Committee on Nutrition

Should Milk Drinking by Children Be Discouraged?

During the early weeks of life, most infants depend on some form of milk to meet most of their nutritional needs. Even after other foods are introduced, milk continues to supply more than half of the needed calories and is the major source of many essential nutrients in technically advanced countries. In developing areas of the world, where protein-calorie malnutrition is a serious childhood problem, the use of cows’ milk for child feeding programs has been advocated and encouraged for several decades. Cows’ milk is a good source of high quality protein, fat, carbohydrate, B vitamins, and minerals. However, in the natural state cows’ milk is an incomplete source of iron, copper, and some vitamins, particularly vitamins C and D. Of these, only vitamin D is usually added to processed milk.

Man is unique among mammals in that his age of natural weaning has not been established with any certainty. In the so-called developed countries, breast feeding is increasingly uncommon; and in such countries, even when an infant is breast fed, what we call weaning is largely a transfer of lactic affections to the milk of another mammalian species, usually the cow. To what degree this transfer is conditioned by the nutritional needs of the individual as opposed to the cultural and economic pressures of a society’s traditional feeding practices is a matter for speculation.

Some recent articles, chiefly in the lay press, have suggested that cows’ milk can lead to untoward effects in older infants and children, and have raised doubts as to the desirability of feeding milk after the nursing period. Some of these articles have been sensational and alarmist. Therefore, it seems timely for the Committee to re-examine the possible advantages and disadvantages of consuming cows’ milk at various ages and in various quantities.

The concerns expressed recently about milk consumption fall mainly into three categories: (1) lactase deficiency causing milk intolerance; (2) saturated fat and cholesterol content, which may increase the risk of coronary heart disease later in life; and (3) allergy to milk protein. Each of these will be considered briefly.

Milk Intolerance, Lactase Deficiency, and Lactose Intolerance

Some otherwise healthy individuals develop abdominal distension, cramps, and watery diarrhea within an hour or two after drinking milk and are said to have “milk intolerance.” In most, though not all instances, these gastrointestinal disturbances are the result of lactase deficiency. The enzyme lactase (β-D-galactosidase) hydrolyzes lactose into its two component monosaccharides (glucose and galactose). The enzyme lactase, together with other disaccharidases, is located in the brush border or microvilli of the epithelial cells lining the small intestine. It is distributed throughout the small intestine, though its activity is less in the duodenum and terminal ileum. It is the last of the disaccharidases to appear during fetal development. Levels of activity in normal subjects may be low at birth but increase rapidly thereafter, independent of milk intake, and apparently decrease considerably after 3 or 4 years of age.

In individuals deficient in lactase activity, lactose passes unaltered to the colon where its osmotic effect increases gut fluid volume. In addition, the lactose is fermented by bacteria in the colon to produce lactic acid and other organic acids which lower the pH of the stool below 6.0. This sequence results in the symptoms of abdominal distension, cramps, and watery, frothy diarrhea. Deficiency of intestinal lactase may be confirmed by demonstrating little or no enzyme activity in duodenal or jejunal mucosa obtained by peroral biopsy. A presumptive diagnosis may be made indirectly by means of an oral lactose tolerance test using a standard lactose dose of 50 gm/sq m or 2 gm/kg in children. Normally, the blood glucose level in-
creases by more than 25 mg/100 ml at some point between 15 and 60 minutes after the test dose is given; lesser increases usually indicate lactase insufficiency. However, individual responses to lactose tolerance tests are variable, and the test is not without risk in lactase-deficient subjects.

Lactose intolerance was first described in infants by Durand in 1958 and later by Holzel et al. in 1959. Congenital (hereditary) lactase deficiency is the most severe form and is rare. Patients with this disorder do not tolerate the amount of lactose present in the quantities of milk usually ingested at a single feeding. Deficiencies of lactase, and to a lesser extent disaccharidases, have also been documented in association with many gastrointestinal diseases, including celiac disease, cystic fibrosis, ulcerative colitis, protein-calorie malnutrition, following gastrointestinal surgery in infancy, and during and following acute gastroenteritis. In these gastrointestinal diseases, lactase deficiency is usually temporary, and normal levels of the enzyme return soon after the primary disease is relieved. Milk intolerance may be apparent clinically in these diseases when lactase activity is low; at such times it is best to omit milk temporarily from the diet. In children with cystic fibrosis, the degree of deficiency seldom is such that they must avoid milk entirely.

