the acromion process and the tip of the elbow. This point was determined with the arm

flexed at a 90^{o} angle, and marked with a washable ink. The child was then instructed to let the arm hang relaxed at his side while the measurements were taken.

To measure skinfold thickness, Lange skinfold calipers were used. These calipers deliver a standard pressure of 10 g per mm^2 over the jaw faces. Skinfold thickness was measured 3 times, and the average of the readings was recorded to the nearest mm.

A flexible steel tape manufactured by Lufkin was used to measure head and arm circumferences. Head circumference was measured at the level of greatest head length. Arm circumference was measured at mid-arm at the same level as was used for the triceps skinfold measurement. Each circumference measurement was recorded to the nearest sixteenth of an inch and converted to mm.

The form used to record the anthropometric measurements is found in the Appendix A.

Dietary information

A 24-hour recall of food consumption was attempted for each child. Those children of school age (6-14 years) were questioned directly; while the mothers and Head Start Center teachers of pre-school children were interviewed to obtain information on the younger children's food consumption. To aid in estimating portion sizes the children, parents, and teachers interviewed were asked to refer to food models representing 4 ounces and 8 ounces of beverage, ¼ and ½ cup, 2 ounces of meat, and 1 teaspoon and 1 tablespoon. Questions were also asked on vitamin and mineral supplementation, usual milk and dairy products consumption, and the subject's ability to relate milk consumption to the occurrence of

the gastrointestinal symptoms of lactose intolerance.

The form used to record dietary recalls is located in the Appendix A.

The 24-hour recalls were evaluated for calories and for four nutrients: protein, calcium, riboflavin and vitamin A. These nutrients were chosen because they are present in high quantities in milk. The nutrient content of the various foods reported was calculated from standard tables (154,155). Several foods commonly eaten in Mexican American and in some Anglo American homes were not found in any standard tables, so the nutrient content of these foods was calculated from recipes provided by Mrs. Rosemary Casey, Public Health Nutritionist for the City of San Antonio and from Gladney (156). These recipes and the calculations are found in the Appendix B.

RESULTS

Lactose tolerance test

The data on lactose tolerance for the Mexican American and Anglo American children were analyzed on the basis of age, sex, ethnic group, blood glucose rise and the occurrence of symptoms. The results and statistical analyses for each of these follow.

Table 2 presents the incidence of lactose malabsorption in Mexican American males by age groups. In this and the following contingency tables, the lactose malabsorbers are defined as having a blood glucose rise less than 25 mg per 100 ml after the lactose load, while lactose absorbers had a rise greater than or equal to 25 mg per 100 ml. The χ^2 test for homogeneity indicates that there is a significant difference between the age groups with regard to lactose malabsorption. For purposes of summary and to give a clearer picture of the relationship between lactose malabsorption and age, some columns in Table 2 were combined and the results are presented in Table 3. Only 26% of the Mexican American boys aged 2-5 had lactose malabsorption, and this increased to 40% in the 6-9 year old boys, and 65% in boys 10 and over. The overall incidence of lactose malabsorption in the Mexican American boys was 43%.

In Table 4, the incidence of symptoms in Mexican American boys is presented by age groups. The χ^2 test indicates that there are significant differences between the age groups in the incidence of symptoms. Collapsing Table 4 to 3 age groupings yields Table 5, and a clearer picture of the relationship between age and incidence of symptoms. Of the 2-5 year olds 34% had symptoms. The incidence of symptoms increased to 35% in the 6-9 year old boys, and 65% in boys 10 and over. The overall

TABLE 2

Number of Mexican American Males with Lactose Malabsorption

	Age Group (yrs.)					
	2-3	4-5	6-7	8-9	10-14	Total
Malabsorber	4	6	13	11	26	60
Absorber	8	20	11	25	14	78
Total	12	26	24	36	40	138

$x^2 = 16.01$, 4 d.f., $P < .005$

TABLE 3

Incidence of Lactose Malabsorption in Mexican American Males

	Age Group (yrs.)							
	2-5		6-9		10-14		Total	
Malabsorber	10	(26%)	24	(40%)	26	(65%)	60	(43%)
Absorber	28	(74%)	36	(60%)	14	(35%)	78	(57%)
Total	38		60		40		138	

TABLE 4

Number of Mexican American Males Reporting Symptoms After the LTT

	Age Group (yrs.)					
	2-3	4-5	6-7	8-9	10-14	Total
Present	5	8	10	11	26	60
Absent	7	18	14	25	14	78
Total	12	26	24	36	40	138

$\chi^2 = 11.74$, 4 d.f., $P < .025$

TABLE 5

Incidence of Symptoms After the LTT in Mexican American Males

	Age Group (yrs.)							
	2-5		6-9		10-14		Total	
Present	13	(34%)	21	(35%)	26	(65%)	60	(43%)
Absent	25	(66%)	39	(65%)	14	(35%)	78	(57%)
Total	38		60		40		138	

incidence of symptoms in Mexican American boys was 43%, while the remaining 57% had no symptoms.

The Mexican American females show similar incidences of lactose malabsorption and symptoms when compared to the males. Table 6 presents the incidence of lactose malabsorption by age groups for the Mexican American females. The χ^2 test indicates that there is a significant difference between the age groups regarding the incidence of lactose malabsorption. This relationship is more clearly defined in Table 7 which collapses the original 5 age groups to 3. The incidence of lactose malabsorption increases with age from 12% in the 2-5 year old girls, to 39% in the 6-9 year olds, and 46% in those 10-14 years. The overall incidence of lactose malabsorption in the Mexican American girls was 31%, while 69% digested lactose well.

Table 8 presents the incidence of symptoms after the lactose tolerance test in the Mexican American girls by age groups. The χ^2 statistic indicates that there are significant differences between the age groups in the incidence of symptoms. Collapsing this table yields Table 9 which gives a clearer picture of the relationship between symptoms and age. Only 6% of the 2-5 year old Mexican American girls had symptoms after the LTT, but this increased to 49% in the 6-9 year olds and 69% in those 10-14. The overall incidence of symptoms in the Mexican American girls was 39%.

Not all children with lactose malabsorption had symptoms after the LTT. On the other hand, some lactose absorbers experienced symptoms after the LTT. The association of symptoms with lactose malabsorption is presented in Table 10 for the Mexican American males and females. Of

TABLE 6

Number of Mexican American Females with Lactose Malabsorption

	Age Group (yrs.)					
	2-3	4-5	6-7	8-9	10-14	Total
Malabsorber	3	3	6	17	16	49
Absorber	19	25	14	22	19	99
Total	22	28	20	39	35	144

$\chi^2 = 14.86$, 4 d.f., $P<.025$

TABLE 7

Incidence of Lactose Malabsorption in Mexican American Females

	Age Group (yrs.)							
	2-5		6-9		10-14		Total	
Malabsorber	6	(12%)	23	(39%)	16	(46%)	45	(31%)
Absorber	44	(88%)	36	(61%)	19	(54%)	99	(69%)
Total	50		59		35		144	

TABLE 8

Number of Mexican American Females Reporting Symptoms After the LTT

	Age Group (yrs.)					
Symptoms	2-3	4-5	6-7	8-9	10-14	Total
Present	3	0	8	21	24	56
Absent	19	28	12	18	11	88
Total	22	28	20	39	35	144

$\chi^2 = 40.38$, 4 d.f., $P<.001$

TABLE 9

Incidence of Symptoms After the LTT in Mexican American Females

	Age Group (yrs.)							
Symptoms	2-5		6-9		10-14		Total	
Present	3	(6%)	29	(49%)	24	(69%)	56	(39%)
Absent	47	(94%)	30	(51%)	11	(31%)	88	(61%)
Total	50		59		35		144	

those boys who had symptoms, 70% were lactose malabsorbers and 30% were absorbers. In the females of those who had symptoms 54% were lactose malabsorbers and 46% were absorbers. Of those reporting no symptoms, 23% were male lactose malabsorbers and 17% were female malabsorbers. Cochran's method (157) was applied to Table 10 to test whether the degree of association between malabsorption and the presence of symptoms was the same for both males and females (test for homogeneity) and whether this association was significant (test for association). The χ^2 test for homogeneity gives a value of 0.17 with 1 d.f., indicating that the degree of association between lactose malabsorption and symptoms for the males and females is the same. In the χ^2 test for association the value of χ^2 is 51.46 with 1 d.f. and $P < .005$, indicating that the common degree of association of lactose malabsorption with symptoms is highly significant. To further test for association, we can calculate the estimate of the apparently common degree of association as measured by the standardized difference, $\bar{d} = 1.81$ where $\bar{d}$ is referred to a normal distribution with mean zero and standard error estimated to be 0.2525 (158). If there were no association between lactose malabsorption and symptoms in the males and females we would expect $\bar{d} = 0$. Since $\bar{d}$ is significantly greater than 0, this analysis leads one to conclude that the Mexican American males and females constitute a homogeneous population in the incidence of lactose malabsorption and symptoms after the LTT.

Tables 11 and 12 present the foregoing material in a different manner. Table 11 presents the incidence of malabsorption in Mexican American males and females with the incidence of malabsorption being 37% for the

TABLE 10

Association of Symptoms with Lactose Malabsorption in Mexican American Males and Females

	Males				Females			
	Symptoms Present		Symptoms Absent		Symptoms Present		Symptoms Absent	
Malabsorber	42	(70%)	18	(23%)	30	(54%)	15	(17%)
Absorber	18	(30%)	60	(77%)	26	(46%)	73	(83%)

Homogeneity test: $\chi^2 = .17$, 1 d.f.

Association test: $\chi^2 = 51.46$, 1 d.f., $P<.005$

TABLE 11

Incidence of Lactose Malabsorption in Mexican American Males and Females

	Males		Females		Total	
Malabsorbers	60	(43%)	45	(31%)	105	(37%)
Absorbers	78	(57%)	99	(69%)	177	(63%)

$\chi^2 = 4.00$, 1 d.f., $P<.05$

TABLE 12

Incidence of Symptoms after the LTT in Mexican American Males and Females

Symptoms	Males		Females		Total	
Present	60	(43%)	56	(39%)	116	(41%)
Absent	78	(57%)	88	(61%)	166	(59%)

$\chi^2 = .61$, 1 d.f.

whole group. Table 12 presents the incidence of symptoms in males and females, the combined incidence being 41%. The value of χ^2 for Table 11 is barely significant at the 0.05 level (χ^2 = 4.00 with 1 d.f.). This test indicates that sex and the incidence of lactose malabsorption are associated, but in view of the previous analysis of Cochran's method this conclusion can be discounted. Furthermore, given the significant association between lactose malabsorption and symptoms we would expect the χ^2 value for Table 12 to be considerably larger than 0.61 if there were a difference in lactose malabsorption between the sexes. Since the χ^2 statistic is not significant, there is no difference between males and females in the incidence of symptoms after the LTT. This further supports the analysis by Cochran's method and we can conclude that there are no differences between males and females in the incidence of lactose malabsorption or symptoms. In further analysis of the LTT results we can group males and females together since they represent a homogeneous group. Table 13 summarizes the incidence of lactose malabsorption and symptoms in the Mexican American children.

Because of the size of the Anglo American group with only 4 lactose malabsorbers (3 male and 1 female) it is impossible to perform a statistical analysis similar to the preceeding one for Mexican American children. However, since no sex differences in the incidence of lactose malabsorption in Caucasians have been reported in the literature we can safely assume that the Anglo American males and females form a homogeneous population on the basis of lactose malabsorption as do the Mexican American children. Table 14 summarizes the incidence of lactose malabsorption in the Anglo children. As with the Mexican American children,

TABLE 13

Summary of Results of LTT in Mexican American Children

	Absorbers		Malabsorbers					
			Total		Without Symptoms		With Symptoms	
Age Group	No.	%	No.	%	No.	%	No.	%
2-5	72	82	16	18	11	12	5	6
6-9	72	60	47	40	15	13	32	27
10-14	33	44	42	56	7	9	35	47

TABLE 14

Summary of Results of LTT in Anglo American Children

	Absorbers		Malabsorbers					
			Total		Without Symptoms		With Symptoms	
Age Group	No.	%	No.	%	No.	%	No.	%
4-5	3	100	0	-	0	-	0	-
6-9	29	94	2	6	2	6	0	-
10-14	15	88	2	12	0	-	2	12

the incidence of lactose malabsorption increases with age, being 0% in 4-5 year olds, 7% in 6-9 year olds, and 12% in those 10 and over. Also, as was true with the Mexican American children, the incidence of symptoms increased with age and some lactose malabsorbers did not have symptoms after the LTT while some absorbers did have symptoms.

Table 15 compares the association of symptoms with the incidence of lactose malabsorption in both the Mexican American and Anglo American children. Previously we have shown that the incidence of symptoms following the LTT increased with age in both the Anglo and Mexican American groups, and that lactose malabsorption and the occurrence of symptoms after the LTT are significantly related. To determine if the degree of association between symptoms and lactose malabsorption is the same for both populations, we applied Cochran's method (157) to this contingency table. The χ^2 statistic for homogeneity is .046 with 1 d.f., indicating that the degree of association between symptoms and glucose rise is the same in both populations. The χ^2 test for association yields $\chi^2 = 54.46$ with 1 d.f. and $P < .005$. Therefore, the common association of symptoms with lactose malabsorption is significant. The estimate of this common association (158) is $\bar{d} = 1.82$, where $\bar{d}$ is referred to a normal random variable with mean 0 and estimated standard error 0.2459. Since $\bar{d}$ is significantly different from 0 there exists the same association between lactose malabsorption and symptoms for both populations.

Comparing the incidence of symptoms in the Mexican American group with the Anglo American group in Table 16, we find that the Mexican American children had a greater incidence of symptoms than the Anglo

TABLE 15

Association of Symptoms with Lactose Malabsorption in Mexican American and Anglo American Children

	Mexican American				Anglo American			
	Symptoms Present		Symptoms Absent		Symptoms Present		Symptoms Absent	
Malabsorber	72	(69%)	33	(31%)	2	(50%)	2	(50%)
Absorber	44	(25%)	133	(75%)	8	(17%)	39	(83%)

Homogeneity Test: $\chi^2 = .046$, 1 d.f.

Association Test: $\chi^2 = 54.46$, 1 d.f., $P<.005$

TABLE 16

Overall Incidence of Symptoms After the LTT in Mexican American and Anglo American Children

Symptoms	Mexican American		Anglo American	
Present	116	(41%)	10	(20%)
Absent	166	(59%)	41	(80%)

$x^2 = 8.50$, 1 d.f., $P<.005$

children (41% vs. 20%). A χ^2 test reveals that this difference is significant ($\chi^2 = 8.50$, with 1 d.f., and $P<.005$).

Table 17 compares the overall incidence of lactose malabsorption in the Mexican American children with the Anglo American children. The χ^2 statistic indicates that the incidence of lactose malabsorption was significantly greater in the Mexican American children ($\chi^2 = 13.52$, with 1 d.f., and $P<.005$).

The results of the preceding two analyses indicate that there are significant differences between the Mexican American and Anglo American groups with respect to the incidence of lactose malabsorption and symptoms in the total populations: the Mexican American children had a greater incidence of both. This relationship is presented graphically in Figure 1. The analysis by Cochran's method demonstrated that in both populations a low blood glucose rise after a lactose load (lactose malabsorption) is significantly associated with the occurrence of gastrointestinal symptoms. We can therefore conclude that while the degree of association between symptoms and lactose malabsorption is the same for both populations, the frequency of occurrence of both is greater in the Mexican American group. We are therefore dealing with two distinct groups on the basis of lactose tolerance.

The Mexican American and Anglo American lactose malabsorbers and absorbers showed similar patterns in the time of peak glucose rise during the LTT (Table 18). In the Mexican American malabsorbers, 27 (26%) had their peak glucose rise in the 15 min. blood sample, 53 (50%) in the 30 min. sample, and 20 (19%) in the 60 min. sample. Five (5%) of the Mexican American malabsorbers had no change in serum glucose

TABLE 17

Overall Incidence of Lactose Malabsorption in Mexican American and Anglo American Children

	Mexican American		Anglo American	
Malabsorber	105	(37%)	4	(8%)
Absorber	177	(63%)	47	(92%)

$\chi^2 = 13.52$, 1 d.f., $P<.005$

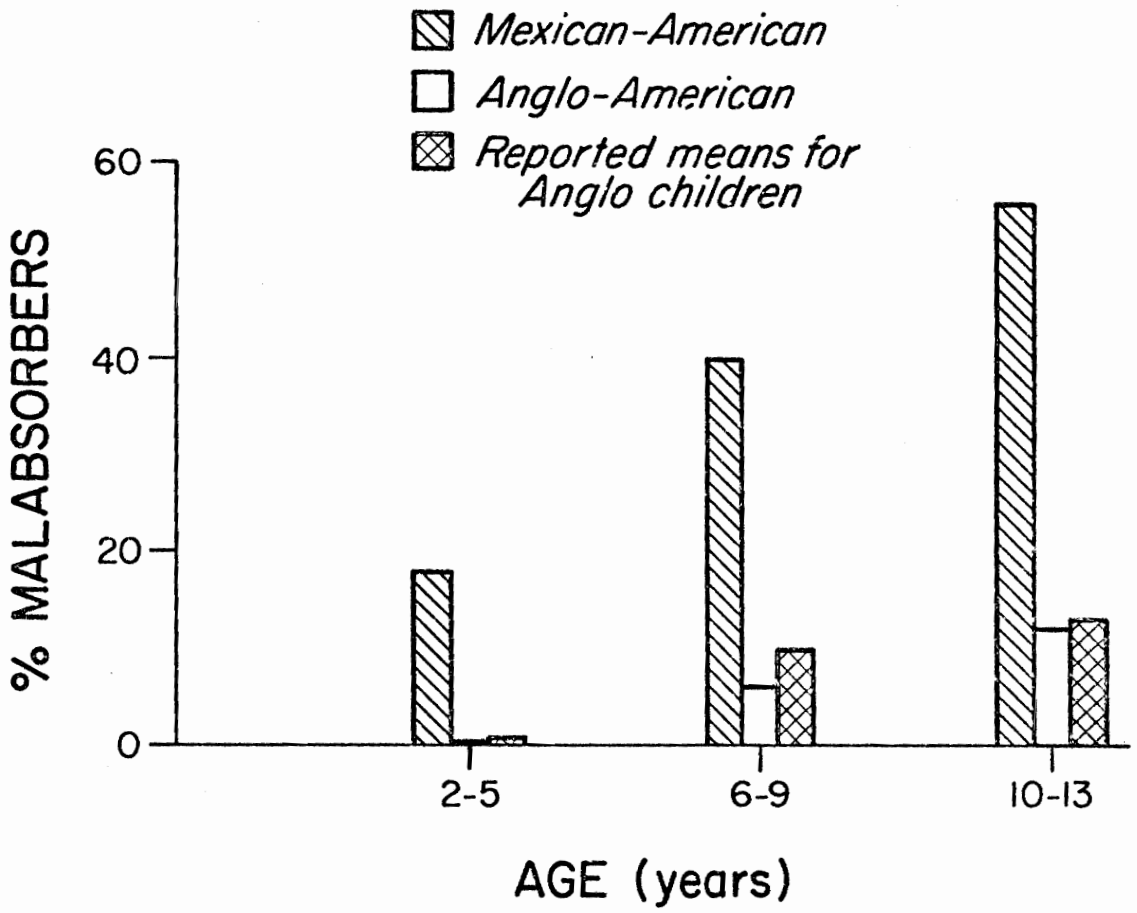

FIGURE 1. Incidence of lactose malabsorption in Mexican-American & Anglo-American children

TABLE 18

Time of Peak Glucose Rise

		Time (min)							
		15		30		60		No. Rise	
	No. of Subjects	No.	%	No.	%	No.	%	No.	%
Mexican American									
Malabsorbers	105	27	26	53	50	20	19	5	5
Absorbers	177	51	29	118	67	8	4	-	
Anglo American									
Malabsorbers	4	1	25	2	50	1	25	0	0
Absorbers	47	11	23	33	70	3	6	-	0

concentration or a slight decrease during the LTT. The Anglo American malabsorbers have the same 1:2:1 ratio for the time of peak glucose rise: 1 (25%) at 15 minutes, 2 (50%) at 30 minutes, and 1 (25%) at 60 minutes. The vast majority of lactose absorbers in both ethnic groups showed their peak serum glucose rise in the 15 and 30 minute samples, with approximately 5% of each group having a peak rise in the 60 minute sample.

On the morning following the LTT, each child was questioned on the occurrence of any gastrointestinal symptoms during the intervening 24 hours. The number of symptoms and the type of symptom (abdominal bloating, flatulence, cramps, loose stool, diarrhea) were recorded as well as an estimate of the length of time each was experienced. Tables 19 through 22 present the results of the questioning.

The number of symptoms reported by the Mexican American children is presented in Table 19. In those who experienced symptoms, the number of symptoms reported increased with age. In the 2-3 year group 29% experienced symptoms and all of them reported only 1 symptom. In the 4-5 year group 33% reported experiencing 1 symptom, but none reported more than 1 symptom. In the 6-7 year group, 32% reported 1 symptom, 26% reported 2 symptoms, and 10% reported 3 symptoms. In the 8-9 and 10-14 groups, the percentage reporting 1 or 2 symptoms decreased in comparison with 6-7 group, while the percentage reporting 3, 4 or more symptoms increased. The lactose absorbers show a similar increase with age in the number of symptoms reported, but the percentage of children experiencing symptoms is much smaller than the lactose malabsorbers.

Table 20 presents the number of symptoms reported by the Anglo

TABLE 19

Number of Symptoms Reported After Lactose Loading in Mexican American Children by Age Groups

	AGE									
	2-3		4-5		6-7		8-9		10-14	
	No.	%	No.	%	No.	%	No.	%	No.	%
Malabsorbers										
None	5	71	6	67	6	32	9	32	7	17
1	2	29	3	33	6	32	5	18	6	14
2	0		0		5	26	5	18	8	19
3	0		0		2	10	4	14	14	33
4 or more	0		0		0		5	18	7	17
Absorbers										
None	21	78	40	89	20	80	34	72	18	55
1	6	22	4	9	4	16	5	11	6	18
2	0		0		1	4	3	6	2	6
3	0		1	2	0		2	4	4	12
4 or more	0		0		0		3	6	3	9

TABLE 20

Number of Symptoms Reported After Lactose Loading in Anglo American Children by Age Groups

	AGE							
	4-5		6-7		8-9		10-14	
	No.	%	No.	%	No.	%	No.	%
Malabsorbers								
None	0		1	100	1	100	0	
1	0		0		0		1	50
2	0		0		0		0	
3	0		0		0		1	50
4 or more	0		0		0		0	
Absorbers								
None	3	100	7	78	17	85	12	80
1	0		2	22	1	5	2	13
2	0		0		2	10	1	7
3	0		0		0		0	
4 or more	0		0		0		0	

American children after the LTT. Neither of the 6-7 or 8-9 year old malabsorbers reported any symptoms, while one 10-14 year old malabsorber reported 1 symptom and the other reported 3 symptoms. None of the Anglo American absorbers reported more than 2 symptoms.

Table 21 presents the frequency with which each symptom was reported by the Mexican American children. In order of frequency the symptoms reported for the malabsorbers were: abdominal cramps (49 times), bloating (40 times), diarrhea (28 times), loose stool (22 times), and flatulence (22 times). The "other" symptoms reported by 6 children included nausea, vomiting and headache. The lactose absorbers reported in order of frequency: cramps (20 times), bloating (18 times), flatulence (15 times), loose stool (14 times), diarrhea (10 times), and "other" (6 times). In both groups abdominal cramps and bloating were reported most often while the malabsorbers had a much greater frequency of diarrhea than the absorbers.

In Table 22 is the frequency with which the Anglo American children reported each symptom. Only 2 of the 4 malabsorbers had symptoms. One reported having loose stool, the other reported bloating, cramps and diarrhea. In the Anglo absorbers, the most frequently reported symptom was bloating, followed by cramps, with flatulence, loose stool and diarrhea each reported once.