Partial or complete deficiency of intestinal lactase has been documented in many healthy adults who have no lesions of the intestinal mucosa and in whom levels of disaccharidases are normal. These adults have milder clinical findings and may tolerate without symptoms the approximately 12 gm of lactose in an 8-oz serving of milk, but often develop symptoms with larger intakes. The prevalence of this primary (physiological?) adult form of lactase deficiency varies greatly with racial origin and geographic location; it is lowest (2% to 8%) in white adults of Scandinavian and West European ancestry, and highest (60% to over 90%) in those of Mediterranean, African, and Asiatic extraction. Since the majority of adults in the world have low levels of intestinal lactase, this could be the norm for many human adults. The incidence and clinical expression of this form of lactase deficiency is not fully established for children of different ethnic origins, though a few limited studies have been done.

Paige and his associates investigated the lactose tolerance and milk consumption of 90 impoverished Peruvian Mestizo children. They compared these children to 22 well-nourished siblings who had been raised in a protected environment and were well developed, and to 50 siblings from the same poor households.

Of the 90 impoverished children, 76 (84%) had an abnormally low response to an oral lactose load. However, 73% of those less than 3 years of age had normal responses, with a notable change after that age. All subjects more than 12 years of age had flat curves. Gastrointestinal symptoms were noted in 75% of those with flat tolerance curves within 45 minutes after the administration of lactose.

The 22 well-nourished siblings showed a similar incidence of lactose intolerance after 3 years of age. Also, those with normal lactose tolerance had significantly greater milk consumption. The authors found no relation between the duration of breast feeding and either lactose tolerance or milk consumption. These findings confirm that there tends to be a progressive diminution in the ability to hydrolyze lactose after about 3 years of age.

In 1965, Cuatrecasas and co-workers documented notable differences in lactose tolerance between black and white adults in the United States; intolerance was demonstrable in 70% of blacks as compared to 6% to 15% of whites.

Paige and his associates studied black and white school children receiving an organized school feeding program. They found that 20% of black elementary school children rejected 50% or more of the free milk as compared to 10% of white children. More than half of a sampling of the black nonmilk drinkers showed flat lactose tolerance curves.

There is still uncertainty about the extent to which the volume of milk consumed over a period of time can affect the level of intestinal lactase activity, or conversely, the extent to which individual milk intakes reflect the level of lactase activity; the latter is also determined by genetic factors.

For most children, even after the age of 3, drinking moderate amounts of milk has no apparent adverse effects and is nutritionally beneficial. Nevertheless, the problem of lactase deficiency may be one of genuine significance relative to mass feeding programs for poverty groups at home and abroad.

The Protein Advisory Group of the United Nations and The Food and Nutrition Board of the National Research Council have both stated that, based on present evidence, it would be inappropriate to discourage programs for increasing milk supplies and consumption because of a fear of milk intolerance. At this time, the Committee on Nutrition agrees fully with these statements. Suitable substitutes are usually unavailable in the areas involved.

## SATURATED FAT AND CHOLESTEROL CONTENT

Milk is an important source of cholesterol and saturated fats which, in excess, may possibly affect the risk of coronary heart disease later in life.
however, there is no evidence as yet that the prevalence or severity of atherosclerosis can be diminished by limiting the intake of cholesterol beginning early in life. The Committee on Nutrition and a subcommittee of the Committee on Rheumatic Fever and Congenital Heart Disease of the American Heart Association have recommended against a radical reduction of saturated fats in the diets of all children until much more is known about the benefits versus possible adverse effects. This position is supported by a recent statement of the Food and Nutrition Board of the AMA Council on Food and Nutrition. Dietary restriction of saturated fats is considered to be indicated at present only for children with hereditary hypercholesterolemia, a disorder which appears to be genetically determined and can be detected during infancy by screening methods. A family history of coronary heart disease before 50 years of age in a child’s first cousin or closer relative should alert the physician to the possibility that the child may have hereditary hypercholesterolemia or hyperlipidemia. The presence of the abnormality can be established by analysis of fasting serum specimens for cholesterol, triglycerides, and lipoproteins. The incidence of hereditary hypercholesterolemia is estimated to be approximately one per 150 live births. Except when hyperlipoproteinemia is demonstrated, placing the child on skimmed milk or special diets to avoid saturated fats is not adequately justified at present. If carried to extremes, restriction of milk fat could do harm by resulting in a deficiency of essential fatty acids.