We have already shown that the Mexican American children had a higher incidence of symptoms than the Anglo children. As well as having a greater number of symptoms, the symptoms also tended to be more severe and of longer duration in the Mexican American children. Since the symptoms of lactose intolerance generally appear within 6-8 hours

TABLE 21

Frequency With Which Each Symptom Was Reported by Age Groups in Mexican American Children

Symptom	Number of Times Symptom Reported					
	2-3	4-5	6-7	8-9	10-14	Total
Malabsorbers						
Bloating	0	0	3	12	25	40
Flatulence	0	0	2	4	16	22
Cramps	1	0	7	15	26	49
Loose Stool	0	1	5	8	8	22
Diarrhea	1	1	4	8	14	28
Other	0	1	1	1	3	6
Absorbers						
Bloating	0	1	2	7	8	18
Flatulence	0	1	0	7	7	15
Cramps	0	0	3	8	9	20
Loose Stool	0	3	1	5	5	14
Diarrhea	4	2	0	0	4	10
Other	2	0	0	2	2	6

TABLE 22

Frequency With Which Each Symptom Was Reported in Anglo American Children by Age Group

Symptoms	Number of Times Symptom Reported				
	4-5	6-7	8-9	10-14	Total
Malabsorbers					
Bloating	0	0	0	1	1
Flatulence	0	0	0	0	0
Cramps	0	0	0	1	1
Loose Stool	0	0	0	1	1
Diarrhea	0	0	0	1	1
Other	0	0	0	0	0
Absorbers					
Bloating	0	2	3	0	5
Flatulence	0	0	0	1	1
Cramps	0	0	1	2	3
Loose Stool	0	0	1	0	1
Diarrhea	0	0	0	1	1
Other	0	0	0	0	0

after the lactose solution is ingested, most children would be expected to experience symptoms during school hours. None of the Anglo American children considered their symptoms severe enough to warrant going to the nurse's office. However, many of the Mexican American children spent from 1-4 hours with the school nurse and several had such severe diarrhea that they were sent home for the rest of the day. None of the children reported symptoms lasting more than 12 hours; most symptoms had subsided by mid-afternoon. By the morning following the LTT all reported feeling no abdominal distress.

Dietary analysis

The analyses of the 24-hour dietary recalls for five selected nutrients follow. For each ethnic group, a table displays the mean $\pm$ S.D. of the reported intake of each nutrient for males, females and the sexes combined by age groups. Also for each nutrient examined, a graph compares the Mexican American and Anglo American lactose absorbers and malabsorbers in 3 condensed age groups. Student's t-tests were used to determine differences between lactose malabsorbers and absorbers, males and females, and Mexican American and Anglo American ethnic groups.

Table 23 presents the mean caloric intake of the Mexican American and Anglo American males and females. There was no difference in caloric intake between the lactose absorbers and malabsorbers at any age in the males or females of either ethnic group. Furthermore, there was no significant differences in caloric intake between Mexican American males and females. Therefore, the mean caloric intake for Mexican American males and females can be combined and compared to the intake for Anglo children. A t-test reveals that the Anglo children reported consuming

TABLE 23

Mean ± S.D. Caloric Intake (KCal/day) of Mexican American and Anglo American Males and Females

	Males		Females		Combined	
Age (Yrs.)	Absorber	Malabsorber	Absorber	Malabsorber	Absorber	Malabsorber
Mexican American						
2-3	1289±193	1498±217	1459±266	1814±503	1409±256	1633±369
4-5	1411±399	1463±674	1445±302	1620±391	1430±345	1515±573
6-7	1194±337	1240±346	1295±607	1078±274	1265±533	1186±325
8-9	1195±417	1613±1152	1280±549	1289±556	1240±475	1405±838
10-14	1258±321	1249±406	1278±483	1195±442	1275±415	1228±416
Anglo American						
4-5	1534±138	-	-	-	1534±138	-
6-7	2175± 88	1702*	1787±435	-	1898±404	1702*
8-9	1924±524	1277*	1975±357	-	1954±422	1277*
10-14	1655±563	2198*	1782±464	2164*	1743±476	2181± 24

* Single observation

significantly more calories ($t = 3.29$, 15 d.f., $P<.005$). Figure 2 summarizes the data for caloric intake. If the caloric intake of each child is converted to a percent of the RDA (159) for his age and these percentages are averaged, the results can be graphed as in Figure 2. The 2-5 year old children of both ethnic groups came closest to meeting the RDA for calories than either of the other age groups. The caloric intakes reported by the Anglo children were generally higher at all ages than those reported by the Mexican American children. Finally, as age increased the number of children who reported consuming 100% of the RDA for calories decreased.

Table 24 presents the mean protein intake of the Mexican American and Anglo American children. There were no significant differences between lactose absorbers and malabsorbers in protein intake in the Mexican American group. There were also no significant differences between Mexican American males and females in protein intake, and between Mexican American and Anglo American children. Figure 3 summarizes the results of the protein intake data. The mean intake for protein of children in both ethnic groups at all ages far exceeded the RDA for protein.

In Table 25 the mean vitamin A intake of Mexican American and Anglo American males and females is shown. Figure 4 summarizes and compares the vitamin A intake data for the two ethnic groups. There were no significant differences between lactose absorbers and malabsorbers in the Mexican American males and females. Furthermore, there were no significant differences in vitamin A intake between Mexican American males and females. Comparing the vitamin A intake data between Mexican

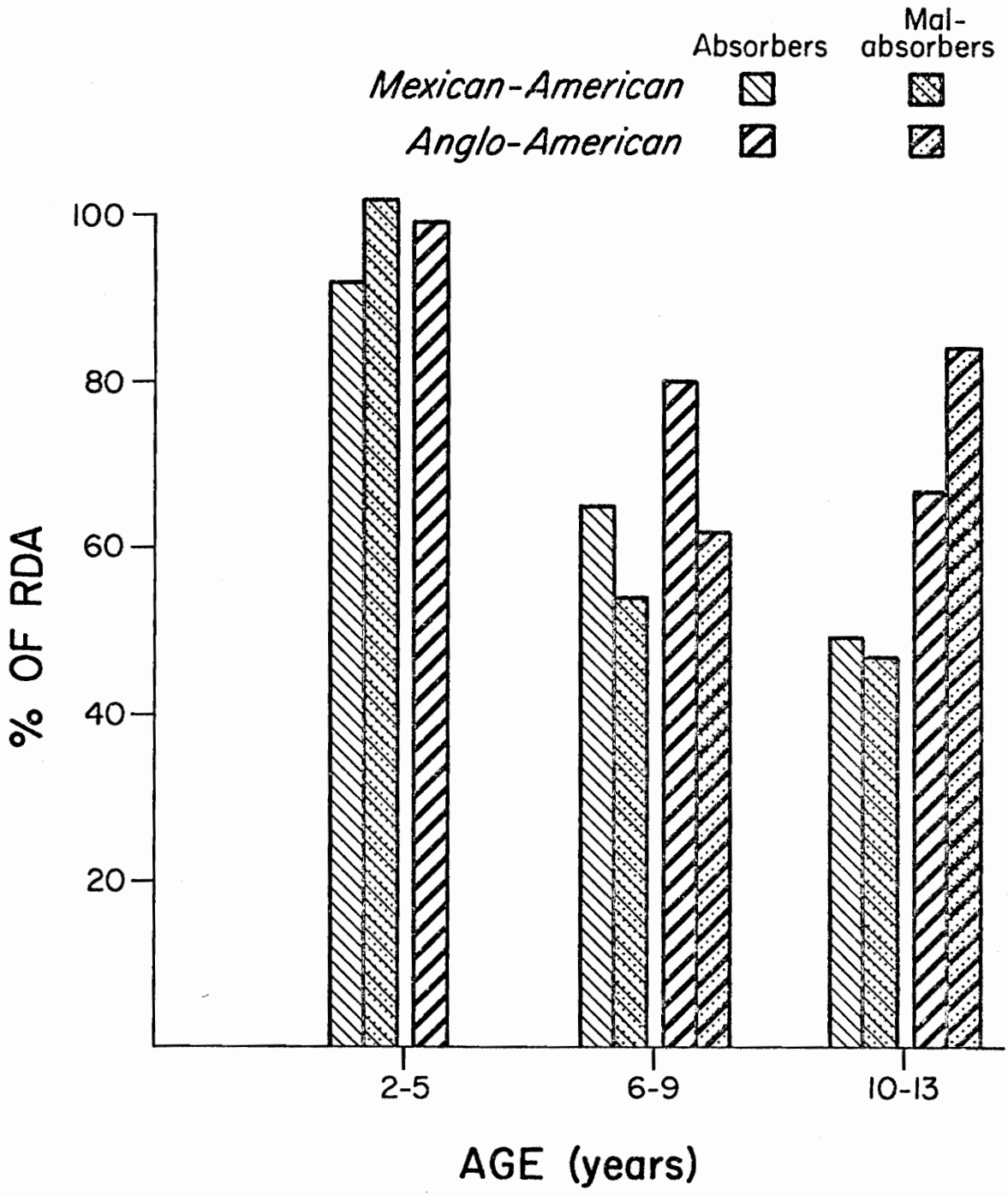

FIGURE 2. Caloric intake of Mexican-American & Anglo-American children

TABLE 24

Mean ± S.D. Protein Intake (g/day) of Mexican American and Anglo American Males and Females

	Males		Females		Combined	
Age (Yrs.)	Absorber	Malabsorber	Absorber	Malabsorber	Absorber	Malabsorber
Mexican American						
2-3	65.4±14.5	67.5± 8.7	62.9±13.2	73.2±26.0	61.0±13.6	70.0±16.5
4-5	55.3±14.9	54.0±27.5	57.4±12.8	71.6±23.9	56.5±13.7	59.8±26.3
6-7	45.4±17.6	58.0±19.4	56.1±28.2	45.3±11.0	53.0±25.5	53.8±17.8
8-9	52.0±19.5	73.1±52.5	50.5±25.4	54.6±17.9	51.3±22.1	61.4±35.1
10-14	53.5±20.8	59.2±25.4	59.0±23.7	49.4±19.0	56.7±22.3	55.4±23.4
Anglo American						
4-5	54.0± 9.6	-	-	-	54.0± 9.6	-
6-7	86.0± 9.9	61.0*	61.2±21.7	-	68.3±21.8	61.0*
8-9	81.0±37.0	40.0*	76.1±19.0	-	78.2±27.2	40.0*
10-14	65.0±41.8	106.0*	61.1±19.3	97.0*	62.3±26.3	101.5± 6.4

* Single observation

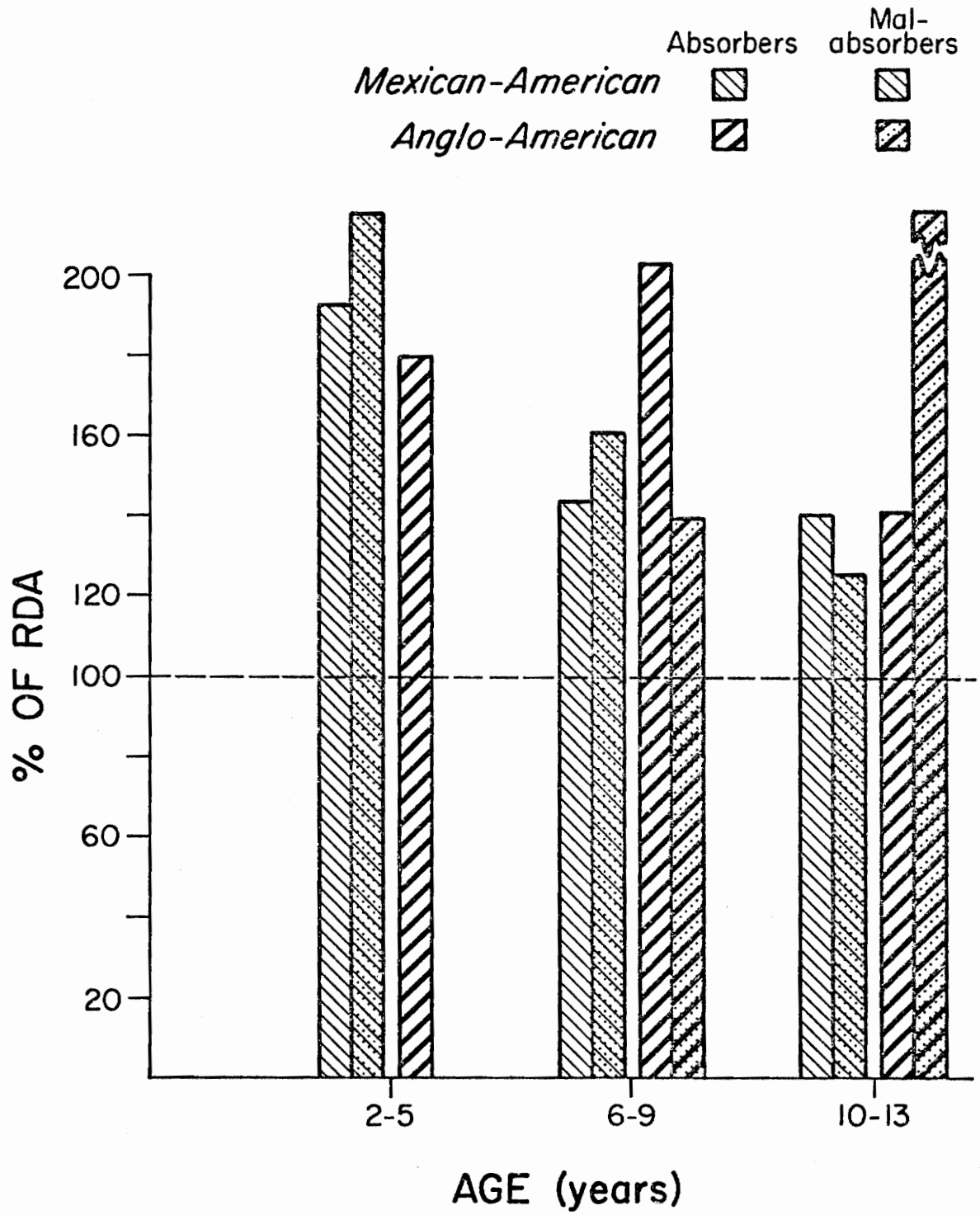

FIGURE 3. Protein intake of Mexican-American & Anglo-American

TABLE 25

Mean ± S.D. Vitamin A Intake (I /day) of Mexican American and Anglo American Males and Females

	Males		Females		Combined	
Age (Yrs.)	Absorber	Malabsorber	Absorber	Malabsorber	Absorber	Malabsorber
Mexican American						
2-3	3895±2098	4819±2328	3850±1932	3265± 865	3863±1942	4154±1910
4-5	3194±1634	3274±2111	2979± 956	2274±1062	3075±1290	2940±1821
6-7	1943± 894	1835± 879	2018±1215	1660±1104	1996±1103	1777± 930
8-9	1930±1437	2336±2203	2632±3086	2487±1833	2249±2335	2431±1937
10-14	2034±3105	2060±1700	1750±1038	1301± 530	1874±2157	1771±1415
Anglo American						
4-5	2707±1685	-	-	-	2707±1685	-
6-7	3330± 499	738*	5438±4086	-	4836±3497	738*
8-9	3552±1397	4883*	3140±1479	-	3313±1420	4883*
10-14	3925±3515	2481*	4817±2612	4588*	4542±2797	3985±2126

* Single observation

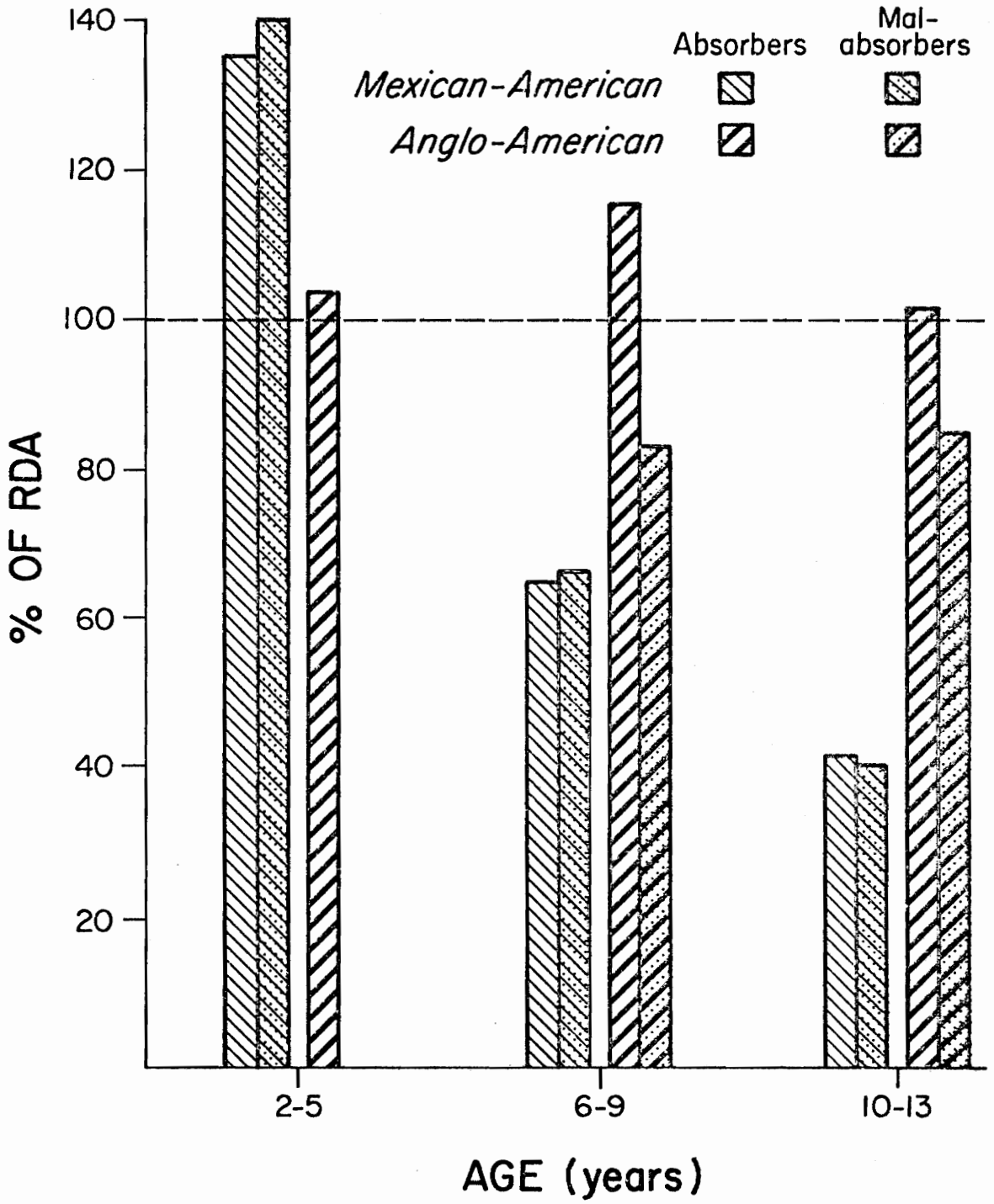

FIGURE 4. Vitamin A intake of Mexican-American & Anglo-American children

American and Anglo American children, there were no statistically significant differences between groups as a whole. However, the vitamin A intake of the Mexican American children when expressed as a percent of the RDA decreased greatly with age.

Table 26 shows the mean calcium intake of Mexican American and Anglo American children. Figure 5 summarizes and compares the data for calcium intake for Mexican and Anglo American children. There were no significant differences in calcium intake between Mexican American lactose absorbers and malabsorbers in either males or females. There were also no significant differences between Mexican American males and females in calcium intake, and between the Mexican American and Anglo American groups as a whole.

The mean riboflavin intakes of Mexican American and Anglo American children are presented in Table 27. Figure 6 summarizes and compares the riboflavin intake data for the two groups. There were no differences in riboflavin intake between the Mexican American lactose absorbers and malabsorbers except for the 4-5 year old females where the lactose malabsorbers reported consuming more riboflavin than the absorbers. However, when the Mexican American males and females were compared, there was no difference in reported riboflavin intake. Furthermore, there was no significant difference between the Mexican American children and the Anglo American children in reported riboflavin intake when the two groups were compared.

Figure 7 compares the nutrient intake of the Mexican American and Anglo American children with the nutrient intakes expressed as a percent of the RDA. The adequacy of the Mexican American children's diets de-

TABLE 26

Mean ± S.D. Calcium Intake (g/day) of Mexican American and Anglo American Males and Females

	Males		Females		Combined	
Age (Yrs.)	Absorber	Malabsorber	Absorber	Malabsorber	Absorber	Malabsorber
Mexican American						
2-3	0.6±0.3	0.8±0.2	0.8±0.2	0.9±0.3	0.7±0.2	0.9±0.3
4-5	0.6±0.3	0.6±0.3	0.6±0.2	0.8±0.2	0.6±0.2	0.6±0.3
6-7	0.7±0.2	0.6±0.3	0.6±0.3	0.6±0.2	0.6±0.3	0.6±0.3
8-9	0.5±0.3	0.7±0.7	0.6±0.4	0.6±0.3	0.6±0.4	0.6±0.4
10-14	0.6±0.4	0.6±0.4	0.5±0.3	0.4±0.2	0.6±0.3	0.5±0.3
Anglo American						
4-5	0.5±0.1	-	-	-	0.5±0.1	-
6-7	1.2±0.2	0.2*	0.9±0.4	-	1.0±0.3	0.2*
8-9	1.0±0.8	0.7*	1.1±0.5	-	1.1±0.6	0.7*
10-14	0.7±0.3	1.5*	0.9±0.4	1.9*	0.8±0.4	1.7±0.3

* Single observation

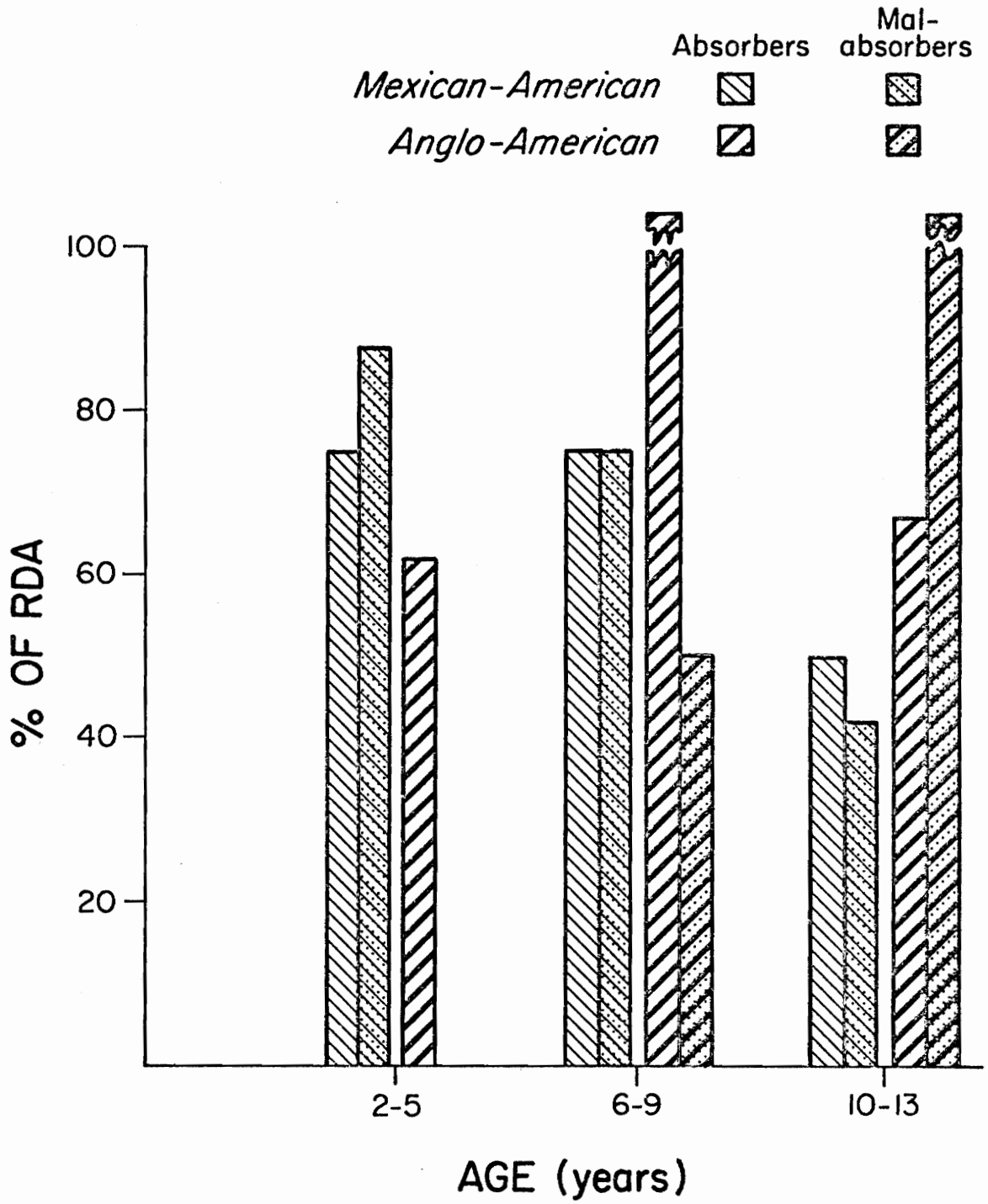

FIGURE 5. Calcium intake of Mexican-American & Anglo-American children

TABLE 27

Mean ± S.D. Riboflavin Intake (mg/day) of Mexican American and Anglo American Males and Females