There is no evidence that milk consumption per se makes any specific contribution to the development of obesity. Nor is there any evidence that the use of skimmed or partly skimmed milk, in the absence of other dietary measures, results in a reduction in total caloric intake of children who usually make up the missing calories from other sources. However, skimmed milk or low-calorie milk substitutes may have a role in voluntary weight control programs for older individuals.

For those at risk of coronary artery disease, the use of skimmed or partly skimmed milk is an important and justifiable method of reducing the dietary intake of saturated fats and cholesterol. A general recommendation to restrict milk fat is difficult to justify scientifically and may promote unnecessary anxiety in the general population. Moreover, the widespread indiscriminate consumption of low fat milk might well deprive some children of needed calories.

**MILK ALLERGY**

The incidence of documented human clinical allergy to bovine milk continues to be a subject of great dispute; estimates vary widely.

The manifestations of cows’ milk allergy may vary from mild rhinorrhea to more dramatic instances of anaphylactic shock. The symptoms can include diarrhea, vomiting, abdominal pain, colic, rhinitis, asthma, atopic dermatitis, urticaria, anaphylaxis, or involvement of the central nervous system. Goldman et al. could elicit most of the symptoms by challenging the patient with an oral load of one of several purified milk proteins; they also noted that it was uncommon to find only a single, presenting symptom. A syndrome of recurrent pulmonary disease, iron-deficiency anemia, poor growth, and gastrointestinal symptoms, has also been ascribed to milk allergy. Waldmann et al. described an allergic gastroenteropathy with edema, growth retardation, hypoalbuminemia, hypogammaglobulinemia, anemia, eosinophilia, and allergic symptoms of the skin, and gastrointestinal and respiratory tracts. Since most body systems have been reported to be reactive to bovine milk antigens, it is not surprising that allergic reactions to cows’ milk might occasionally be confused with other disorders.

Sensitization to cows’ milk in utero has been demonstrated in the guinea pig but not in man. Almost all proteins isolated in pure form from bovine milk have been shown to be potential allergens. Since the bulk of milk protein consists of the caseins, \( \beta \)-lactoglobulin, \( \alpha \)-lactalbumin, and bovine serum albumin, most recent immunological studies have focused on these particular constituents.

Although heat denaturation alters basic protein structure by decreasing its antigenicity or allergenicity, this has not proved useful in the treatment of severe milk allergies. The degree of immunological inactivation depends on the duration and intensity of the temperature used. However, even with extreme heating, the various caseins do not lose their allergenic properties completely. Actually, the allergenicity of \( \beta \)-lactoglobulin may be potentiated by heating because of the formation of a heat-stable product with the lactose of milk. Utilizing purified milk proteins for patient challenges, Goldman et al. demonstrated that most children thought to be sensitive to milk gave positive responses to \( \beta \)-lactoglobulin, \( \alpha \)-lactalbumin, bovine serum albumin, or the composite casein fraction. Spies et al. have shown that the milk proteins, when subjected to brief peptic hydrolysis, may form new fragments which are immunologically distinct from the parent proteins. If low molecular-weight antigens such as these are produced in animals during digestion, they could have a bearing on the nature of material to be used for routine clinical testing. Conceivably, the human
may react not only to intact protein but also to portions of the protein molecule produced by the gastric enzymes.

It is doubtful that heated milk products are effective in the treatment of milk allergy, except for those related to bovine serum albumin sensitivity or the more heat-sensitive euglobulins. On the basis of current clinical data, it appears imprudent to prescribe any form of heated milk in known instances of allergy to casein, alphalactalbumin, or \( \beta \)-lactoglobulin.

If the incidence of milk allergy in the general population and in the infant group is at the currently estimated level of approximately 2%,\(^{87} \) clinical experience warrants restriction or elimination of milk intake for such individuals when clear-cut, positive clinical reactions are seen. When total avoidance of milk is deemed advisable, a number of milk substitute formulas, prepared from soy or meat protein or hydrolyzed protein and adequately supplemented with needed vitamins and minerals, are readily available. Older children and adults may avoid milk or milk substitutes without harm. A milkless diet is adequate.

There is evidence that gastrointestinal blood loss may be associated with ingestion of fresh cows' milk, but the nature of this association is not completely clear. In one study,\(^{88} \) severe iron deficiency in some infants was accompanied by intestinal mucosal changes and blood loss. Treatment with iron reversed the mucosal changes which were therefore presumed to be related primarily to the iron deficiency. In other studies, the bleeding was attributed to an enteropathy induced by fresh cows' milk.\(^{89,40} \) Blood loss as measured by the appearance of \( ^{51} \)Cr from labeled red blood cells in the stool was increased by feeding fresh milk and reduced by the substitution of heat-treated cows' milk or a soy bean formula. In a small group of normal infants fed fresh cows' milk from 2 months of age, microcytosis, ironopenia, and minimal decreases in hemoglobin concentration were found at 6 to 12 months of age despite iron supplementation of the diet.\(^{41} \) This finding was thought to be compatible with induction of gastrointestinal bleeding, but other explanations such as an altered absorption of iron are also possible.

Although the magnitude, frequency, and significance of enteric bleeding associated with consumption of cows' milk in infancy has not been firmly established, the available evidence suggests that infants fed fresh cows' milk should receive iron from another source. These infants should also be watched for indications of iron deficiency and gastrointestinal bleeding. Cows' milk should be eliminated from the diets of infants with demonstrated gastrointestinal blood loss. Excessive or persistant bleeding should, of course, be studied to exclude other causes.

The concept that children and teen-agers should drink plenty of milk as a source of calcium (and phosphorus) to ensure "healthy bones and teeth" is a tenet of North American health culture that is rarely questioned and one to which many physicians, dentists, and nutritionists subscribe. It is not unusual for the milk intake of children with dental caries or congenital enamel defects to be questioned or even for calcium supplements to be suggested.

Although the central role of calcium and phosphorus in osseous and dental structure is clear, experts' attempts to define minimum (much less optimum) daily requirements for calcium in healthy individuals at different ages have been notably unsuccessful and reveal a major gap in current knowledge of human nutrition.

This subject was reviewed by an Expert Group of the Food and Agriculture Organization and WHO in 1961.\(^{42} \) The situation has not changed significantly in the interval. The capacity of man and other animals to adapt to wide ranges of calcium intake was noted (because of the adaptability of intestinal and renal transport processes), emphasizing the unreliability of short-term balance studies as indications of daily requirements. It was observed that total daily calcium intakes varied widely from one country to another. The evidence of ill health possibly related to low or high intakes of calcium was reviewed; it was concluded that there is no convincing evidence (assuming adequate vitamin D intake and good general nutritional status) that calcium intakes of less than 300 mg/day or more than 1,000 mg/day from all sources were harmful to human health. The report pointed out that the development of rickets and dental caries was largely independent of calcium intake, and that the mean concentration of calcium was normal in the breast milk of mothers used to low calcium diets.

This expert group was unable to define minimum or optimum calcium requirements for infants and children based on any data then available. In an attempt to give some guidance, they cautiously elaborated "suggested practical allowances." These ranged from 500 to 600 mg/day for infants to 600 to 700 mg/day for the 10- to 15-year age group from all dietary sources. They made a final strong plea for basic research to determine human comprehensive requirements. The considerable variation in recommended daily adult intakes in different countries (from 400 to 800 mg/day) gives ample testimony to the lack of conclusive data on human calcium needs and to the many ways in which the limited, available information can be
interpreted.

The detailed evidence bearing on calcium requirements is beyond the scope of this paper, but it will be the subject of a later Committee statement. The conclusion can be made that, from the viewpoint of calcium requirements alone, the amounts of milk recommended for daily consumption by children and adolescents in both popular and official health statements could be above what is required for normal skeletal, dental, and general growth and development.

There are three fairly common situations in pediatric practice which may call for restriction of milk intake: first, the child with nutritional iron deficiency. The dietary history of these children typically reveals unusual prolongation of bottle feeding, refusal of solid foods, and the fact that almost all the child's caloric requirement is being met by milk alone. It may be difficult to ascertain which of these three phenomena is of primary etiological importance. But, from a practical viewpoint, stringent restriction of the quantity of milk consumed is an essential component of management.