	Males		Females		Combined	
Age (Yrs.)	Absorber	Malabsorber	Absorber	Malabsorber	Absorber	Malabsorber
Mexican American						
2-3	1.2±0.4	1.6±0.3	1.6±0.6	1.6±0.6	1.5±0.6	1.6±0.4
4-5	1.2±0.4	1.1±0.5	1.2±0.4	1.5±0.2	1.2±0.4	1.2±0.5
6-7	1.3±0.4	1.1±0.4	1.2±0.6	1.1±0.3	1.2±0.5	1.1±0.4
8-9	1.1±0.5	1.4±1.3	1.1±0.7	1.1±0.3	1.1±0.6	1.2±0.8
10-14	1.1±0.6	1.1±0.5	1.0±0.5	0.9±0.4	1.1±0.5	1.0±0.5
Anglo American						
4-5	1.0±0.2	-	-	-	1.0±0.2	-
6-7	2.0±0.4	0.7*	1.6±0.6	-	1.7±0.6	0.7*
8-9	1.7±1.0	1.4*	2.0±0.6	-	1.9±0.8	1.4*
10-14	1.2±0.6	2.4*	1.8±0.7	3.3*	1.7±0.7	2.8±0.6

* Single observation

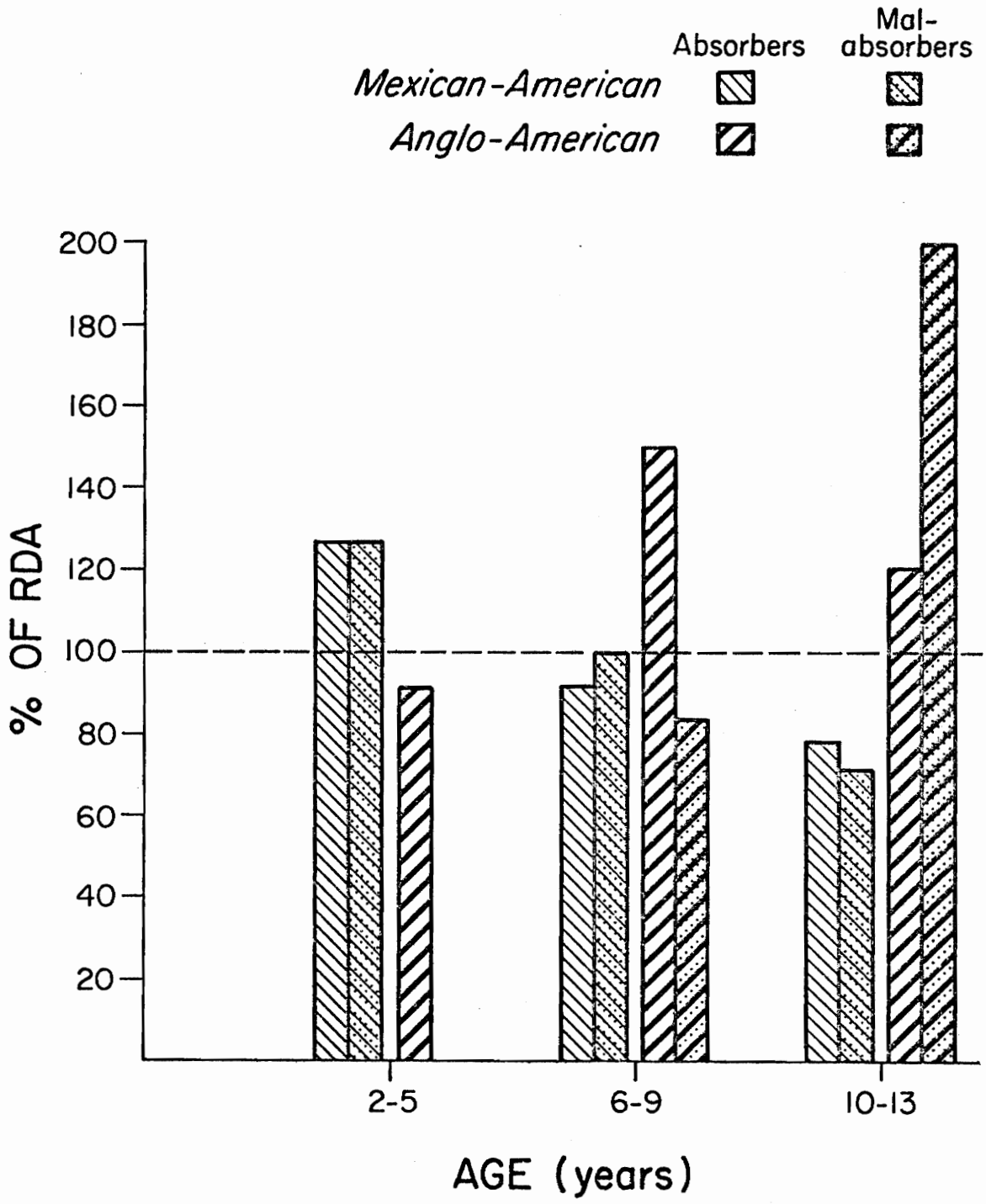

FIGURE 6. Riboflavin intake of Mexican-American & Anglo-American children

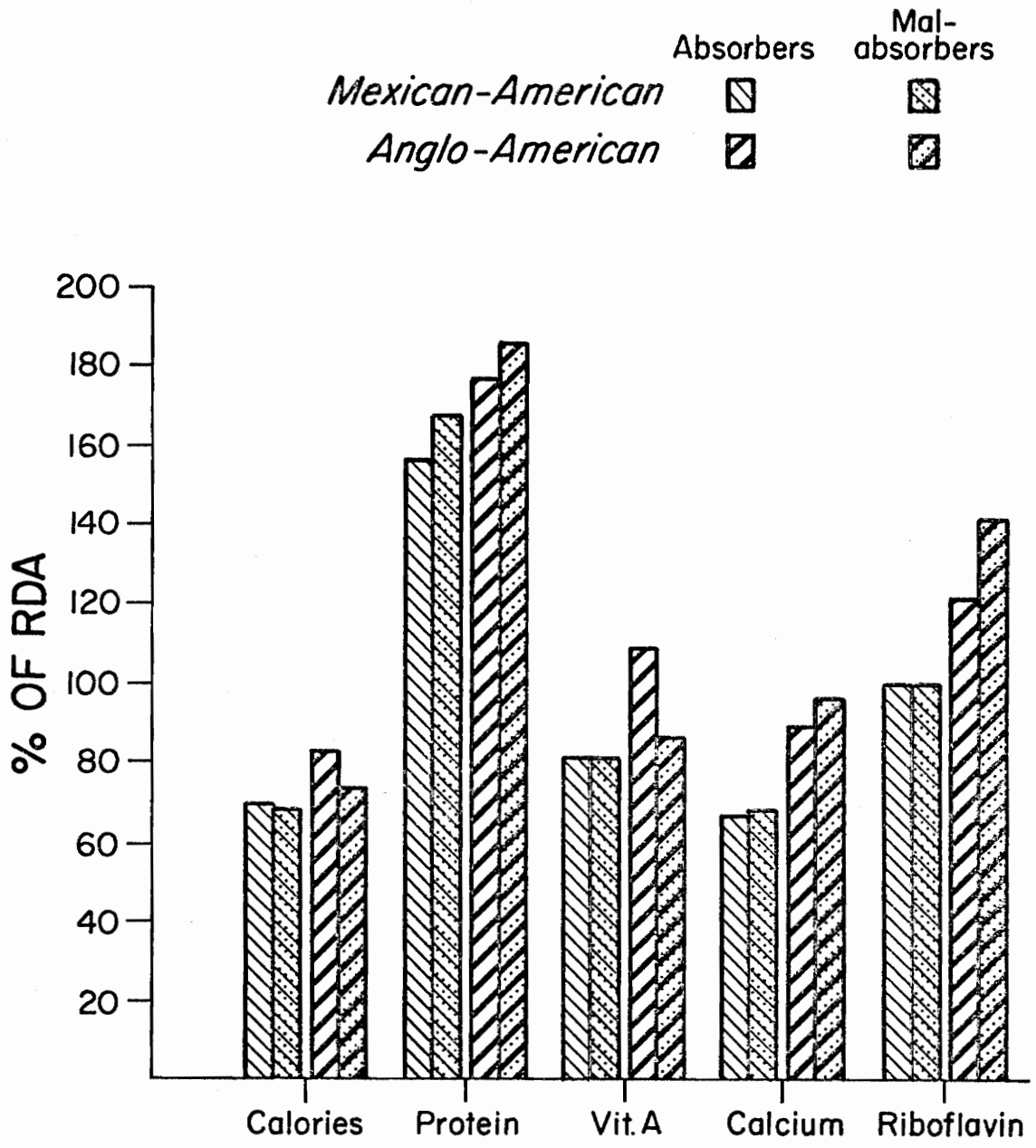

FIGURE 7. Caloric & nutrient intakes of Mexican-American & Anglo-American children

creased with age. The mean intakes of 2-5 year old Mexican American children exceeded the RDA for protein, vitamin A and riboflavin, and fell slightly below the RDA for calories. Mean calcium intake for 2-5 year old Mexican American children was approximately 80% of the RDA. The diets of the 2-5 year old children came closest to fulfilling their RDA for calories and the 4 nutrients examined. The mean intakes of 6-9 year old Mexican American children exceeded the RDA for protein and met the RDA for riboflavin, but fell below the RDA for calories, vitamin A and calcium. The mean intakes of 10-14 year old Mexican American children exceeded the RDA for protein but fell below the RDA for calories, vitamin A, calcium and riboflavin.

The Anglo children's intakes when expressed as a percent of the RDA did not show the decline with age seen in the Mexican American children. The mean intake in 2-5 year old Anglo American children exceeded the RDA for protein and vitamin A, met the RDA for calories, was 92% of the RDA for riboflavin, and only 62% of the RDA for calcium. The mean intakes of 6-9 and 10-14 year old Anglo children were adequate in protein, vitamin A and riboflavin, but fell below the RDA for calories. The mean intake of calcium exceeded the RDA in 6-9 year olds fell below the RDA for 10-14 year olds.

Table 28 shows the average daily milk consumption of the Mexican American and Anglo American children by age groups. Figure 8 summarizes the milk consumption data for the two ethnic groups. There were no significant differences between the lactose absorbers and malabsorbers in the Mexican American children. The Anglo American children drank significantly more milk than the Mexican American children ($t = 3.37$, 174

TABLE 28

Average Daily Milk Consumption of Mexican American and Anglo American Children

No. of Glasses	Age Group (Yrs.)					
	2-5		6-9		10-14	
	Absorber	Malabsorber	Absorber	Malabsorber	Absorber	Malabsorber
Mexican American						
0	7	2	19	12	11	16
1	17	4	28	15	10	18
2	13	1	10	9	6	7
3	5	2	3	1	3	0
4	0	0	1	2	0	1
Mean ± S.D.	1.4±0.9	1.3±1.0	1.0±0.9	1.1±1.1	1.0±1.0	0.9±0.9
Anglo American						
0	0	-	1	1	2	-
1	1	-	11	1	6	-
2	2	-	6	-	4	-
3	0	-	4	1	1	-
4	0	-	5	-	0	2
Mean ± S.D.	1.7±0.6	-	2.0±1.2	0.5±0.7	1.3±0.8	4.0

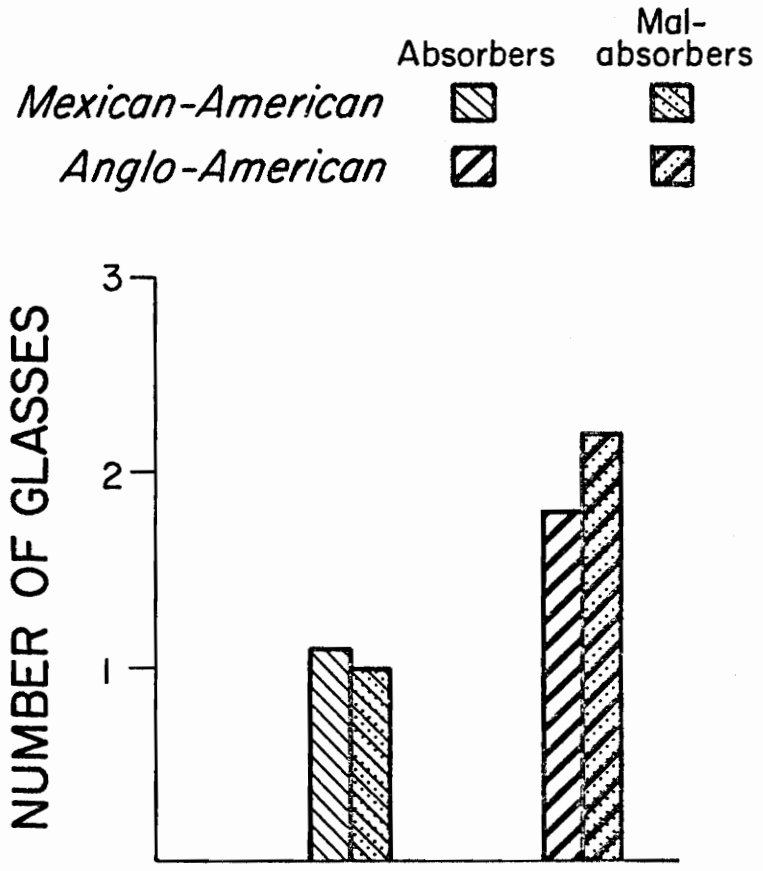

FIGURE 8. Average daily milk consumption

d.f., P<.001).

Table 29 shows the weekly cheese consumption of Mexican American and Anglo American children. Most of the children in both ethnic groups reported eating 1 to 2 servings of cheese per week. Sixty-seven percent of the Mexican American and 52% of the Anglo American children consumed 1 to 2 servings of cheese per week.

Table 30 shows the weekly ice cream consumption of Mexican American and Anglo American children by age groups. Most of the children in both groups reported eating ice cream 1 to 2 times per week. Seventy percent of the Mexican American and 47% of the Anglo American children consumed 1 to 2 servings of ice cream per week.

The number of Mexican American and Anglo American children reporting dietary supplementation with vitamin and/or mineral preparations is shown in Table 31. Twenty-four percent of the Mexican American children and 45% of the Anglo American children took some type of vitamin and/or mineral supplement.

Table 32 shows the percentage of Mexican American and Anglo American children who reported experiencing gastrointestinal symptoms after drinking milk. None of the Anglo American children had symptoms after milk, but 15% of the Mexican American lactose absorbers and 23% of the malabsorbers reported symptoms after milk.

Anthropometric measurements

The results of the five anthropometric measurements performed follow. For each measurement, the results are shown in tabular and graphic form.

Tables 33 and 34 show the mean weight in kg $\pm$ S.D. of Mexican

TABLE 29

Average Weekly Cheese Consumption of Mexican American and Anglo American Children

Servings Per Week	Age Group (Yrs.)					
	2-5		6-9		10-14	
	Absorber	Malabsorber	Absorber	Malabsorber	Absorber	Malabsorber
Mexican American						
<1	5	1	11	9	4	6
1-2	14	2	43	30	23	29
3-4	4	2	3	1	3	6
5-6	1	0	2	2	1	1
>6	2	2	2	0	0	0
Anglo American						
<1	0	-	2	1	4	0
1-2	0	-	16	0	6	1
3-4	2	-	2	1	2	1
5-6	0	-	2	0	0	0
>6	0	-	3	0	1	0

TABLE 30

Weekly Ice Cream Consumption of Mexican American and Anglo American Children

Servings Per Week	Age Group (Yrs.)					
	2-5		6-9		10-14	
	Absorber	Malabsorber	Absorber	Malabsorber	Absorber	Malabsorber
Mexican American						
<1	4	1	8	3	4	3
1-2	9	5	41	34	25	33
3-4	8	0	6	6	2	6
5-6	1	1	2	0	0	0
>6	3	1	4	0	0	0
Anglo American						
<1	0	0	0	1	0	0
1-2	1	0	10	0	9	1
3-4	1	0	4	1	3	1
5-6	0	0	1	0	0	0
>6	0	0	10	0	1	0

TABLE 31

Number of Mexican American and Anglo American Children Reporting Dietary Supplementation With Vitamins and Minerals

Type of Supplement	Age Group (Yrs.)					
	Mexican American			Anglo American		
	2-5	6-9	10-14	4-5	6-9	10-14
None	24	79	62	2	14	7
Vitamin	13	23	8	0	11	7
Mineral	0	0	1	0	0	0
Vitamin and Mineral	2	3	1	0	1	1
Unknown	0	1	1	0	2	0

TABLE 32

Percentage of Mexican American and Anglo American Children Reporting Symptoms After Milk

	Mexican American %	Anglo American %
Absorbers	15	0
Malabsorbers	23	0

TABLE 33

Mean ± S.D. Weight (kg) of Mexican American Children

	Males		Females	
Age (Yrs.)	Absorber	Malabsorber	Absorber	Malabsorber
2	11.4*	12.0*	–	–
3	15.2± 2.0	15.8± 0.9	14.7± 1.6	14.8± 0.6
4	16.3± 1.8	33.4*	17.5± 1.5	14.1*
5	18.8± 5.1	17.0± 2.2	17.3± 2.1	22.2± 9.1
6	20.8± 2.2	21.3± 3.8	21.4± 2.6	21.7± 5.4
7	26.9± 6.9	20.8± 1.7	21.9± 1.5	24.0± 2.7
8	27.6± 8.6	24.7± 2.6	24.0± 2.0	28.2± 2.5
9	30.8± 5.5	28.3± 7.1	29.2± 4.8	29.0± 6.6
10	38.4±12.3	27.4± 3.8	33.8± 8.9	33.4± 6.7
11	39.4±10.7	37.2± 4.2	40.4± 9.8	37.1± 7.2
12	33.6± 5.1	40.3± 9.9	34.4± 8.5	36.7± 3.5
13	37.7*	46.8±20.6	-	-

* Single observations

TABLE 34

Mean ± S.D. Weight (kg) of Anglo American Children

	Males		Females	
Age (Yrs.)	Absorber	Malabsorber	Absorber	Malabsorber
4	17.7*	-	-	-
5	21.4± 1.3	-	-	-
6	-	22.7*	23.4*	-
7	30.7± 4.8	-	27.0± 1.0	-
8	31.9± 5.8	28.6*	26.2± 4.4	-
9	31.2±10.0	-	31.9± 5.5	-
10	34.0± 5.2	35.0*	33.9± 8.2	25.0*
11	49.1*	-	33.8± 5.6	-

* Single observations

American and Anglo American children. Figures 9 and 10 compare the mean weight of the Mexican American and Anglo American boys and girls to several U.S. and other standards (160-166). Figures 11 and 12 show the distribution of the actual weights about the mean for the Mexican American and Anglo American children.

In Figures 9 and 10 it can be seen that the mean weights for the Mexican American males and females are above the Ten-State Nutrition Survey means (164) for Mexican American children and above the standard for Mexican children (166). They are close to the Stuart-Meredith standards (165) and National Center for Health Surveys' 50th percentile (162,163). Although the means compare well to weight standards, the mean does not give information on the two extremes of the population--those below the 5th percentile and those above the 95th percentile. Figures 11 and 12 are scatter diagrams comparing the weights of Anglo American and Mexican American children to their means and to the Stuart-Meredith 5th, 50th, and 95th percentiles (165). Twenty percent of the Mexican American males and 8% of the females were below the 5th percentile for weight, and 10% of the males and 6% of the females were above the 95th percentile. In the Anglo American children, none of the males and 7% of the females were below the 5th percentile for weight, and 23% of the males and 7% of the females were above the 95th percentile.

The mean height in cm $\pm$ S.D. of Mexican American males and females is shown in Table 35 and of Anglo American males and females in Table 36. Figures 13 and 14 compare the mean heights of Mexican American and Anglo American children to several standards (160-166). Tables 15 and 16 show the distribution of the actual heights about the mean for the Mexican

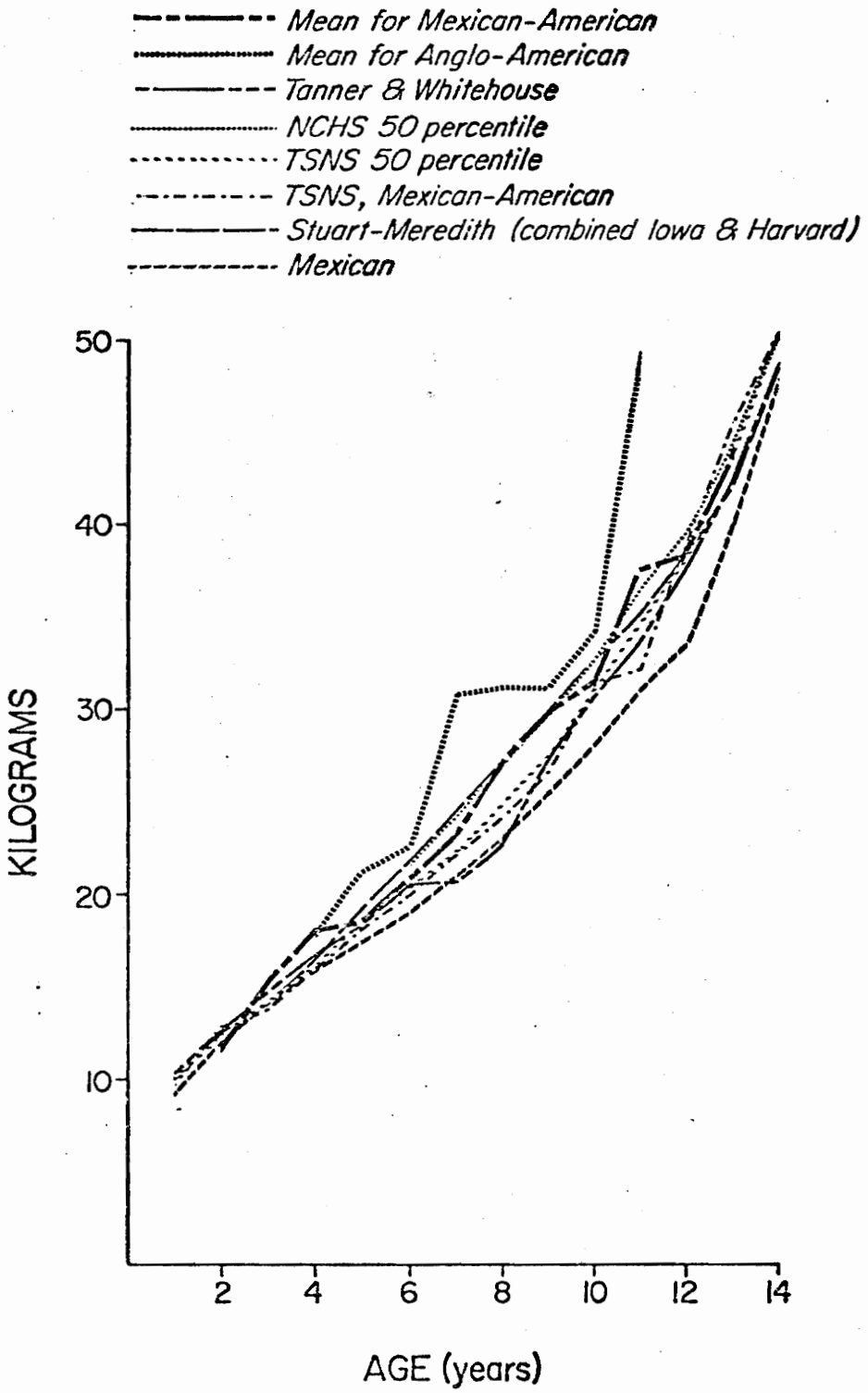

FIGURE 9. Mean weight of Mexican-American & Anglo-American males compared to weight standards

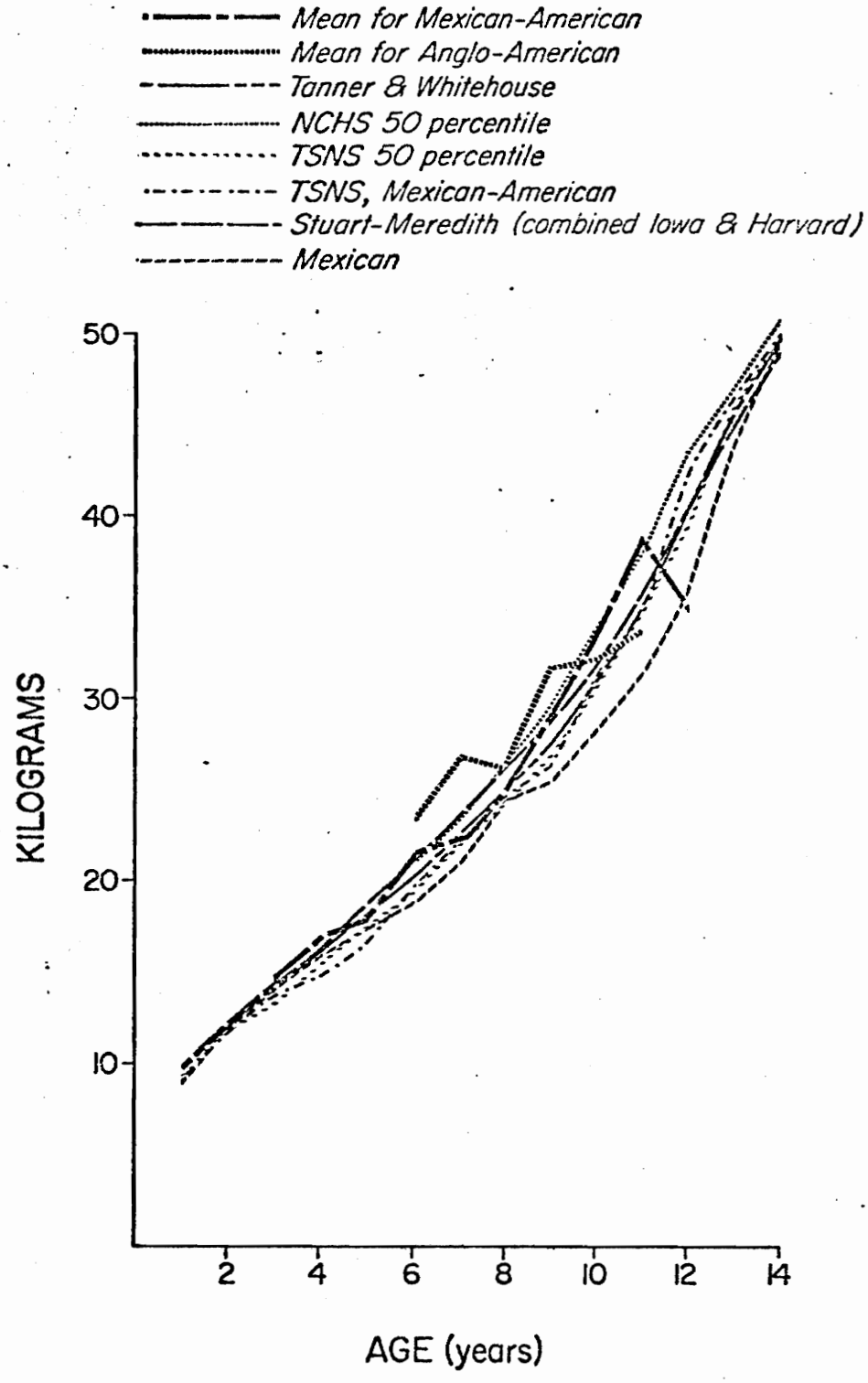

FIGURE 10. Mean weight of Mexican-American & Anglo-American females compared to weight standards

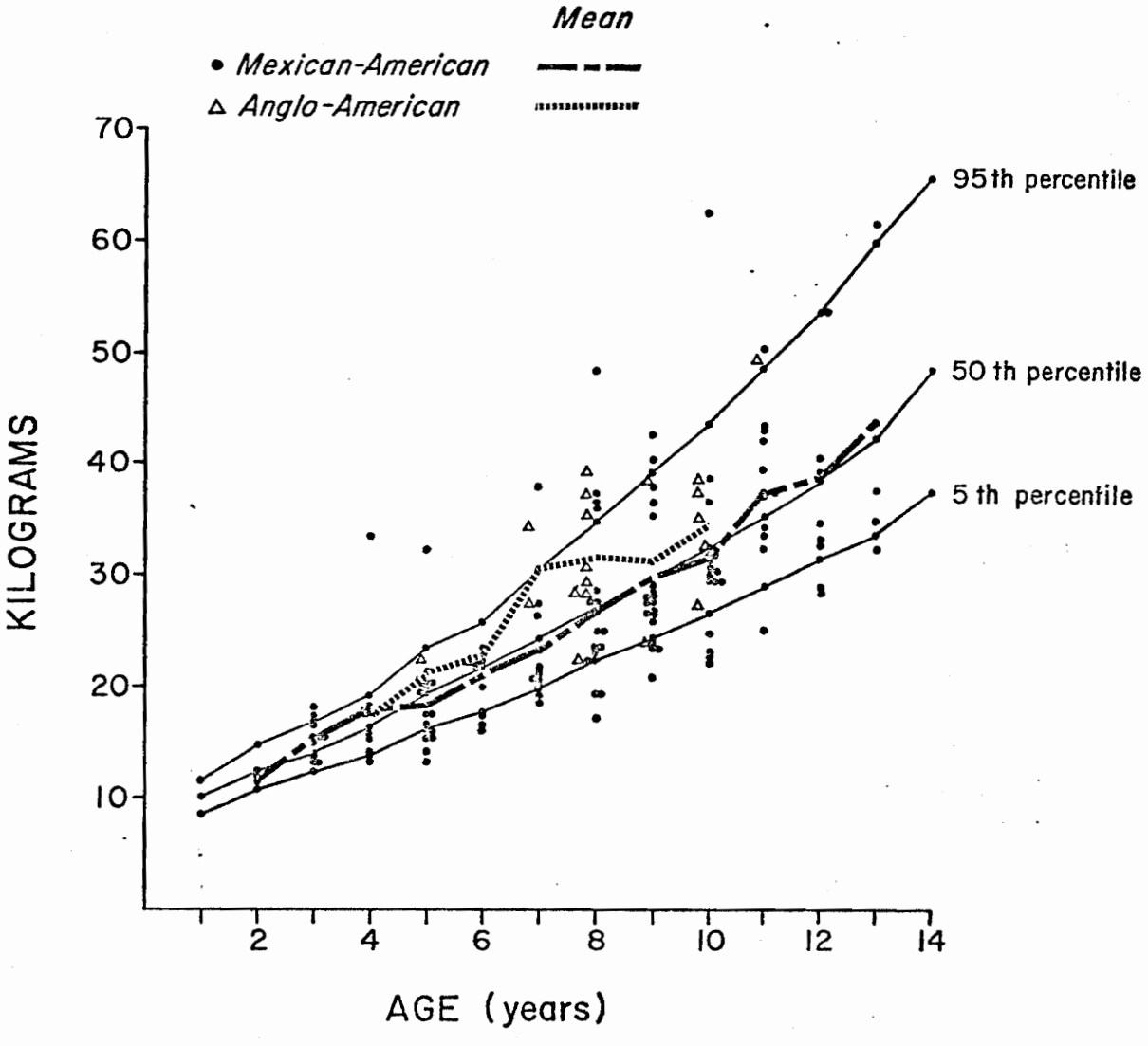

FIGURE II. Weights of Mexican-American and Anglo-American males compared to Stuart-Meredith Standard

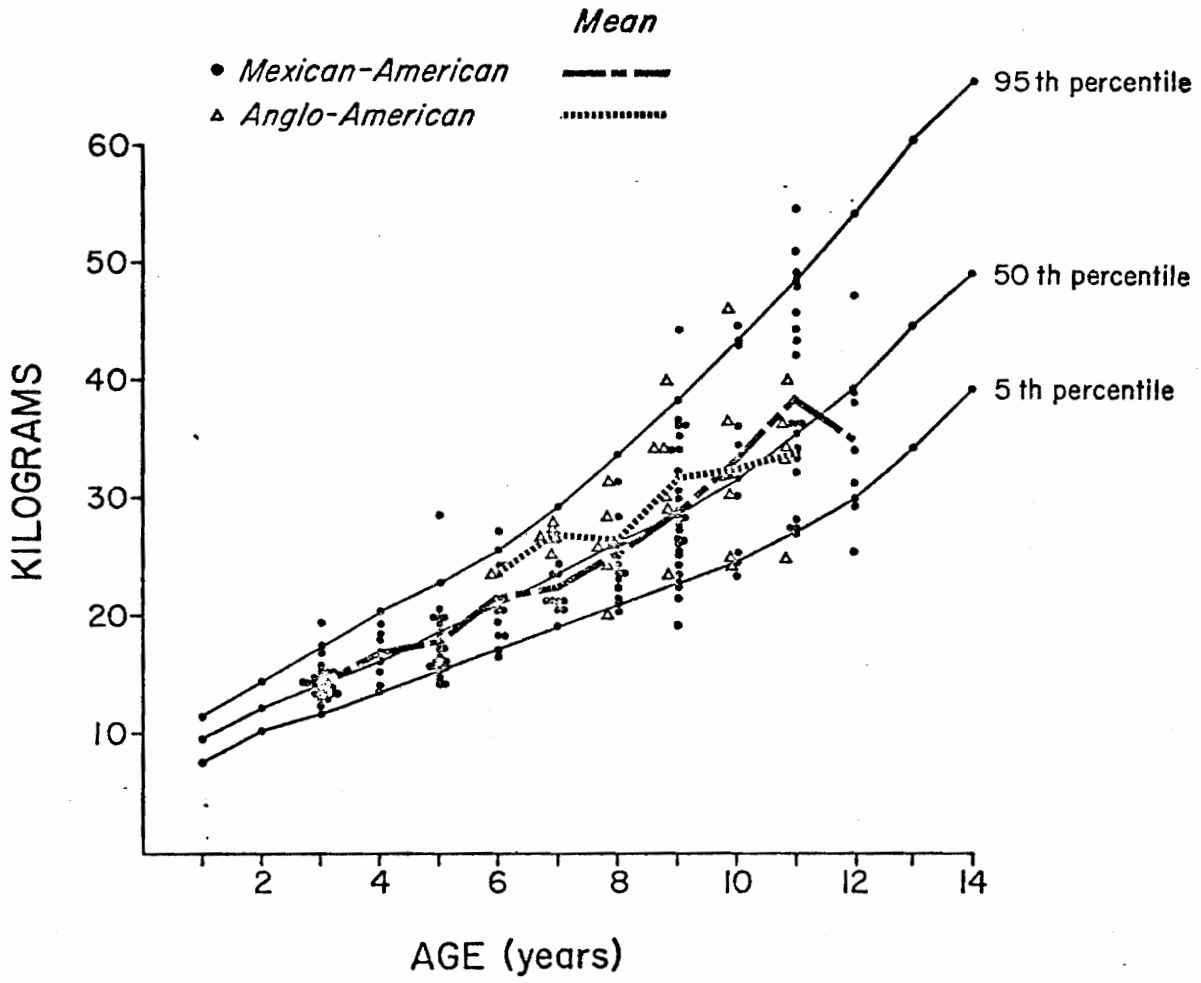

FIGURE 12. Weights of Mexican-American and Anglo-American females compared to Stuart-Meredith Standard

TABLE 35

Mean ± S.D. Height (cm) of Mexican American Children

	Males		Females	
Age (Yrs.)	Absorber	Malabsorber	Absorber	Malabsorber
2	84.0*	90.2*	-	-
3	96.6± 4.2	98.1± 2.8	96.9± 3.1	97.7± 2.1
4	102.8± 3.7	114.3*	104.7± 3.7	98.4*
5	108.6± 6.6	107.0± 6.8	107.5± 4.8	108.3±10.3
6	115.5± 5.0	114.7± 5.7	115.7± 3.7	119.4± 8.9
7	121.7± 5.9	116.3± 4.9	118.0± 3.4	123.8± 5.4
8	125.9± 8.8	124.2± 3.0	125.0± 4.4	130.3± 1.7
9	133.1± 5.0	130.0± 8.0	132.7± 5.4	129.6± 6.7
10	138.8± 8.8	131.5± 5.2	141.2±10.4	136.6± 7.4
11	144.5±10.8	138.8± 6.0	143.8± 6.8	142.3± 7.5
12	139.1± 4.6	147.6± 9.3	145.1± 8.1	142.9± 4.5
13	143.5*	152.7±12.2	-	-

* Single observation

TABLE 36

Mean ± S.D. Height (cm) of Anglo American Children

	Males		Females	
Age (Yrs.)	Absorber	Malabsorber	Absorber	Malabsorber
4	108.0*	-	-	-
5	113.7± 2.7	-	-	-
6	-	124.5*	127.0*	-
7	130.8± 1.8	-	130.6± 4.4	-
8	137.5± 5.6	135.9*	131.7± 8.4	-
9	134.6± 5.4	-	142.0± 4.0	-
10	141.9± 7.5	142.2*	143.0±11.0	132.1*
11	153.7*	-	143.8±11.6	-

* Single observation

American and Anglo American males and females.

Figures 13 and 14 show that the heights of Mexican American males and females fell above the means for Mexican American children in the Ten-State Nutrition Survey (164) and below the Stuart-Meredith standard (165). The height of the Mexican American males was lower in comparison to male standards than the height of Mexican American females in comparison to female standards. As was true for weight, scatter diagrams such as Figures 15 and 16 reveal important information on the number of children above and below the normal range in height. Twenty-five percent of the Mexican American males and 13% of the females were below the 5th percentile for height. Four percent of the males and 3% of the females were above the 95th percentile for height. None of the Anglo boys and 3% of the girls were below the 5th percentile for height, and 23% of the boys and 45% of the girls were above the 95th percentile.

Tables 37 and 38 show the mean head circumference in cm of the Mexican American and Anglo American children. Figures 17 and 18 compare the mean head circumference of the Mexican American and Anglo American children to 2 standards (164,167). Figures 19 and 20 show the distribution of actual head circumference measurements about the mean for Mexican American and Anglo American children.

The mean for Mexican American males was below the Nellhaus standard (167) and below the Ten-State Nutrition Survey mean (164) for head circumference until age 9 (Figure 17). After 9 years, the Mexican American males caught up to and closely paralleled the Nellhaus standard. Figure 18 shows that the Mexican American females were close to the Ten-State Nutrition Survey mean head circumference, but below the Nellhaus

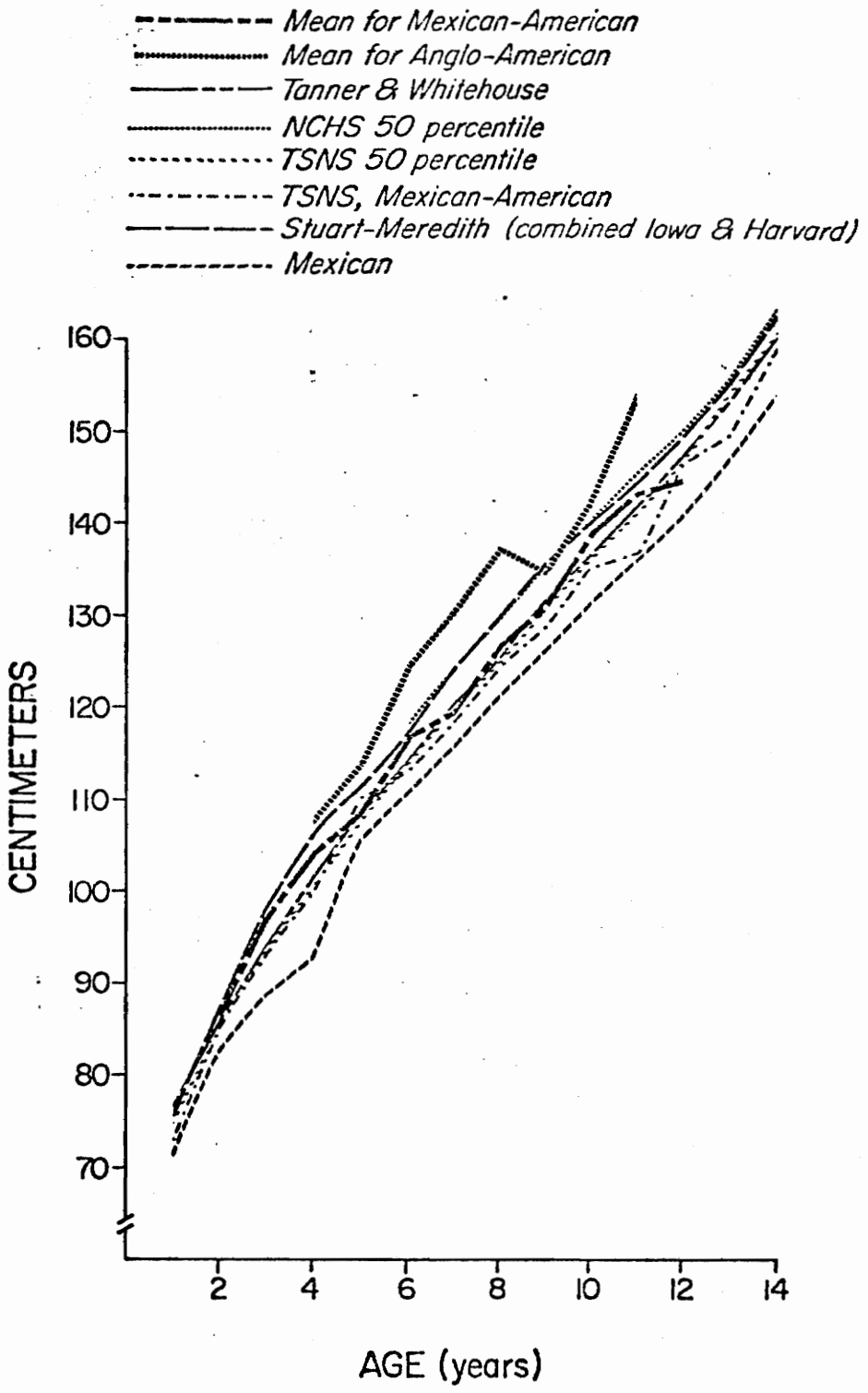

FIGURE 13. Mean height of Mexican-American & Anglo-American males compared to height standards

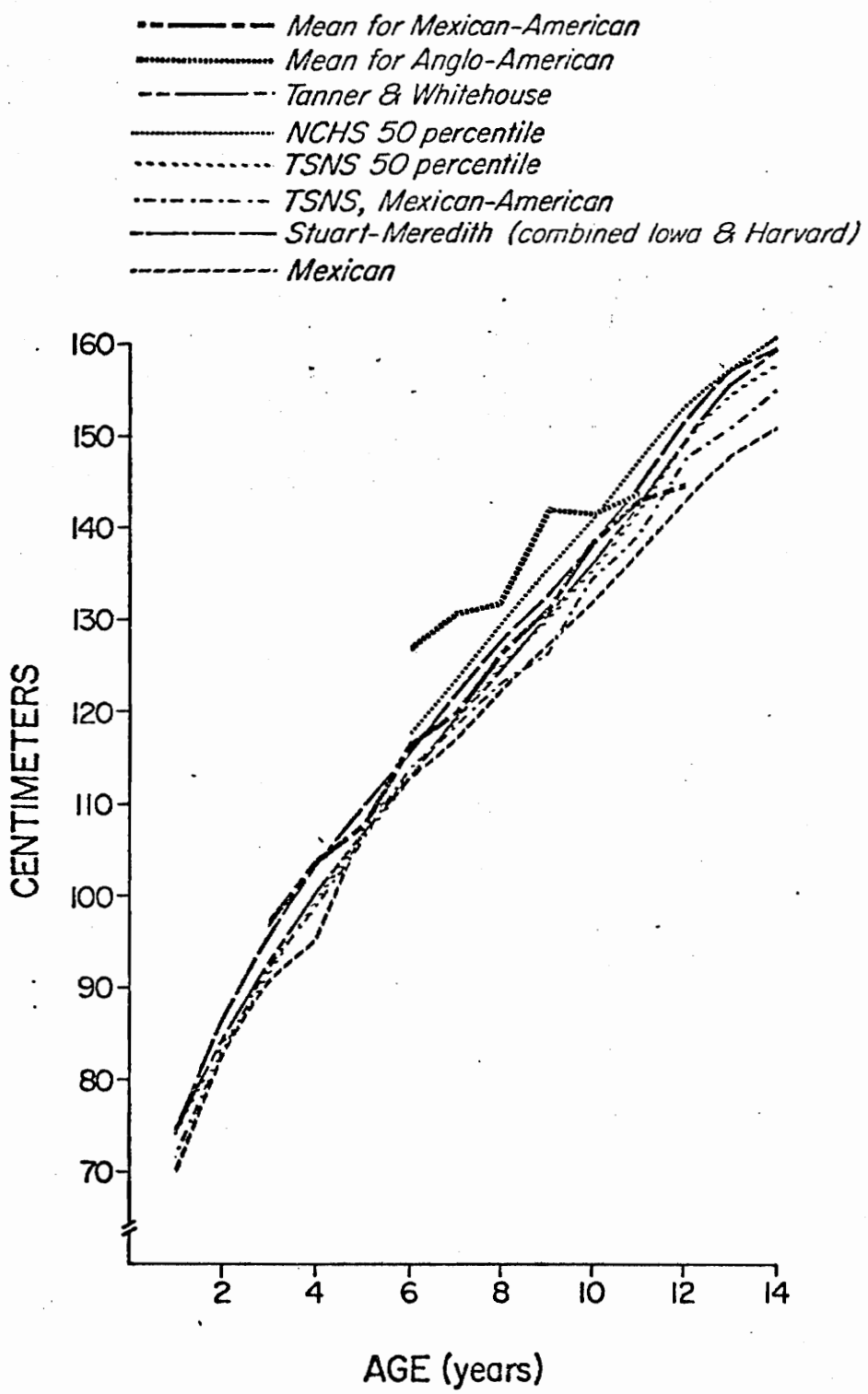

FIGURE 14. Mean height of Mexican-American & Anglo-American females compared to height standards

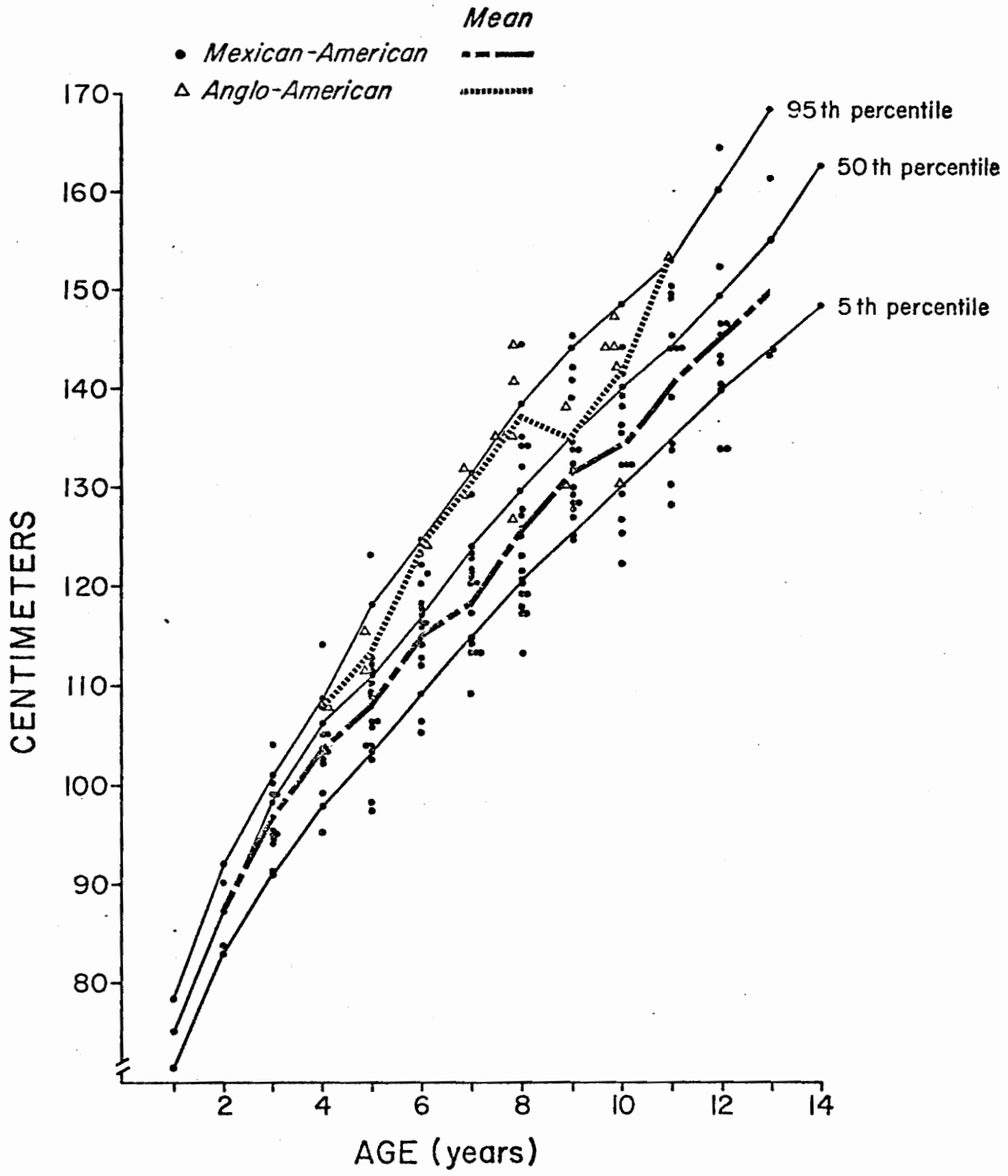

FIGURE 15. Heights of Mexican-American and Anglo-American males compared to Stuart-Meredith Standard