The second problem, often encountered in the middle-class 1- to 3-year-old in pediatric office practice, is the child who "won't eat." When such apparent anorexia is accompanied by a history of the child being full of energy and normally grown, determination of the daily milk consumption often reveals that milk accounts for 90% or more of the youngster's daily caloric requirement. Again, in such children, the appetite for a variety of foods can be restored by restricting milk consumption to one third or less of the estimated daily caloric requirement.

Finally, the otherwise healthy, whole-milk-drinking child who tends to be constipated may be considerably relieved of his problem by a combination of limiting the milk intake and using skimmed or partly skimmed milk instead of whole milk. This measure reduces the formation of insoluble calcium soaps and results in stools of softer consistency. In simple constipation, this measure alone may suffice. In more severe cases, it is an important adjunct to therapy.

CONCLUSIONS

Although human milk is the optimum form of nourishment for young infants, cows' milk is a valuable substitute and a useful food for older infants and young children. It supplies a large proportion of essential nutrients and calories but it is not an essential component of the diet for anyone whose diet is otherwise adequate.

Milk should be eliminated from the diets of children with the rare congenital (hereditary) form of lactase deficiency, those with galactosemia, and those with convincing evidence of milk allergy. Temporary elimination of milk may be beneficial in children with transient lactase deficiency associated with various gastrointestinal disorders.

Important racial and age differences in lactase activity are recognized, but our understanding of the etiology and clinical significance of these differences is incomplete. Thus, no firm conclusions can be drawn as yet about the relevance of such differences to the planning of nutritional rehabilitation programs for developing countries and poverty groups, especially in that comparable sources of protein, calories, and other nutrients may not be readily available.

The use of skimmed milk is recommended for the approximately one in 150 children born with hereditary hypercholesterolemia as one of the dietary procedures required to lower the blood cholesterol in an attempt to delay the onset and reduce the severity of premature atherosclerosis. Evidence available to date does not support the use of skimmed milk in the child population at large, and such a measure might diminish already inadequate calorie intake in some groups. In infants and young children, the use of skimmed milk may provide an excess of protein and minerals in relation to calories.

The amount of milk that should be consumed by healthy older infants and children cannot be stated with convincing accuracy, partly because this will depend to a large extent on economic factors and on the availability of other foods. When a mixed diet is available in adequate quantities, milk consumption should probably not exceed one third of the total daily calorie intake after the latter part of the first year of life (two or three glasses per day).

The evidence available at present is insufficient to warrant discouraging programs for increasing the supply and consumption of milk in developing countries where childhood malnutrition is rife, particularly since nutritionally and economically suitable alternatives may not be available in such countries. The distributor of milk under these circumstances should be accompanied by instruction in preparation for feeding to reduce bacterial contamination.

If the position stated with regard to some of these issues appears equivocal, it is because the Committee believes that the sum of pertinent, current knowledge does not permit a more dogmatic position. As additional information becomes available, the Committee will review the situation and stands ready to revise its opinions.

COMMITTEE ON NUTRITION
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REFERENCES


AMERICAN ACADEMY OF PEDIATRICS

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John Burke, son of Dr. Frederic G. Burke, F.A.A.P., movingly describes one of his nocturnal attacks of asthma in these words:

Le Jour Est Dans Moi

When will the morning come that frees me from my captor? He is a cruel, unrelenting foe who prisons me in a world of insufficient air. . . . My body seems to atrophy and starve for the vigor like that which spirits all my friends. They grow taller yet I remain small. They will never know how brief my days are, how anxious night is to collude with my foe and take what little wind I have.
Yet it is folly to dwell on my infirmity. I will not be shamed by a fear I nurture with self-pity. I defy my fears and I defy my foe.
The day is mine. My soul overflows with life. . . . it is a fire that dispels the dark, that scatters my trepidations. The day consumes me, it is a surge of defiance and life.
I have the day and it shall be my captive. I will be the tyrant who will not let it be seized away by lowly fears and lowly pity. Le jour est dans moi.
Noted by THOMAS E. CONE, JR., M.D.
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*Pediatrics* 1974;53;576

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