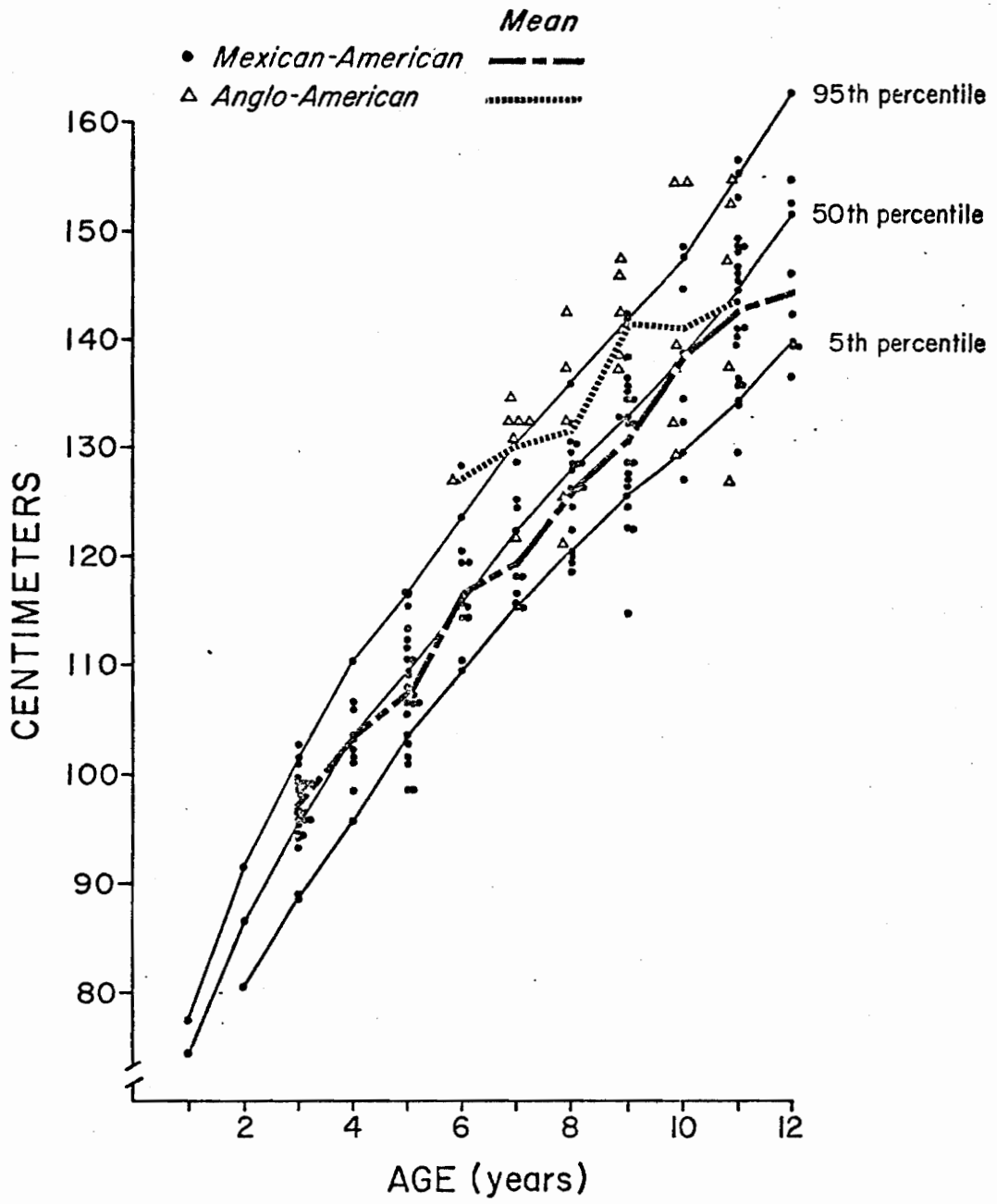

FIGURE 16. Heights of Mexican-American and Anglo-American females compared to Stuart-Meredith Standard

TABLE 37

Mean ± S.D. Head Circumference (cm) of Mexican American Children

	Males		Females	
Age (Yrs.)	Absorber	Malabsorber	Absorber	Malabsorber
2	48.3*	48.3*	-	-
3	49.2± 1.3	49.4±1.1	48.5± 1.5	48.8± 0.5
4	49.8± 1.3	53.0*	49.3± 1.6	49.5*
5	49.9± 1.6	49.5± 1.5	49.0± 1.0	50.2± 1.8
6	51.8± 0.9	50.4± 1.9	49.6± 1.5	50.8± 1.9
7	53.1± 1.4	50.8± 2.5	51.3± 1.4	52.8± 1.9
8	52.2± 2.1	51.3± 1.7	51.6± 1.6	52.3± 1.3
9	53.7± 1.6	52.4± 1.3	51.6± 1.1	51.4± 1.5
10	53.6± 1.7	52.3± 1.8	53.0± 1.1	52.9± 2.4
11	55.4± 1.4	52.6± 1.7	53.9± 1.8	53.8± 1.5
12	52.3± 0.8	54.2± 1.9	52.8± 1.1	52.2± 1.1
13	54.6*	56.2±	-	-

* Single observation

TABLE 38

Mean ± S.D. Head Circumference (cm) of Anglo American Children

Age (Yrs.)	Males		Females	
	Absorber	Malabsorber	Absorber	Malabsorber
4	50.2*	-	-	-
5	52.8± 0.6	-	-	-
6	-	50.8*	51.4*	-
7	54.4± 0.1	-	51.5± 0.9	-
8	54.1± 1.2	53.6*	51.7± 0.5	-
9	53.0± 2.3	-	53.1± 1.4	-
10	54.3± 1.0	53.6*	52.8± 0.7	52.1*
11	57.2*	-	53.7± 1.0	-

* Single observation

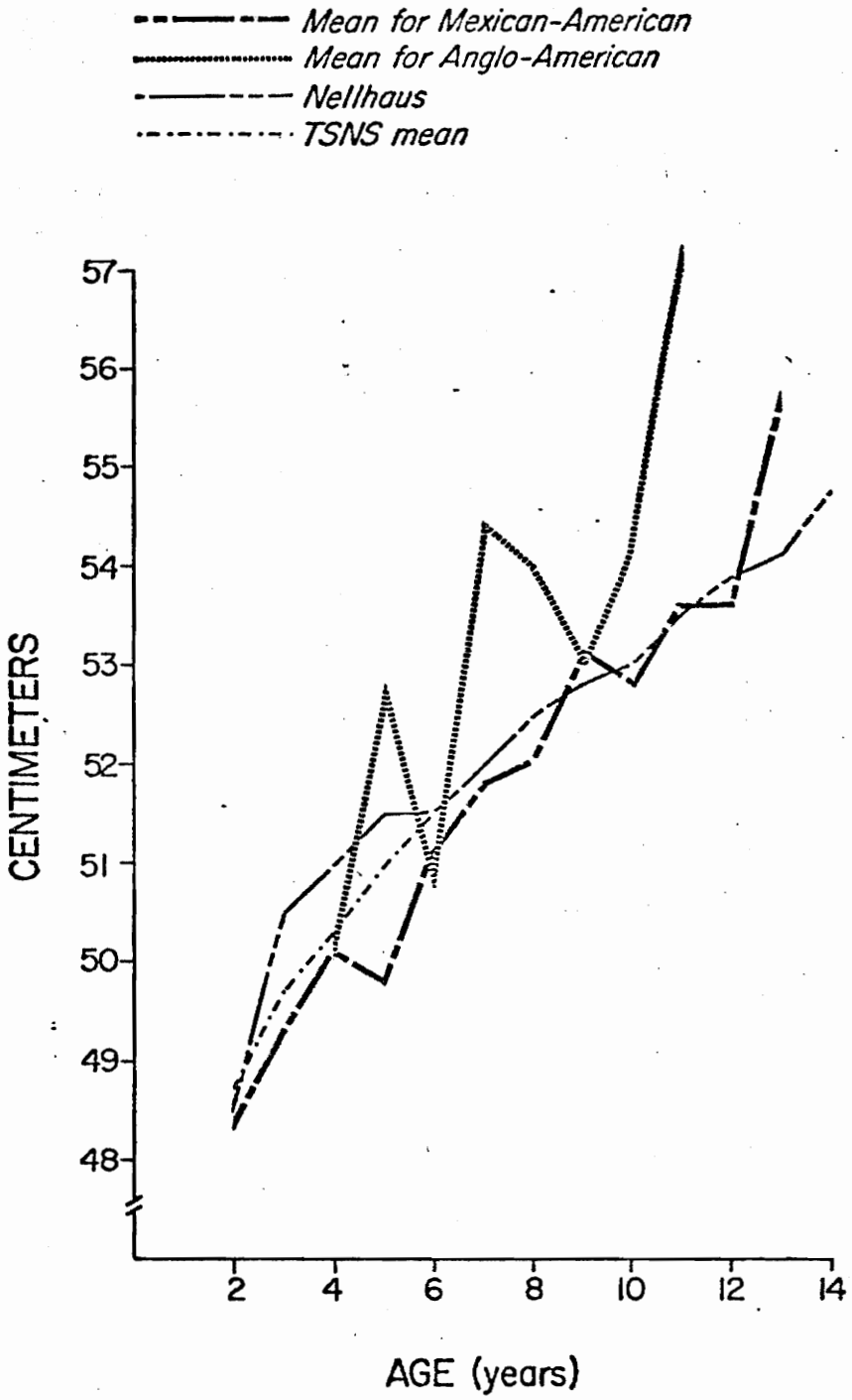

FIGURE 17. Mean head circumference of Mexican-American & Anglo-American males compared to head circumference standards

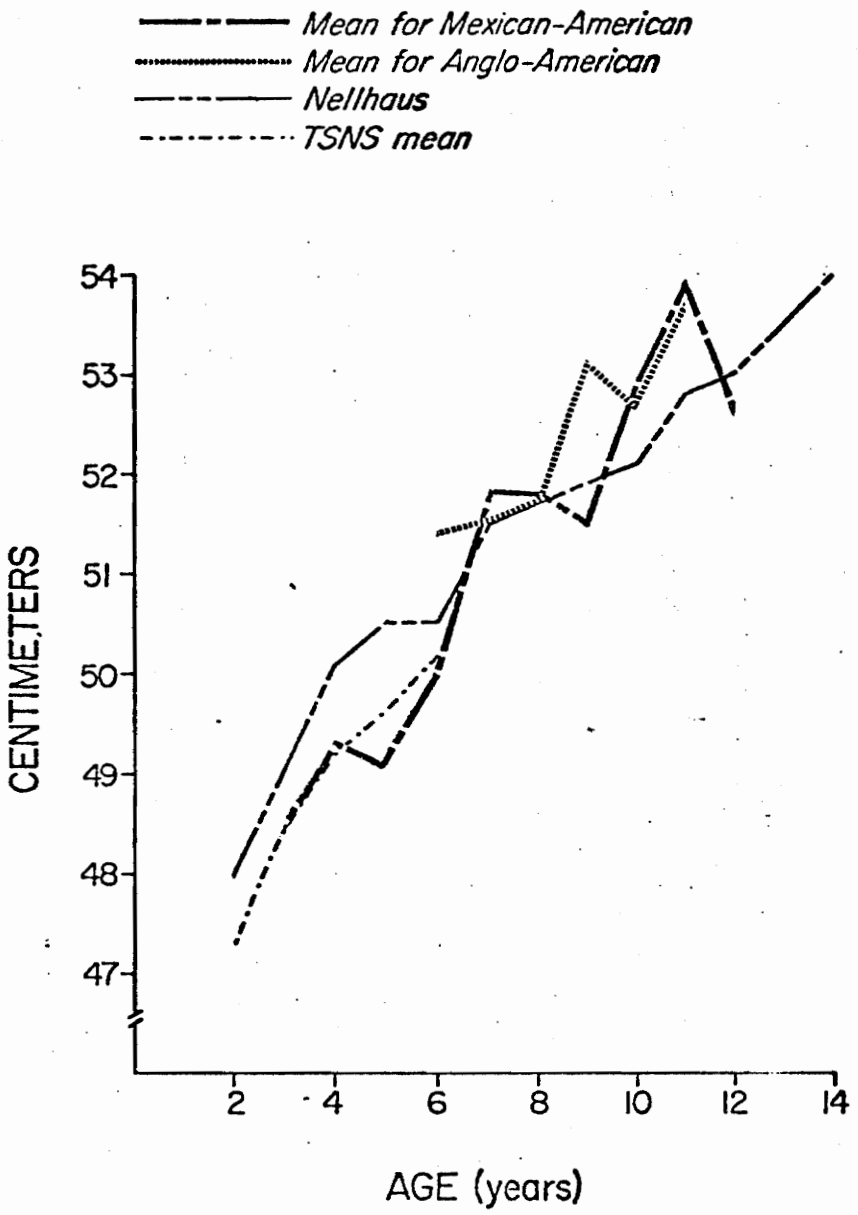

FIGURE 18. Mean head circumference of Mexican-American & Anglo-American females compared to head circumference standards

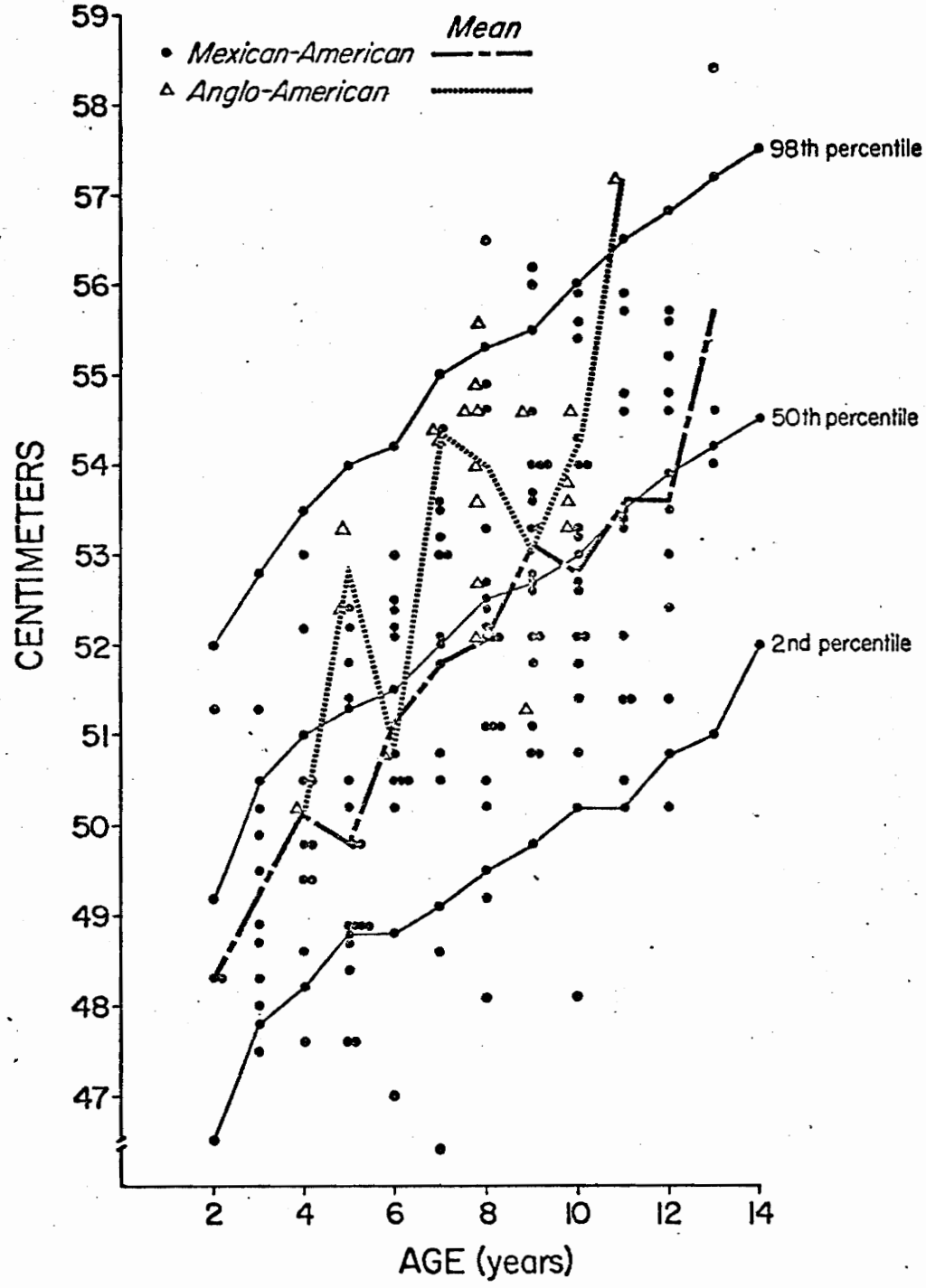

FIGURE 19. Head circumference of Mexican-American and Anglo-American males compared to Nellhaus Standard

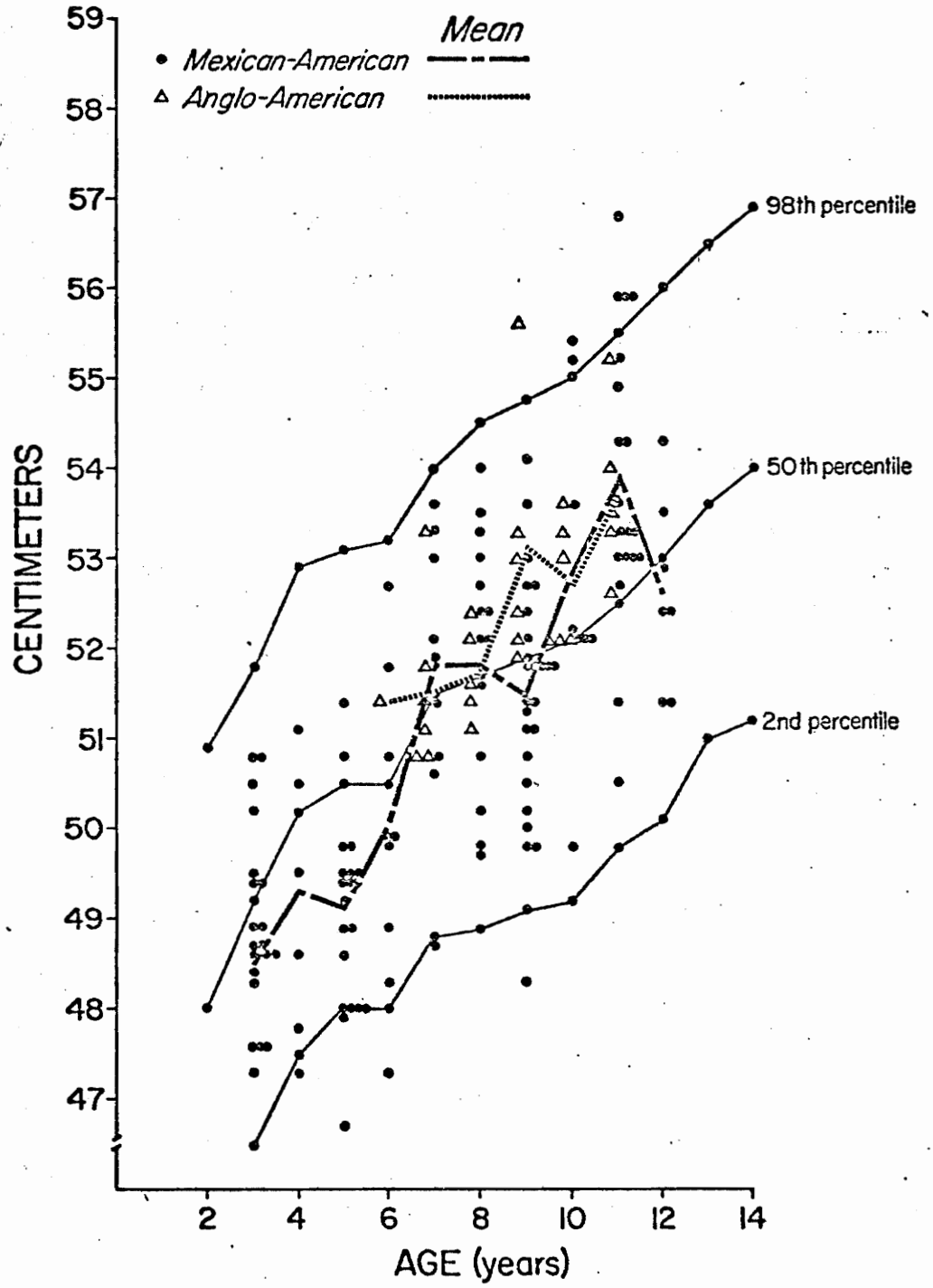

FIGURE 20. Head circumference of Mexican-American and Anglo-American females compared to Nellhaus Standard

standard until 7 years. After 7 years, the Mexican American females caught up to and followed the Nellhaus standard. Figure 19 shows that 9% of the Mexican American males and 4% of the females were below the 2nd percentile for head circumference. Three percent of the Mexican American males and 4% of the females exceeded the 98th percentile. For the Anglo American children, none fell below the 2nd percentile in head circumference and 4% of the males and 3% of the females were above the 98th percentile.

Tables 39 and 40 present the mean arm circumference in cm $\pm$ S.D. for Mexican American and Anglo American children. Figures 21 and 22 compare the mean arm circumference to several standards (168-171). Figures 23 and 24 show the distribution of the actual measurements about the mean.

The arm circumference has been proposed as an easily performed measure of protein-calorie status (200). Several standards have been proposed for arm circumference, but none are generally accepted (168-171). Since no percentile distributions are yet available for arm circumference, and since the arm circumference is still largely an experimental anthropometric measurement, the scatter diagrams in Figures 23 and 24 are not compared to percentile standards. Figures 21 and 22 show that the mean arm circumferences for Mexican American boys and girls were above the proposed standards. The decline in arm circumference for both Mexican American and Anglo American girls over age 11 is probably due to the small size of both samples (7 Mexican American girls and 3 Anglo American girls). One would expect arm circumference to continue increasing in females of this age.

TABLE 39

Mean ± S.D. Arm Circumference (cm) of Mexican American Children

	Males		Females	
Age (Yrs.)	Absorber	Malabsorber	Absorber	Malabsorber
2	15.9*	16.5*	-	-
3	16.1± 1.0	17.8± 0.6	16.5± 1.1	16.8± 0.3
4	17.0± 1.3	24.8*	17.2± 0.9	15.9*
5	17.3± 2.2	16.7± 1.2	17.1± 1.0	19.9± 4.3
6	17.0± 0.5	17.3± 1.4	17.7± 1.0	17.0± 1.7
7	20.1± 3.5	17.2± 1.1	17.7± 1.9	17.7± 0.7
8	19.1± 3.3	17.5± 1.0	18.0± 1.5	18.9± 1.8
9	19.6± 2.0	18.5± 2.4	19.9± 2.3	19.3± 2.5
10	23.0± 4.0	18.4± 1.9	19.4± 2.6	21.0± 2.1
11	21.0± 3.5	22.9± 2.2	22.3± 3.4	20.9± 2.3
12	20.2± 1.3	22.1± 3.7	19.0± 2.0	21.2± 0.2
13	21.6*	23.0± 4.2	-	-

TABLE 40

Mean ± S.D. Arm Circumference (cm) of Anglo American Children

Age (Yrs.)	Males		Females	
	Absorber	Malabsorber	Absorber	Malabsorber
4	16.5*	-	-	-
5	18.4± 0.1	-	-	-
6	-	16.5*	19.4*	-
7	20.5± 3.4	-	19.6± 1.0	-
8	20.5± 2.5	19.0*	19.3± 1.3	-
9	21.1± 4.2	-	21.0± 2.5	-
10	20.8± 2.0	22.9*	21.7± 3.6	22.2*
11	24.8*	-	19.9± 2.4	-

* Single observation

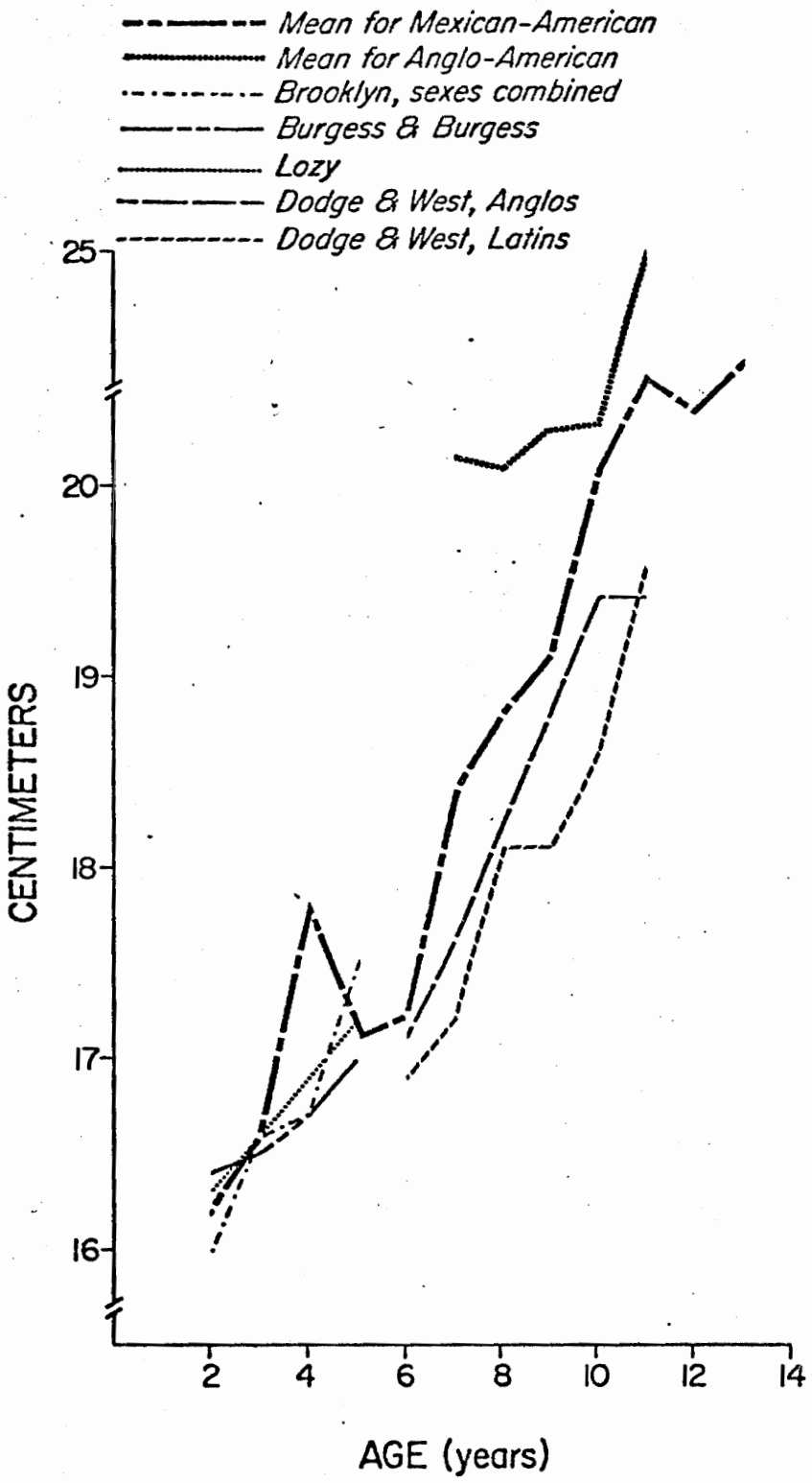

FIGURE 21. Mean arm circumference of Mexican-American & Anglo-American males compared to arm circumference standards

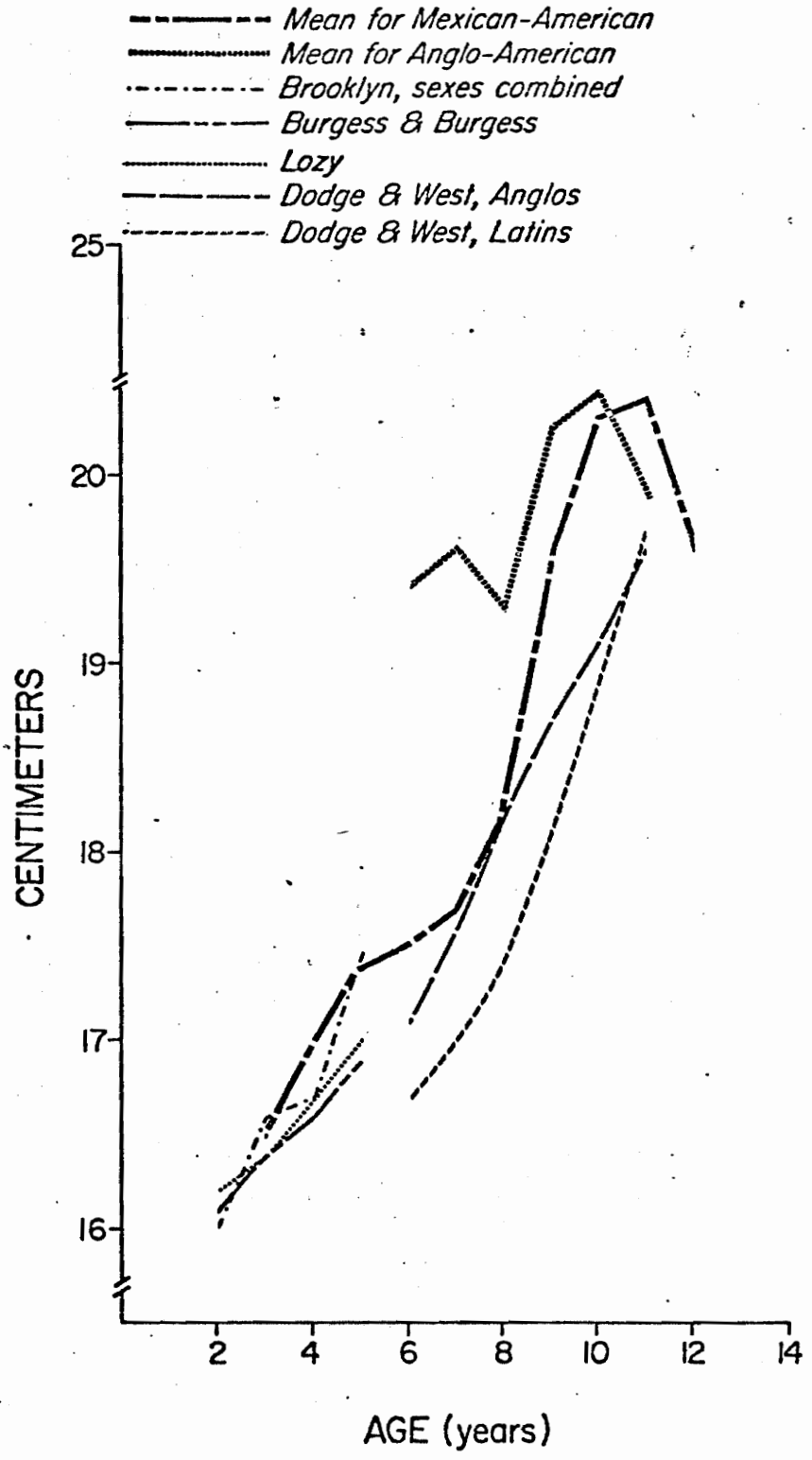

FIGURE 22. Mean arm circumference of Mexican-American & Anglo-American females compared to arm circumference standards

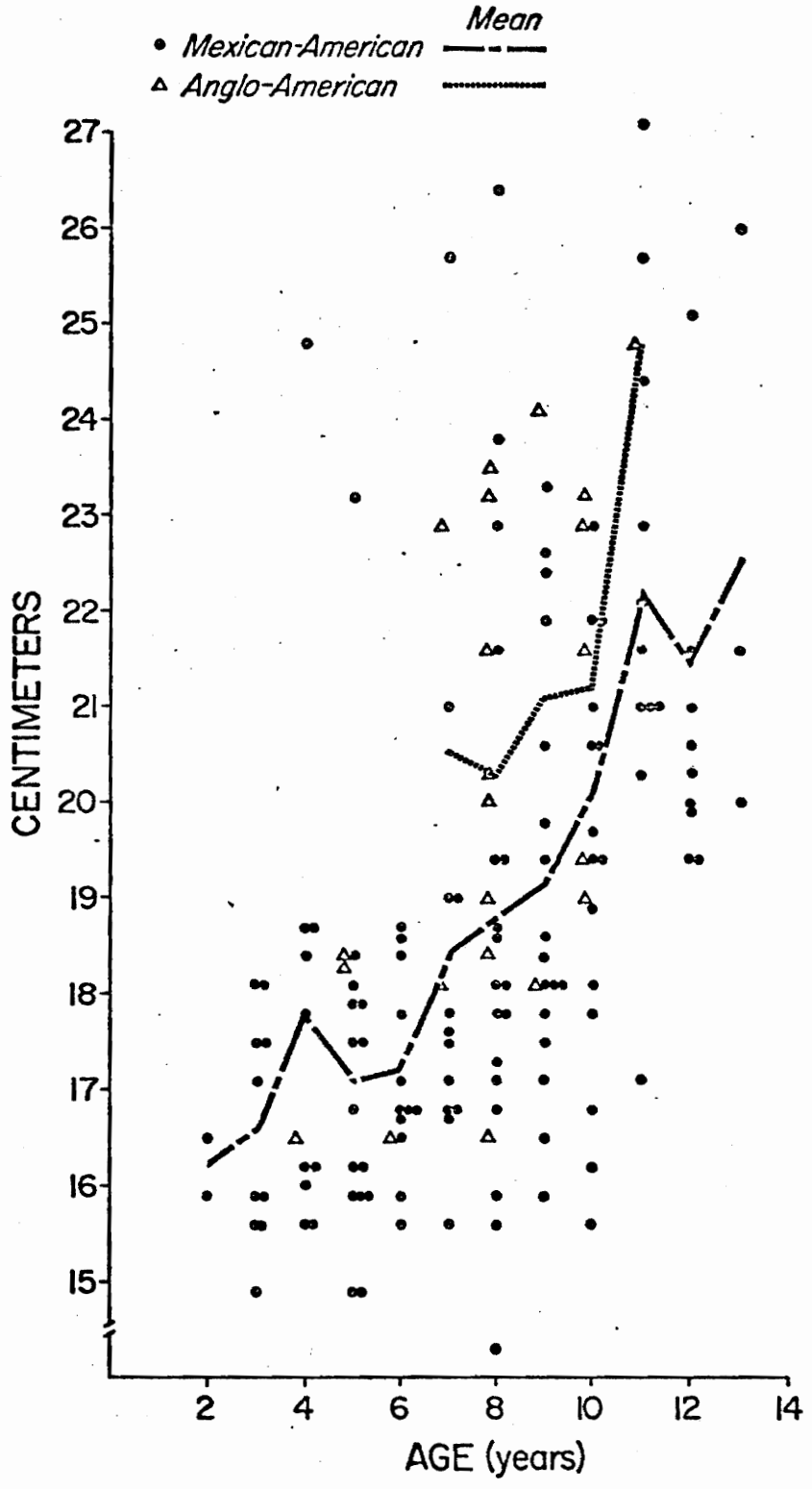

FIGURE 23. Arm circumference of Mexican-American and Anglo-American males

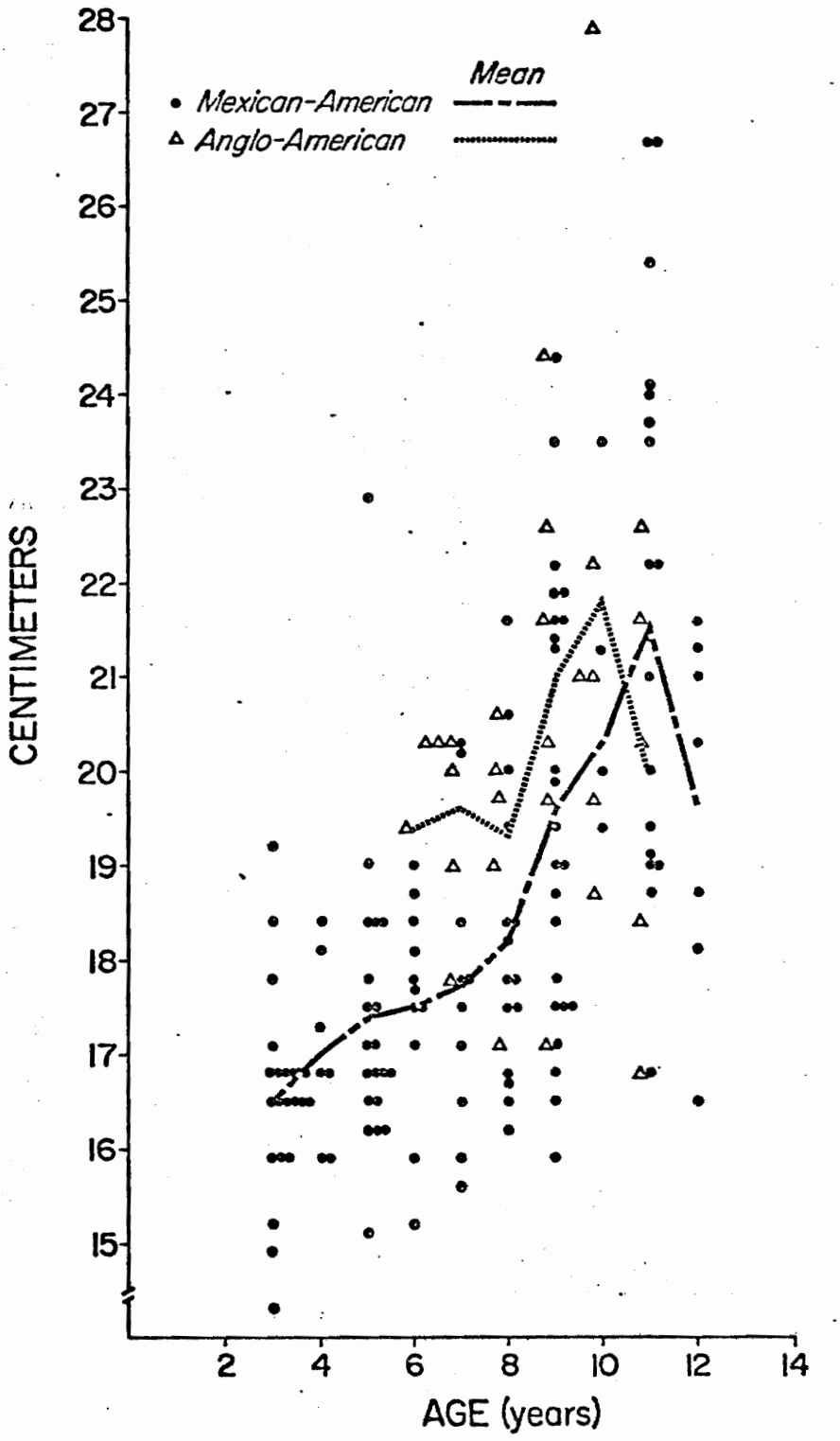

FIGURE 24. Arm circumference of Mexican-American and Anglo-American females

Tables 41 and 42 show the mean triceps skinfold thickness in mm ± S.D. for Mexican American and Anglo American children. Figures 25 and 26 compare the means to the 5th, 50th, and 95th percentiles of the National Health Survey (172), and also show the distribution of the observed skinfold measurements about the mean. The triceps skinfold means were not compared to several other standards because the standards proposed by Tanner and Whitehouse (201), Corbin (202), Montoye et al. (203), and Pett and Ogilvie (204) are so erratic. The mean triceps skinfold of Mexican American males was above the 50th percentile, but 4% of the boys were below the 5th percentile and 6% were above the 95th percentile. The mean triceps skinfold of Mexican American females closely followed the 50th percentile, and 1% of the females were below the 5th percentile and 2% were above the 95th percentile. Of the Anglo American children none were below the 5th percentile and 9% of the boys and 7% of the girls were above the 95th percentile.

Correlations between lactose tolerance test, anthropometric, and dietary results

Tables 43 and 44 show the correlation matrices for the data derived from the LTT, 5 anthropometric measurements, and analysis of the 24-hour recalls for 5 nutrient factors for the Mexican American and Anglo American children.

In the Mexican American children (Table 43) age was negatively correlated with vitamin A intake and with the maximum blood glucose rise. Age was positively correlated with the number of symptoms experienced after the LTT, and with all 5 anthropometric measurements. Each of the dietary factors was significantly positively correlated with each of the

TABLE 41

Mean ± S.D. Triceps Skinfold Thickness (mm) of Mexican American Children

	Males		Females	
Age (Yrs.)	Absorber	Malabsorber	Absorber	Malabsorber
2	7*	9*	-	-
3	8±1	9±2	10±2	9±1
4	9±2	19*	9±1	8*
5	8±3	8±2	9±2	13±7
6	8±1	8±2	9±2	9±2
7	11±7	7±2	10±5	9±2
8	10±5	8±2	10±3	12±4
9	9±4	8±4	12±4	11±5
10	15±7	7±3	10±4	12±5
11	11±6	13±6	16±6	11±3
12	7±4	10±7	10±3	12±1
13	7*	13±6	-	-

* Single observation

TABLE 42

Mean ± S.D. Triceps Skinfold Thickness (mm) of Anglo American Children

	Males		Females	
Age (Yrs.)	Absorber	Malabsorber	Absorber	Malabsorber
4	7*	-	-	-
5	10±1	-	-	-
6	-	5*	9*	-
7	12±5	-	13±3	-
8	11±4	9*	10±3	-
9	12±10	-	13±4	-
10	9±3	14*	14±6	18*
11	16*	-	10±3	-

* Single observation

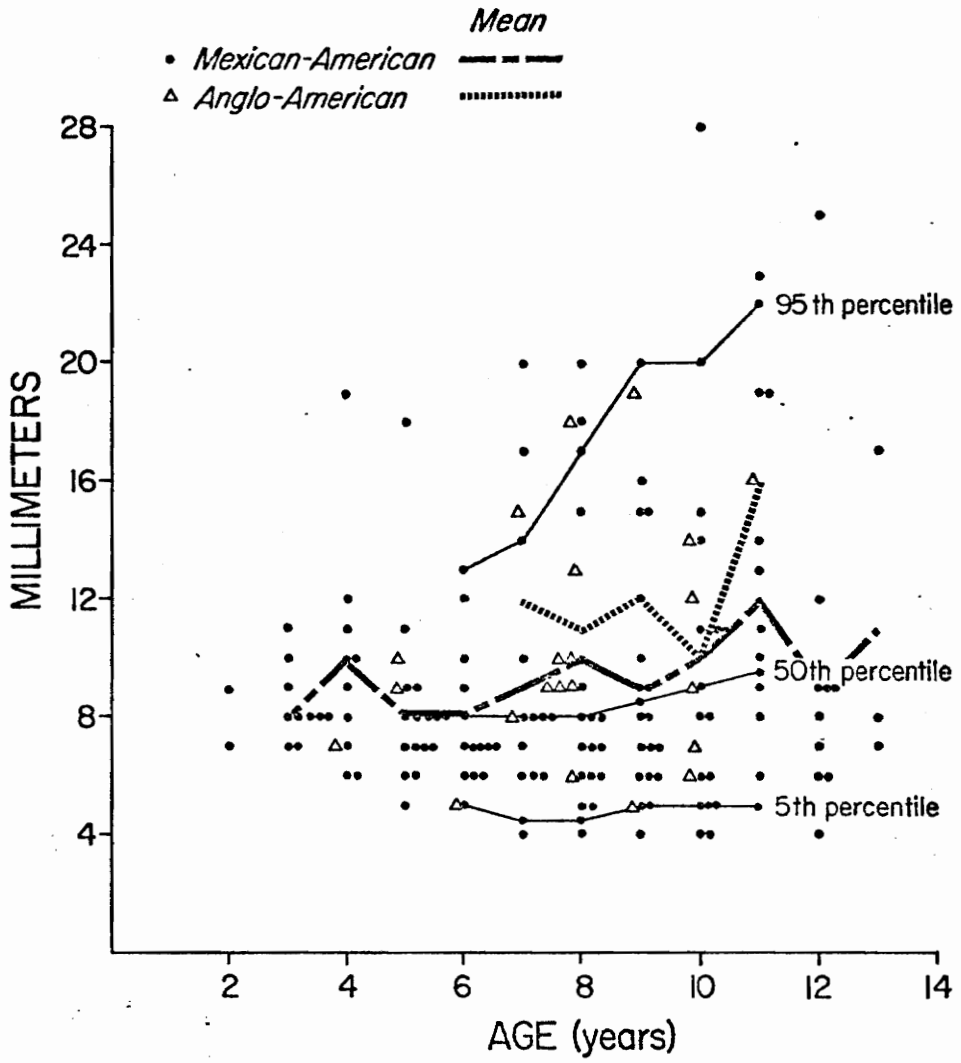

FIGURE 25. Triceps skinfold thickness of Mexican-American and Anglo-American males compared to National Health Survey

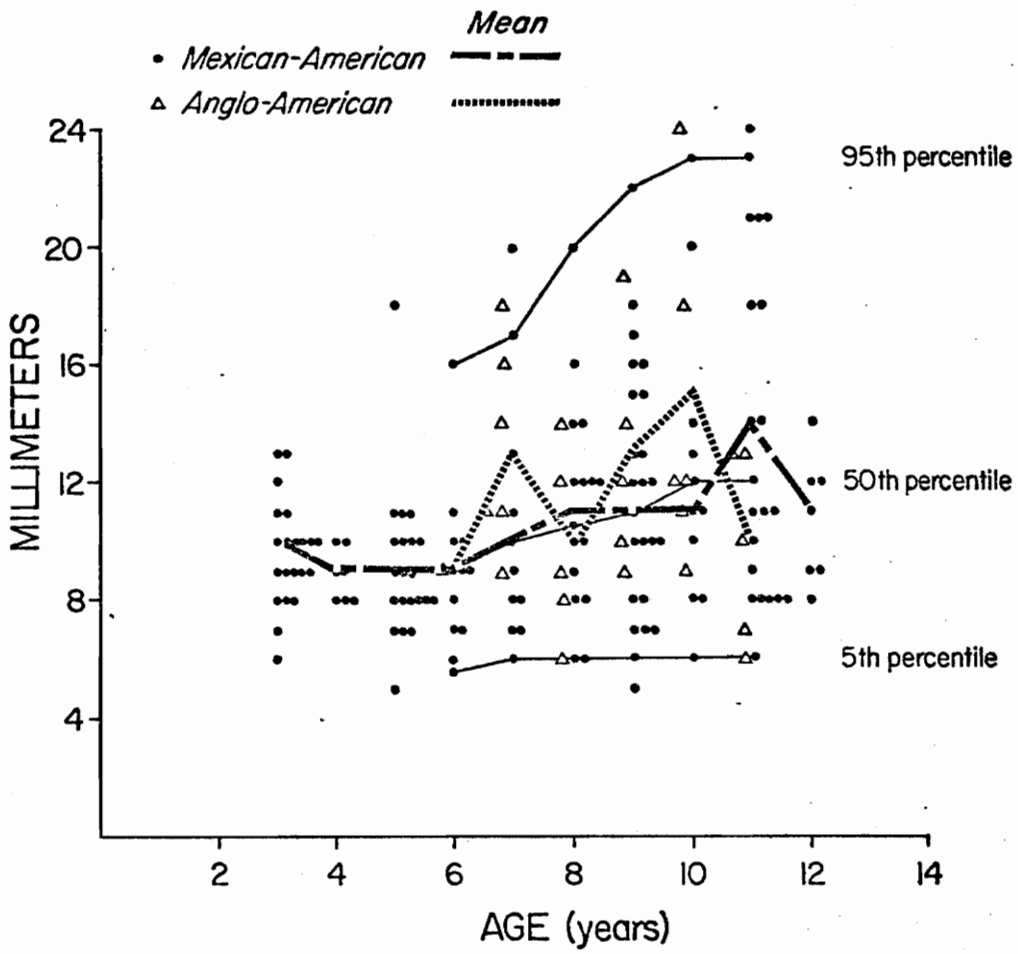

FIGURE 26. Triceps skinfold thickness of Mexican-American and Anglo-American females compared to National Health Survey

TABLE 43

Correlation Matrix for Mexican American Children

	Age	Protein	Calories	Calcium	Riboflavin	Vit. A	# Symptoms
Age	1.000	0.048	0.013	-0.039	-0.197	-0.327	0.437
Protein	0.048	1.000	0.894	0.732	0.681	0.316	0.103
Calories	0.013	0.894	1.000	0.758	0.665	0.353	0.073
Calcium	-0.039	0.732	0.758	1.000	0.740	0.412	0.102
Riboflavin	-0.197	0.681	0.665	0.740	1.000	0.672	-0.004
Vit. A	-0.437	0.316	0.353	0.412	0.672	1.000	-0.114
# Symptoms	0.437	0.103	0.073	0.102	-0.004	-0.114	1.000
Initial Glucose	0.169	0.016	0.023	-0.005	-0.116	-0.147	0.043
Max.Glucose Rise	-0.274	-0.052	-0.009	0.011	0.047	0.121	-0.405
Weight	0.800	0.125	0.084	0.044	-0.079	-0.168	0.338
Height	0.932	0.070	0.033	0.000	-0.163	-0.264	0.401
Head Circ.	0.708	0.030	0.010	-0.029	-0.162	-0.212	0.223
Arm Circ.	0.574	0.169	0.141	0.116	0.010	-0.084	0.250
Triceps Skinfold	0.215	0.124	0.142	0.123	0.072	0.012	0.110

TABLE 43 CONTINUED

	Initial Glucose	Max. Glucose Rise	Weight	Height	Head Circ.	Arm Circ.	Triceps Skinfold
Age	0.169	-0.274	0.800	0.932	0.708	0.574	0.215
Protein	0.016	0.052	0.125	0.070	0.030	0.169	0.124
Calories	0.023	0.009	0.084	0.033	0.010	0.141	0.142
Calcium	-0.005	0.011	0.044	0.000	-0.029	0.116	0.123
Riboflavin	-0.116	0.047	-0.079	-0.163	-0.162	0.010	0.072
Vit. A	-0.147	0.121	-0.168	-0.264	-0.212	-0.084	0.012
Symptoms	0.043	-0.405	0.338	0.401	0.223	0.250	0.110
Initial Glucose	1.000	-0.229	0.123	0.138	0.093	0.091	0.037
Max.Glucose Rise	-0.229	1.000	-0.238	-0.240	-0.149	-0.201	-0.071
Weight	0.123	-0.238	1.000	0.908	0.803	0.895	0.607
Height	0.138	-0.240	0.908	1.000	0.803	0.696	0.349
Head Circ.	0.093	-0.149	0.803	0.803	1.000	0.658	0.400
Arm Circ.	0.091	0.201	0.895	0.696	0.658	1.000	0.786
Triceps Skinfold	0.037	-0.071	0.607	0.349	0.400	0.786	1.000

TABLE 44

Correlation Matrix for Anglo American Children

	Age	Protein	Calories	Calcium	Riboflavin	Vit. A	# Symptoms
Age	1.000	0.043	0.055	0.139	0.169	0.146	0.147
Protein	0.043	1.000	0.880	0.845	0.844	0.341	0.280
Calories	0.055	0.880	1.000	0.768	0.809	0.366	0.273
Calcium	0.139	0.845	0.768	1.000	0.932	0.427	0.448
Riboflavin	0.169	0.844	0.809	0.932	1.000	0.447	0.423
Vit. A	0.146	0.341	0.366	0.427	0.447	1.000	0.203
# Symptoms	0.147	0.280	0.273	0.448	0.423	0.203	1.000
Initial Glucose	-0.089	-0.123	-0.071	-0.196	-0.169	-0.415	-0.096
Max.Glucose Rise	-0.056	-0.053	0.017	-0.031	-0.011	-0.023	-0.160
Weight	0.592	0.123	-0.044	0.071	0.039	-0.007	-0.047
Height	0.375	0.069	-0.076	0.100	0.092	-0.103	-0.124
Head Circ.	0.445	0.096	-0.024	0.018	-0.003	-0.109	0.070
Arm Circ.	0.393	0.238	0.087	0.211	0.190	0.043	0.107
Triceps Skinfold	0.160	0.283	0.162	0.268	0.250	0.137	0.030

TABLE 44 CONTINUED

	Initial Glucose	Max. Glucose Rise	Weight	Height	Head Circ.	Arm Circ.	Triceps Skinfold
Age	-0.089	-0.056	0.592	0.375	0.445	0.393	0.160
Protein	-0.123	-0.053	0.123	0.069	0.096	0.238	0.283
Calories	-0.071	0.017	-0.044	-0.076	-0.024	0.087	0.162
Calcium	-0.196	-0.031	0.071	0.100	0.018	0.211	0.268
Riboflavin	-0.169	-0.011	0.039	0.092	-0.003	0.190	0.250
Vit. A	-0.415	-0.023	-0.007	-0.103	-0.109	0.043	0.137
# Symptoms	-0.096	-0.160	-0.047	-0.124	0.070	0.107	0.030
Initial Glucose	1.000	-0.264	0.027	-0.021	0.076	-0.010	-0.125
Max.Glucose Rise	-0.264	1.000	-0.184	-0.016	-0.258	-0.148	0.021
Weight	0.027	-0.184	1.000	0.569	0.723	0.866	0.538
Height	-0.121	-0.016	0.569	1.000	0.466	0.437	0.325
Head Circ.	0.076	-0.258	0.723	0.466	1.000	0.534	0.141
Arm Circ.	-0.010	-0.148	0.866	0.437	0.534	1.000	0.787
Triceps Skinfold	-0.125	0.021	0.538	0.325	0.141	0.787	1.000

other dietary factors, but not with any of the anthropometric measurements or LTT results. The number of symptoms after the LTT was negatively correlated with the blood glucose rise in the LTT and positively correlated with the height. The fasting blood glucose levels were not significantly correlated with any other factor. Each of the 5 anthropometric measurements had a significant positive correlation with the other anthropometric measurements.

In the Anglo American children (Table 44) age was positively correlated with symptoms and negatively correlated with the maximum blood glucose rise. The magnitude of these correlations was much smaller than in the Mexican American children. Each of the dietary factors was significantly positively correlated with each other dietary factor. Each of the 5 anthropometric measurements had a significant positive correlation with the other anthropometric measurements.

DISCUSSION

The Mexican Americans are the second largest minority group in the U.S., now numbering over 5 million (173). Most Mexican Americans live in the states of California, Texas, New Mexico, Colorado and Arizona. The population of the city of San Antonio, Texas is over 50% Mexican American, but this does not represent a homogeneous group. Actually, 7 different groups compose the Mexican American population of San Antonio, these include: native Creoles (descendents of Spanish colonial families), native Mestizos, and native Indians whose families have lived in the U.S. since before 1836, and the Mexican Creoles, Mexican Mestizos, and Mexican Indians all of whom have migrated to the U.S. in the twentieth century (174). Also often mistakenly included in the Mexican American group because of Spanish surname and because they usually speak Spanish are the Filipinos, Cubans, Puerto Ricans, Central and South Americans (174). However, the Mexican Americans are generally considered to form one ethnic group (175).

The Mexican Americans have a greater neonatal and infant death rate, and greater incidences of pneumonia and tuberculosis than the general population. But, there is no evidence that the Mexican Americans suffer from a higher incidence of total illness or from a greater prevalence of chronic disease than the general population (173). However, it has been shown that Mexican American children have poorer diets than Anglo American children (164,189,190,191), and that they are generally shorter and lighter in weight than Anglos (164,189,191,205). This study was undertaken to determine the lactose tolerance of healthy Mexican American children. These findings were correlated with selected dietary

and anthropometric parameters to assess any possible effects of lactose malabsorption on diet and growth.

Lactose tolerance test

In recent months the LTT has received criticism with regard to the large lactose load employed (50 g, the equivalent of one quart of milk) on the grounds that most people do not consume one quart of milk at a sitting. However, the LTT is not designed to determine how much milk an individual can drink, but rather to determine indirectly the amount of lactase present in the intestinal mucosa. Numerous studies have shown that a low blood glucose rise (less than 20 or 25 mg per 100 ml) after ingesting 50 g of lactose correlates well with low intestinal lactase activity as determined by assay of jejunal biopsy tissue (11,12, 19,67,78,85,88,114,118,130). The LTT is at best an approximation and is by no means a quantitative determination of the enzymatic hydrolysis and absorption of lactose. However, the LTT does provide information on the amount of lactase present, and when judiciously evaluated, the LTT is of great value in clinical work as well as in population surveys.

Two authors have reported that in 10 - 40% of lactase deficient patients an LTT employing capillary blood was normal (176,177). The LTT employing capillary blood eliminated falsely flat curves in normal subjects (176,177). It is therefore preferable to base a diagnosis of lactose intolerance on the occurrence of symptoms after the LTT coupled with the results of the capillary blood glucose rise than on either independently (177,178).

The incidence of lactose malabsorption increased with age in both the Mexican American and Anglo American children (Figure 1, Tables 2,3,

6,7,13,14). Newcomer and McGill (144) and Jussila (90) found that age did not correlated with lactose malabsorption in adults, but Paige et al. (132,133) in Peruvian children, Elliott et al. (114) in Australian Aboriginal children, Gilat et al. (105) in Jewish children, and Sahi et al. (89) in Finnish children have shown that as age increases in children so does the incidence of lactose malabsorption.

The tendency to have decreasing lactase levels with increasing age is probably under genetic control, but several other factors may hasten the appearance of lactose malabsorption. Stoopler et al. (123) in explaining the reversibility of lactose tolerance in Jamaican children under 4 years of age suggest that transient environmental insults may temporarily suppress intestinal lactase activity. These environmental influences include parasitic infestation and subclinical malabsorption of unknown etiology which is found in adults and children in tropical countries and associated with histological abnormalities. Desai et al. (54) and Gracey and Burke (48) suggest that along with parasitic infestation and dietary insults, frequent bacterial infections and infectious diarrhea may be related to low lactase levels. According to Dr. A. W. Pierce, Jr., M.D. (personal communication) the Mexican American children of San Antonio have a high incidence of diarrheal diseases which are frequently bacterial in origin. They also have higher incidences of amoebic abcesses, amoebic dysentery, Ascaria and Giardia than are found in the general population (Dr. A. W. Pierce, Jr., M.D., personal communication). We therefore cannot rule out the contribution of parasitic and diarrheal diseases to the development of lactose malabsorption in Mexican American children in San Antonio.

The incidence of symptoms occurring after the LTT increased with age in both the Mexican American and Anglo American children. This could be the result of better reporting in the older children, or of an actual decrease in intestinal tolerance to the lactose load, or a combination of both. An increase with age in the number of symptoms in children was also noted by Sahi et al. (89) in Finnish children and by Paige et al. (133) in Peruvian children. In the Peruvian children, none of those under 3 years had symptoms, and 75% of those with flat curves had symptoms. In our Mexican American children 70% of the male and 54% of the female malabsorbers had symptoms after the LTT for a total of 68% of the malabsorbers with symptoms. Sahi et al. (89) suggest that symptoms are more common with age because the colon of healthy children does not easily react with increased peristalsis to the increased intraintestinal volume of fluid caused by unabsorbed lactose. Christopher and Bayless (83) suggest that differences in clinical tolerance to lactose may be related to:

1. the rate of gastric emptying,
2. the speed of transit of materials through the intestine,
3. the net secretory response to the osmotic load,
4. the intestinal motor response to an increased fluid load, and
5. the general "irritability" of the small bowel and colon.

Figures 10, 11 and 12 and the accompanying statistical analyses show that there was no difference in the incidence of lactose tolerance between Mexican American males and females. Gilat et al. (106) reported a greater incidence of lactose intolerance in Palestinian Arab women than men, however most investigators have found no difference in lactose

tolerance between men and women (63,90,125) or between boys and girls (89, 123).

Table 15 shows that the association of symptoms with lactose malabsorption was the same in both Mexican American and Anglo American children, and that this association was statistically significant. This conclusion was expected because of the cause and effect relationship between low lactase levels and gastrointestinal symptoms. Although the association of symptoms with the LTT was statistically the same, the Mexican American malabsorbers had symptoms more often than the Anglo malabsorbers. This may be related to the milk consumption of the children. Since the Anglo American children drank significantly more milk than the Mexican American children they may have developed a tolerance to unabsorbed lactose and the products of its fermentation by colonic bacteria (145).

The Mexican American children had a greater incidence of lactose malabsorption and symptoms than the Anglo American children (Tables 16 and 17). These differences are statistically significant and we can therefore conclude that we are dealing with two distinct groups on the basis of lactose tolerance.

In both the Anglo American and Mexican American groups, some children with presumably normal lactase levels as determined by the LTT had symptoms after the LTT and some children with low lactase levels had no symptoms. This same phenomenon has been described by several authors (72,80,88,129,131). Sheehy and Anderson (80) estimate that one in every six persons with low lactase levels do not have symptoms during an LTT. Alzate et al. (129) and Newcomer and McGill (72) suggest that

the cause of these symptomless low curves may be:

1. exceedingly rapid gastric emptying causing a blood glucose peak before the 15 min blood sample,
2. delayed gastric emptying,
3. lack of correlation in the times of sampling and the maximum lactose absorption, and
4. asymptomatic lactase deficiency.

Haemmerli et al. (88) propose that patients with low normal blood glucose curves who complain of symptoms after the LTT or after milk ingestion may later become lactase deficient. Continued milk ingestion which contributes to general health and to the health of the intestinal mucosa, and avoidance of parasitic and diarrheal infections may provide some protection against the occurrence of GI symptoms after the LTT (54, 141,145). Development of tolerance to milk may be due to small quantities of lactase dealing satisfactorily with ingested lactose, or to the development of a tolerance to unabsorbed lactose and the products of its breakdown by colonic bacteria (145).

The Mexican American and Anglo American children showed similar patterns in their blood glucose rises during the LTT (Table 18), with the lactose malabsorbers tending to be later in their peak serum glucose rises than the absorbers. In the Mexican American malabsorbers 5 children (5%) showed no rise or a slight decrease in serum glucose during the LTT. Kattamis et al. (94) reported a similar observation in Greek children, with 40% of their group showing glucose levels lower than fasting levels during the LTT. Bolin et al. (110) in a group of hospital patients in Singapore found that 20% of them had a peak glucose

rise at 15 min. Newcomer and McGill (72) found that 50% of their healthy subjects had a peak glucose rise at 15 min. McGill and Newcomer (176) and Stoopler et al. (123) both found lactose malabsorbers to have peak serum glucose rises later than lactose absorbers.

In the Mexican American malabsorbers the most frequently reported symptoms were abdominal cramps, bloating and diarrhea, and in the absorbers cramps, bloating and flatulence. Only 2 of the 4 Anglo American malabsorbers had symptoms: one reported loose stool, the other bloating, cramps and diarrhea. In the Anglo absorbers, the most frequently reported symptoms were bloating, cramps and diarrhea. Rotthauwe et al. (107) reported that in Arab adults the most common symptoms during an LTT were borborygmi, flatulence and diarrhea. Dunphy et al. (81) found in 12 adults with isolated lactase deficiency that the most common symptoms were diarrhea, cramps, and bloating. As previously discussed, children do not seem to react with diarrhea after a lactose load as frequently as adults.

Caloric, nutrient, and milk intakes

In obtaining information on the children's intake of calories, milk nutrients, and milk the 24-hour dietary recall method was used. The mothers and teachers of children 2-5 years were questioned on the children's diet during the previous 24 hours, while those children 6 years and older were interviewed directly. A child's ability to give accurate information on his food intake depends on his understanding the concept of "yesterday", a knowledge of the names of foods, a good memory, a sufficiently long attention span, and a willingness to cooperate (180). Emmons and Hayes (180) found that the 24-hour recall

method met these constraints for elementary school children.

Bransby et al. (181) found that children aged 10 to 15 years gave as accurate information on their previous day's intake of food as was obtained from weighing the foods consumed or recording the amounts consumed in household measures. Trulson (182) found that the 24-hour recall and interviews on usual food patterns provided similar information on the consumption of protein, milk, and foods high in vitamin A. Trulson does not state how much help the mothers of the 47 children gave during the interviews. Young et al. (183) using a 7-day dietary record method showed that young children 4 to 9 years had a better nutrient intake compared to the RDA than children 10 to 15 years old. However, since the mothers kept the records of children under 10 years, it is difficult to know whether the younger children had better diets or whether the difference was due to better reporting.

For groups, it is generally agreed that 24-hour recalls provide valid information on usual nutrient intake (184,185). Anderson and Sandstead (186) and Young et al. (187) believe that the nutritive intake of a group is best obtained through a large number of one day records.

Emmons and Hayes (180) found that the ability of elementary school children to recall correctly the foods eaten improved with age. Children in fourth grade correctly remembered 80.6% of the foods in their school lunch while children in first grade remembered an average of 60.5% of the food items in their school lunch. Emmons and Hayes (180) also found that primary items were easier for the children to remember than secondary items. Primary items consisted of foods such as spaghetti and meat

balls, and frankfurters; secondary items such as bread and butter were forgotten because they were considered a staple that accompanied the tray.

Meredith et al. (188) found that 71% of 94 children ages 9 to 18 years underestimated the quantity of food eaten, while Young et al. (187) found that children overestimated their serving sizes.

Analysis of the 24-hour recalls of the Mexican American children revealed that there were no differences between the lactose absorbers and malabsorbers in the intake of calories, protein, riboflavin, vitamin A or calcium. There were also no differences between Mexican American males and females in the intake of calories or milk nutrients.

Comparing the Mexican American and Anglo American children, the Mexican American children reported consuming fewer calories and less protein, riboflavin, vitamin A and calcium than the Anglo American children. The only difference that was statistically significant was calories. The 10-14 year old Mexican American children reported the poorest diets, the mean vitamin A intake being only 40% of the RDA and mean calcium intake 42-50% of the RDA. These differences in caloric and nutrient intakes between Mexican American and Anglo American children may be economic and cultural in origin. The San Antonio Independent School District from which the Mexican American children were drawn is largely lower-income, while the Northside Independent School District is largely middle-income. The Ten-State Nutrition Survey (164) found that economic level correlates with nutrient intakes. The low caloric intakes reported by both the Mexican American and Anglo American children were probably attributable in part to the children's forgetting

to report high-caloric foods that are secondary items in the diet (180).

Several authors have found the diets of Mexican Americans to be lower in certain nutrients than the diets of Anglo Americans in the same community. Lantz and Wood (189) found the diets of "Spanish American" adolescents living in New Mexico to be significantly lower in energy, protein, fat, calcium and riboflavin than Anglo children. Hacker and Miller (190) found the diets of Mexican American children to be deficient in protein, calcium, riboflavin and vitamin C. The results of the Ten-State Nutrition Survey (164,191) indicated that in general the intakes of calories, protein, calcium, vitamin A and riboflavin were lower in Spanish American infants and young children (0 to 36 months) than in White children. For adolescents (10 to 16 years) the mean intake of calories, protein, calcium, and vitamin A were lower in the Spanish Americans than Whites; riboflavin showed little consistent relationship between mean intakes and ethnic group.

The finding of skin lesions related to nutritional deficiencies in the population surveyed by the Ten-State Nutrition Survey in Texas (191) emphasizes the point that although mean intakes of nutrients for a group may be close to the recommended amounts there are individuals who may fall far below or far above the mean. Those individuals far below the recommended will be those most at risk nutritionally. In our Mexican American children, the range in nutrient intakes was very large for all 5 dietary factors examined.

The food habits of Mexican Americans give insight into the low dietary intakes of some nutrients. The staple foods of Mexican American families on low or marginal incomes are beans and tortillas (156).

When more money is available, a more varied diet is consumed including meat, eggs, fruits and vegetables (156). Chile peppers are a staple, and are put in a basic sauce (See Appendix B) which is used liberally in food preparation and as a condiment (156). The vegetables in salads are limited to lettuce and tomato, and other vegetables are used infrequently (156). Tomatoes and chiles are the most dependable dietary sources of vitamins A and C (156,192). Children generally do not acquire a taste for chile until they are 10 or 11 years old (190), and therefore would not benefit from the chile's vitamin content. In the past when corn tortillas were made at home, they supplied 500 mg of calcium for every 280 g of tortilla (193). In San Antonio, corn tortillas are usually purchased. Commercially made corn tortillas are a poor source of calcium because the lime water used in treating the corn is almost completely removed in the processing (Mrs. Rosemary Casey, Public Health Nutritionist, Bexar County, Texas, personal communication). If tortillas are prepared at home, the easier wheat flour tortillas are usually made (Appendix B).

Milk is not a common beverage in Mexico because of its scarcity and minimal refrigeration facilities. Although milk is readily available in the U.S., many Mexican American families use little or no milk (156,194). Our Mexican American children consumed an average of one glass of milk a day, usually obtained through the school breakfast or lunch program. This was significantly less than the average 2 glasses a day consumed by the Anglo American children.

There was no difference in milk consumption between the lactose absorbers and malabsorbers in either ethnic group. Not everyone with low

lactase levels will be aware of symptoms after one glass of milk (2). Some people with low lactase levels note symptoms after consuming a small portion of a glass of milk, others only after 3 or 4 glasses (2). No difference in milk consumption between lactose absorbers and malabsorbers has been found in Jewish adults (104), healthy Caucasian adults (144), tropical sprue patients in India (54), Palestinian Arabs (106), Jamaican children (123), and Orientals residing in the U.S. (139). Significant differences in milk consumption between lactose absorbers and malabsorbers were found in hospital patients (196), Peruvian children (133), and Arabs (107). Paige et al. (121) found that lactose intolerance does not appear to be the main factor in milk rejection in White children, but there was an association between poor milk consumption, lactose-induced symptoms and lactose malabsorption in Black children.

Milk consumption is influenced by many factors including income, family size and composition, price of milk, the individual's age, sex, nationality, race, health, and educational level (2). Physiological intolerance to lactose may be another factor to be considered in low milk consumption (2,121). Cheese is consumed more frequently than milk by Mexican American adults (156,195). Although desserts are not a traditional part of Mexican meals, ice cream is a popular food (156).

None of the Anglo American children recognized having symptoms after milk ingestion, but 15% of the Mexican American lactose absorbers and 23% of the malabsorbers reported having symptoms after milk. Zografos et al. (95) in Greek children found that 78% of the malabsorbers and 9% of the absorbers reported symptoms after drinking milk. Kattamis et al. (94) found no correlation between milk intolerance and the results

of the LTT in Greek children, and concluded that milk intolerance on the basis of lactose malabsorption is uncommon in Greek children. In the majority of Mexican American children either the connection between gastrointestinal symptoms and milk ingestion had not been made, or the children tolerated the small amounts of milk in their diets.

Symptoms following milk ingestion may be modified by consuming other foods along with milk (137). Reddy and Pershad (42) showed in lactose intolerant children that intestinal motility was normal when skim milk was given, but was rapid with lactose in water. The children developed symptoms when skim milk was given containing 2 g lactose per kg body weight, but had no symptoms when the same load was given in 2 divided doses.

Lactose malabsorption and growth

If lactose malabsorption were of nutritional significance in the growing child, one would expect the lactose malabsorbers to be smaller in height, weight, and other anthropometric measurements. Some persons with low lactase levels voluntarily curtail milk intake to avoid the symptoms of lactose intolerance (88,105,119,131,135). In growing children, decreased milk consumption might cause a low intake of calories and the nutrients found in significant amounts in milk--protein, calcium, riboflavin, vitamin A. Lactose malabsorption implies a loss of calories in the stool of the lactose malabsorber (143,144), and may be related to decreased calcium absorption (147). Two authors have reported decreased fat and protein absorption in lactose malabsorbers fed meals containing lactose (144,146).

Nutritional anthropometry provides a good tool for the assessment

of mild malnutrition (197). Anthropometric measurements are useful in identifying populations where nutritional inadequacies are reflected in retarded growth and development (164). Greater size for anthropometric measurements at a given age is presumptive evidence of better nutritional status on a group basis, and lesser size suggests relative nutritional inadequacy (164).

Since many studies are criticized on the standards chosen to compare to means for the groups examined, we have used several standards for each measurement. The anthropometric standards compared to our means for Mexican American and Anglo American children were chosen because they are widely used. The Stuart-Meredith standards for height and weight are used extensively by pediatricians in the U.S. and were endorsed in 1971 by the American Academy of Pediatrics for use until other standards are developed (164).

The choice of standards may not be as critical as some believe. Nellhaus (167) has shown that there are no significant racial, national or geographic differences in head circumferences. Purchase (198) and Habicht et al. (199) have shown that there are greater variations in height and weight within an ethnic group due to socio-economic and nutritional factors than between ethnic groups of equivalent socio-economic status. Habicht et al. (198) suggest that height and weight standards chosen to represent optimal growth in preschool children can be drawn from already published studies of well-to-do children, regardless of race or ethnic background because any racial or ethnic effect is small compared with environmental effects.

The results of our measurements on Mexican American lactose ab-

sorbers and malabsorbers showed little consistent difference in the means for height, weight, head circumference, arm circumference and triceps skinfold thickness in either males or females (Tables 42,44,46, 48,50). It therefore appears that if lactose malabsorption does have a nutritional effect, this effect is not of sufficient magnitude to be revealed in the growth patterns of healthy children.

As discussed before, greater size is usually indicative of better nutritional status (164). The dietary analysis revealed no differences in intake of calories and 4 nutrients between lactose absorbers and malabsorbers. This supports the anthropometric findings of no differences between lactose absorbers and malabsorbers. The Mexican American children as a group consumed significantly fewer calories than the Anglo American children, and less calcium, riboflavin, vitamin A and protein. The Mexican American children were generally shorter, lighter, with smaller head and arm circumferences and smaller triceps skinfolds than the Anglo American children.

The means for the Anglo American children for each of the 5 anthropometric measurements were higher than the means for Mexican American children of both sexes. Lantz and Wood (189) found Mexican American children in New Mexico to be shorter and lighter in weight than Anglo children. The Ten-State Nutrition Survey in Texas (164,191) found Mexican American children to be shorter and lighter in weight than Whites. Chase et al. (205) found 18% of Mexican American migrant farm children to be below the 3rd percentile for height, 6% below the 3rd percentile for weight, and 6% below the 3rd percentile for head circumference. In Chase's study (205) 38% of the girls and 34% of the boys were below the

10th percentile in triceps skinfold.

The large numbers of Anglo American children above the 95th percentile for height and weight may be an artifact of the method of recording age. In all cases, age was recorded as the age at last birthday. The Stuart-Meredith standards (206) were constructed using measurements performed within 2 weeks of the age indicated. The children in our study could be from 0 to 12 months older than the exact age of 1, 2, 3, or 4 years. Therefore, measurements would be expected to be higher than the standards.

The large numbers of Mexican American children below the 5th percentile for height, weight, head circumference and arm circumference is matter for concern. Twice as many boys as girls fell below the 5th percentile for weight, height, and head circumference. In view of the fact that the children may be 0 to 12 months older than the standard for a given age, the numbers of children below the 5th percentile are large.

CONCLUSIONS

The purpose of this study was to determine the incidence of lactose malabsorption in Mexican American children. These findings were correlated with selected dietary and anthropometric parameters to assess any possible effects of lactose malabsorption on diet and growth. The following conclusions were reached:

1. Lactose malabsorption and symptoms occurred significantly more often in Mexican American children than in Anglo American children.

2. The incidence of lactose malabsorption and symptoms increased with age in both Mexican American and Anglo American children.

3. The incidence of lactose malabsorption and symptoms was the same in males and females.

4. Lactose malabsorbers had significantly more symptoms after a lactose load than absorbers in both Mexican American and Anglo groups.

5. There were no significant differences between lactose absorbers and malabsorbers in milk intake or in the consumption of calories, protein, riboflavin, vitamin A and calcium. Anglo American children drank more milk and consumed more calories than Mexican American children.

6. Recognition of symptoms after drinking milk was more frequent in Mexican American children than in Anglo children, and was more frequent in lactose malabsorbers than absorbers.

7. No consistent differences were apparent between lactose malabsorbers and absorbers in height, weight, head circumference, arm circumference and triceps skinfold thickness in either Mexican American or Anglo American children. The means of anthropometric measurements for the Mexican American children tended to be smaller than the Anglo

American children. The Mexican American children had significant numbers of children below the 5th and above the 95th percentiles.

If lactose malabsorption does have a nutritional effect, the effect is not of sufficient magnitude to be revealed in the growth patterns of otherwise healthy children. Furthermore, most children can drink moderate amounts of milk with no adverse gastrointestinal symptoms. Consumption of moderate amounts of milk is nutritionally beneficial because of its high quality protein, and large amounts of calcium, riboflavin and vitamin A.

In view of the fact that most of the Mexican American children tested could comfortably consume the small amounts of milk in their diets, we recommend that they continue ingesting milk as tolerated. If milk intolerance on the basis of lactose malabsorption develops, tolerance to milk can be increased by consuming it with a meal, by drinking smaller amounts throughout the day, or by substituting lower-lactose content milk products such as aged cheese, yogurt, or buttermilk for milk.

Health professionals in areas with large Mexican American populations should be alerted to the high incidence of lactose malabsorption in this population. Pediatricians, school physicians and school nurses may find that recurrent unexplained abdominal pains and diarrhea in some children may be due to lactose intolerance. Physicians should also be aware that use of large quantities of milk in peptic ulcer disease or as a calcium and protein source in pregnancy may cause gastrointestinal symptoms in Mexican American adults.

Lactose malabsorption should not be used as an argument to dis-

continue the use of milk in overseas food aid programs. Skim milk has been used with success for years in the rehabilitation of severely malnourished children, although physicians routinely treating these children realize that some children respond better to a lactose-free diet. Administrators of food aid programs using milk should begin introducing milk in small quantities accompanied by other foods, and should be aware that some people may not tolerate even small amounts of lactose. On the whole, the benefits of milk nutrients to the majority of the population will probably justify its continued use in food aid programs.

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APPENDIX A

The University of Texas
Health Science Center at San Antonio
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San Antonio, Texas 78284

Department of Medicine

Dear Parents:

Milk is a very important food in our diet. It is a food that helps your child grow up to have strong bones and teeth and be healthy. Yet, many children cannot digest milk well. Milk may not be good for these children. Since this is a problem in many children, it is important to find out how many children can or cannot drink milk.

Doctors from the University of Texas Medical School are going to test many children. We wish to encourage you to have your child participate in this testing. The results of this test will be of great value to many children.

In this simple test, your child will drink a small glass of lemonade which will contain the sugar from milk. Tiny finger-prick blood samples will be taken before and after drinking the lemonade. Reactions to the test will be no different than when your child drinks milk.

In order for you to take advantage of this opportunity, please fill in and return the attached consent form to school by _____. On the day of the test your child should go to school without eating or drinking anything. We will remind you of this the day before the test. A snack will be provided at school after the test is over, about 9:00 A.M.

The information you learn from this test will help you to help your child grow up strong and healthy. We encourage you to return the attached consent form.

Sincerely,

Eleanor A. Young

Eleanor A. Young, Ph.D.
Assistant Professor
Section of Gastroenterology

Elliot Weser

Elliot Weser, M.D.
Professor and Head,
Section of Gastroenterology

The University of Texas
Health Science Center at San Antonio
7703 Floyd Curl Drive
San Antonio, Texas 78284

Department of Medicine

Queridos Padres:

Leche es una comida muy importante.de nuestro alimento. Es la comida que ayuda a su niño crecer y tener los huesos y dientes fuertes. Pero muchos niños no pueden digerir bien la leche. Leche no es buena para estes niños. Ya que este es un problema que se encuentra en muchos niños es importante saber cuantos niños pueden o no pueden tomar leche.

Médicos de la Escuela de Medicina de la Universidad de Texas aquí en San Antonio probarán muchos niños. Quisiéramos que su hijo tome parte en esta prueba. Los resultados de la prueba serán de gran valor a muchos niños.

En esta prueba sencilla, su niño beberá un vaso de limonada endulzada con azúcar de leche. Antes y después de beber la limonada, sacaremos una pequeña muestra de sangre de un dedo. La reacción que su hijo tiene después de la prueba no será distinta a la reacción que tiene después de tomar leche.

Para que usted aproveche esta oportunidad en participar en esta prueba, favor de llenar la forma y regresarla antes del _____. El día de la prueba, mande su hijo a la escuela sin comer o beber nada, unicamente agua si gusta. Recordaremos a usted el día antes de la prueba. La prueba será terminada a las 9:00 de la mañana, y serviremos una merienda.

La información que usted gana de esta prueba la ayudará a usted ayudar a su hijo crecer fuerte y sano. Le aconsejamos que vuelva la forma prendida tan pronto como sea posible.

Sinceramenta,

Eleanor A. Young
Eleanor A. Young, Ph.D.
Assistant Professor
Section of Gastroenterology

Elliot Weser
Elliot Weser, M.D.
Professor and Head,
Section of Gastroenterology

Consent Form for Blood Sampling

We are studying how well your child digests milk sugar. We would like your permission to take small finger-prick samples of blood from your child before and after he drinks a lemonade containing milk sugar. The amount of blood each time will be very small and will fill the tube pictured on the left. Reactions to the test will be no different than when your child drinks milk. We are also interested in how much milk your child usually drinks and we will be asking him questions about this.

There is no charge to you for this blood test, and you will be told the results.

In order for your child to help us in this study, please sign and complete the following:

This research has been explained to me and I hereby give permission for the blood tests to be taken for research purposes.

_____
Parent or Guardian's Signature

_____
Investigator

Child's
Name_____ Age_____ Grade_____

Height_____ Weight_____

Autorización Para Prueba de Sangre

Nosotros estamos estudiando como su niño digiere el azúcar de leche. Queremos su permiso para tomar una pequeña muestra de sangre del dedo de su niño antes y después de beber una limonada endulzada con el azúcar de leche. Cada vez la cantidad de sangre será muy pequeña y llenará el tubo dibujado a la izquierda. La reacción que su hijo tiene después de la prueba no será distinta a la reacción que tiene después de tomar leche. También estamos interesados en la cantidad de leche que su niño bebe cada día y le preguntaremos al niño acerca de esto.

Esta prueba será gratis y le avisaremos a usted del resultado.

Para que su niño nos ayuda en este estudio, por favor firme y complete lo siguiente:

Me han explicado esta investigación y yo doy mi permiso para que tomen esta prueba de sangre a propósito de investigaciones.

_____

Firma de Pariente o Guardián

_____

Investigador

Nombre del Niño _____ Edad_____ Grado_____

Altura _____ Peso_____

The University of Texas
Health Science Center at San Antonio
7703 Floyd Curl Drive
San Antonio, Texas 78284

Department of Medicine

Dear Parent:

We wish to thank you and your child for participating in the nutrition survey recently held at your child's school.

Your child, _____, was found to be tolerant to the sugar in milk. This means that he/she digests milk sugar well.

Milk is very important in helping your child grow, to develop strong teeth and bones and to keep him/her healthy. Children should drink three or more glasses of milk each day, and teenagers should drink at least four glasses of milk each day. Milk products such as cheddar cheese, cottage cheese or ice cream can be substituted for a glass of milk.

Remember, milk and milk products such as cheese are very important to your child's growth and health. He/She should have three or more servings of milk or milk products each day.

If you have questions about any of this information, please contact Sister Young or Mrs. Woteki at 696-6514. We will be happy to discuss any of this information with you.

Sincerely,

Sister Eleanor A. Young

Sister Eleanor A. Young, Ph.D.
Assistant Professor
Section of Gastroenterology

Elliot Weser

Elliot Weser, M.D.
Professor and Head,
Section of Gastroenterology

The University of Texas
Health Science Center at San Antonio
7703 Floyd Curl Drive
San Antonio, Texas 78284

Department of Medicine

Queridos Padres:

Muchas gracias por la participación de su hijo en el estudio recién de nutrición en su escuela.

Hemos encontrado que su hijo _____es tolerante al azúcar de leche. Esto quiere decir que el digiere bien el azúcar de leche.

La leche es muy importante en el crecimiento de su hijo, para los dientes y huesos fuertes, y para mantenerlo sano. Los niños deben tomar tres vasos de leche o más cada día, y los jóvenes deben tomar por lo menos cuatro vasos de leche cada día. En vez de un vaso de leche, Ud. puede reemplazar productos de leche como queso amarillo, requesón o helado.

Recuerde, leche y productos de leche como queso son muy importantes para el crecimiento y salud de su hijo. El debe tomar tres porciones o más de leche o productos de leche cada día.

- Si Ud. tiene preguntas acera de esta información, llame al teléfono a la Hermana Young o la senora de Woteki, number 696-6514. Nos dará placer en responder a sus preguntas.

Sinceramente,

Sister Eleanor A. Young

Sister Eleanor A. Young, Ph.D.
Assistant Professor
Section of Gastroenterology

Elliot Weser

Elliot Weser, M.D.
Professor and Head,
Section of Gastroenterology

The University of Texas
Health Science Center at San Antonio
7703 Floyd Curl Drive
San Antonio, Texas 78284

Department of Medicine

Dear Parent:

We wish to thank you and your child for participating in the nutrition survey recently held at your child's school.

Your child, _____, was found to be intolerant to the sugar in milk. This means that after drinking large quantities of milk or eating food containing milk sugar your child may have a stomach ache or diarrhea. If he/she does feel uncomfortable after drinking milk, he/she probably avoids drinking milk or eating foods containing milk. However, milk is very important in helping your child grow, to develop strong teeth and bones, and to keep him/her healthy. Therefore if your child refuses to drink milk, try some of the following hints:

1. Try giving milk in smaller servings. If a whole glass of milk causes your child discomfort, try serving a half glass of milk or less.

2. Don't serve milk alone as a snack; always include some other food, or serve milk only with a meal.

3. If your child refuses to drink milk, try buttermilk or substitute cheddar cheese or cottage cheese. These foods also contain the nutrients for strong bones and teeth and are low in milk sugar.

Remember, milk and milk products such as cheese and ice cream are very important to your child's growth and health. He/She should have three or more servings of milk or milk products each day.

If you have questions about any of this information, please contact Sister Young or Mrs. Woteki at 696-6514. We will be happy to discuss any of this information with you.

Sincerely,

Sister Eleanor A. Young

Sister Eleanor A. Young, Ph.D.
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Professor and Head,
Section of Gastroenterology

The University of Texas
Health Science Center at San Antonio
7703 Floyd Curl Drive
San Antonio, Texas 78284

Department of Medicine

Queridos Padres:

Muchas gracias por la participación de su hijo en el estudio recién de nutrición en su escuela.

Hemos encontrado que su hijo _____ es intolerante al azúcar de leche. Esto quiere decir que después de beber una grande cantidad de leche o de comer comida que contiene el azúcar de leche, tal vez su hijo tiene un dolor de estómago o diarrea. Si siente incómodo después de tomar leche, probablemente su hijo evita leche o comida que contiene leche. En todo caso, la leche es muy importante en el crecimiento de su hijo, para los dientes y huesos fuertes, y para mantenerlo sano. Por eso si su hijo rehusa leche, pruebe algunas de estas sugestiones:

1. Trate dándole leche en porciones más chicas. Si un vaso entero de leche le causa incomodidad, trate dándole medio vaso de leche, o menos.

2. No sirva leche sola como merienda; siempre incluya otro tipo de comida, o sirva leche con una comida.

3. Si su hijo rehusa tomar leche, ofrézcale suero de manteca, queso amarillo o requesón. Estas comidas contienen los alimentos para huesos y dientes fuertes y son bajos en azúcar de leche.

Recuerde, la leche y productos de leche como queso son muy importante para el crecimiento y la sulud de su hijo. Cada día el debe tomar tres o más porciones de leche o productos de leche.

Si Ud. tiene preguntas acerca de esta información, por favor llame al teléfono a la Hermana Young o la senora de Woteki, numero 696-6514. Nos dará placer en responder a sus preguntas.

Sincerамente,

Sister Eleanor A. Young

Sister Eleanor A. Young, Ph.D.
Assistant Professor
Section of Gastroenterology

Elliot Weser

Elliot Weser, M.D.
Professor and Head,
Section of Gastroenterology

LACTOSE INTOLERANCE SURVEY - 24-HOUR DIETARY RECALL

Name Address Phone #

Date Time School ☐ Sex 1-Male 2-Female ☐ Age ☐☐ Interviewer 1-Quintana ☐ 2-Woteki Day of Recall 1-Sun 2-Mon 3-Tues 4-Wed 5-Thur ☐

I.P.[a]	Food Item	Description	Size of Portion: oz.	fl. oz.	T	t	other: No	other: Unit	Srce[b]

[a] Ingestion Period Code
1 - A.M. 4 - P.M.
2 - Noon 5 - School Lunch
3 - Between Meals

[b] Source of Food Code
H - Home
S - School
P - Purchased (specify)
O - Other (specify)

Protein ☐☐
Calories ☐☐☐
Calcium ☐☐
Riboflavin ☐☐
Vitamin A ☐☐☐

1. Is this what you usually eat? ☐
   1-yes 3-less, illness 5-less, mother ill
   2-more food 4-less, no food available 6-less, other

2. Do you take vitamins or minerals? ☐
   1-no 3-Minerals 5-Unknown
   2-Vitamins 4-Both
   Type_____

3. How many vitamin or mineral pills do you take per day ? ☐

4. How much milk do you usually drink per day (include chocolate milk, hot chocolate, whole, skim, and evaporated milk)? ☐
   1. none or less than 1 glass 4. three glasses
   2. one glass 5. four glasses
   3. two glasses 6. more than four glasses

5. How many times per week do you eat: cheese ☐
   Ice cream, milk, pudding, custard

6. After you drink milk do you have gas or stomach ache? ☐
   1. no 3. stomach ache 5. all
   2. gas 4. diarrhea 6. other

LACTOSE INTOLERANCE SURVEY

Name # 1 - 3 1-Male Sex 2-Female

1. Weight (kg to nearest tenth) 4 - 7

2. Clothing estimate (kg to nearest tenth) 8-9

3. Nude weight (kg to nearest tenth) 10 - 13

4. Height (mm) 14 - 17

5. Head circumference (mm) 18 - 20

6. Arm circumference (mm) 21 - 23

7. Triceps skinfold (mm) 24-25

8. Lactose dose 2.0 g/kg

9. Symptoms (0-no 1-yes 2-missing) Time of onset Duration

bloating	26	
flatulence	27	
cramps	28	
loose stool	29	
diarrhea	30	
other (specify)	31	

10. G # 51 - 56

11. Comments

APPENDIX B

Recipes Used in Computation of Nutrient Content of Mexican American Foods

Flour tortillas

(Mrs. Rosemary Casey, Public Health Nutritionist, Bexar Co., Texas, Personal Communications)

		Calories	Protein(g)	Calcium(mg)	Riboflavin(μg)	Vitamin A(Iμ)
2 cups flour		400	11.6	18	280	0
1 teaspoon salt		-	-	-	-	-
2 teaspoons baking powder		-	-	-	-	-
4 tablespoons lard		504	0	0	0	0
Yield: 12 tortillas	=	904	11.6	18	280	0
1 tortilla	=	75	1.0	2	23	0

Enchilada Sauce

(Mrs. Rosemary Casey, Public Health Nutritionist, Bexar Co., Texas, Personal Communications)

Enchilada Sauce

(Mrs. Rosemary Casey, Public Health Nutritionist, Bexar Co., Texas, Personal Communications)

	Calories	Protein(g)	Calcium(mg)	Riboflavin (µg)	Vitamin A(Iµ)
½ cup minced onion	32	1.6	24	32	32
3 chile peppers	66	3.6	27	240	1260
1 clove garlic	2	0.1		-	-
2 cups canned tomatoes	84	4.0	24	120	3660
1 cup water	-	-	-	-	-
1 teaspoon lard	42	0	0	0	0
Yield: 3 cups =	226	9.3	75	392	4892
2 tablespoons =	9	0.4	3	16	203
Cheese Enchilada					
1 corn tortilla	63	1.5	60	15	6
2 tablespoons grated Chedder cheese	56	3.6	104	42	184
2 tablespoons enchilada sauce	9	0.4	3	16	406
Yield: 1 Enchilada =	128	5.5	167	73	596

Sopa de Arroz (Spanish rice) (156)

	Calories	Protein(g)	Calcium(mg)	Riboflavin(µg)	Vitamin A(Iµ)
3/4 cup rise	121	2.5	3	-	0
1/4 cup tomatoes	10	0.5	3	15	450
1 teaspoon lard	42	0	0	0	0
1 teaspoon onion	4	0.2	3	4	4
Yield: 1 cup =	177	3.2	9	19	454

Sopa de fideo (156)

3/4 cup noodles or macaroni	113	3.6	8	10	0
1/4 cup tomatoes	10	0.5	3	15	450
1 teaspoon lard	42	0	0	0	0
1 teaspoon onion	4	0.2	3	4	4
Yield: 1 cup =	169	4.3	14	29	454

VITA

Catherine Ellen O'Connor Woteki was born October 7, 1947 in Fort Leavenworth, Kansas. Since her father was a career Air Force officer, she traveled widely in the United States and lived in Weisbaden, Germany, Buenos Aires, Argentina and the Panama Canal Zone. Mrs. Woteki entered Mary Washington College in Fredericksburg, Virginia in 1965 where she studied biology and chemistry. While there she met her future husband, Thomas H. Woteki of Queens Village, New York. Mrs. Woteki graduated with a Bachelor of Science degree in Biology in 1969 and entered into a Master's degree program in Human Nutrition and Foods at VPI&SU in the same year. She received her Master of Science degree in 1972 and continued in a doctoral program at the same institution.

Mrs. Woteki is currently employed and makes her home in San Antonio, Texas. She is Assistant Instructor in Community Dentistry and Senior Research Assistant in Gastroenterology at the University of Texas Health Science Center, San Antonio, Texas. Her doctoral research was performed in San Antonio through the cooperation of the Health Science Center and VPI&SU.

Catherine E. Woteki

SOME NUTRITIONAL CONSIDERATIONS ON LACTOSE MALABSORPTION IN MEXICAN AMERICAN CHILDREN

by

Catherine O'Connor Woteki

(ABSTRACT)

Recent reports have appeared on the occurrence of lactose intolerance and low intestinal lactase activity in otherwise apparently healthy populations. In these individuals, administration of an oral lactose load fails to produce a significant increase in blood glucose concentrations and may be associated with moderate to severe gastrointestinal disturbances. Milk and milk products consumed in quantity may also induce these clinical symptoms of lactose intolerance. Since a high incidence of low lactase activity has been found in American Indians, Mestizo children and in Mexican American adult males, the present study was undertaken to determine lactose tolerance in Mexican American children in San Antonio, Texas, and to correlate this data with selected dietary and anthropometric measurements.

After an overnight fast, 282 healthy Mexican American and 51 Anglo American children between the ages of 2 and 14 years were given an oral lactose load of 2 g/kg body weight (up to a maximum of 50 g lactose) as a 20% solution in water. Capillary blood was drawn at 0, 15, 30 and 60 min and assayed for true glucose. A blood glucose rise of 25 mg/100 ml was considered a normal lactose tolerance test. A 24-hour dietary recall was obtained for each child, as were height, weight, head circum-

ference, arm circumference and triceps skinfold measurements. Gastrointestinal symptoms were carefully recorded for a 24-hour period following lactase loading.

Overall incidence of lactose malabsorption was significantly greater in Mexican American children than in Anglos. Lactose malabsorption increased with age in both groups. Incidence of symptoms occurring after lactose in lactose malabsorbers also increased with age. Most common symptoms reported in order of frequency were abdominal cramps, bloating and diarrhea.

Mean protein intake exceeded the RDA at all ages for both ethnic groups. Mexican Americans consumed less protein, riboflavin, vitamin A, calcium and fewer calories than Anglos. There were no differences in caloric and nutrient intakes or in milk consumption between lactose absorbers and malabsorbers, but Anglos drank significantly more milk than Mexican Americans.

Mean height and weight for Mexican American males and females were slightly below the 50th percentile of the Stuart-Meredith standards and slightly above the 50th percentile of the Ten-State Nutrition Survey's means for Mexican American children. Triceps skinfold and head circumference measurements were similar to standards.

Fifteen percent of the Mexican American lactose absorbers and 23% of the malabsorbers reported having symptoms after milk ingestion. None of the Anglos recognized symptoms after drinking milk.

If lactose malabsorption does have a nutritional effect, the effect is not of sufficient magnitude to be revealed in the growth patterns of otherwise healthy children. Most children consumed small to moderate

amounts of milk with no adverse gastrointestinal symptoms. Because milk is a source of high quality protein, calcium, riboflavin and vitamin A, milk drinking should be encouraged as tolerated.