

Is Cow's Milk Harmful to a Child's Health?

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ABSTRACT

Discussions and debates have recently emerged on the potential positive and negative effects of cow's milk in the paediatric community, also under the pressure of public opinion. The negative effects of cow's-milk consumption seem to be limited to iron status up to 9 to 12 months; then no negative effects are observed, provided that cow's milk, up to a maximum daily intake of 500 mL, is adequately complemented with iron-enriched foods. Lactose intolerance can be easily managed and up to 250 mL/day of milk can be consumed. Allergy to cow's-milk proteins is usually transient. Atopic children may independently be at risk for poor growth, and the contribution of dairy nutrients to their diet should be considered. The connection of cow's milk to autistic spectrum disorders is lacking, and even a cause-effect relation with type 1 diabetes mellitus has not been established because many factors may concur. Although it is true that cow's milk stimulates insulin-like growth factor-1 and may affect linear growth, association with chronic degenerative, noncommunicable diseases has not been established. Finally, fat-reduced milk, if needed, should be considered after 24 to 36 months. Cow's milk represents a major source of high nutritional quality protein as well as of calcium. Moreover, it has growth-promoting effects independent of specific compounds. Its protein and fat composition, together with the micronutrient content, is suggestive of a functional food, whose positive effects are emphasised by regular consumption, particularly under conditions of diets poor in some limiting nutrients, although in industrialised countries cow's milk's optimal daily intake should be around 500 mL, adequately complemented with other relevant nutrients.

Key Words: chronic-degenerative disorders, cow's milk, growth, milk composition

(*JPGN* 2011;53: 594–600)

Cow's milk recently has been targeted by the media for having a presumed deleterious role in the development of both acute and chronic diseases in young and older children. Milk consumption has been even recently considered "a major health hazard and 'the promoter of Western chronic diseases'" (1). Pros and cons of its consumption and promotion have been widely discussed in the last few years, and discussions have been raised on the possible negative effects on a child's health. In the present article we consider the critical points regarding the effects of cow's milk and presumed

associated risk conditions. A literature search strategy was performed through MEDLINE, PubMed, Cochrane Controlled Trial Register, and Web of Knowledge using "cow's milk," and "nutritional value," as well as any of the following conditions: iron-deficiency anaemia, lactase deficiency, cow's-milk protein allergy (CMPA), autism spectrum disorders (ASD), type 1 diabetes, metabolic syndrome, chronic-degenerative disorders, and cancer.

NUTRITIONAL VALUE

Whole cow's milk is a complete source of energy, made up of all of the major macronutrients and partly of some micronutrients, particularly calcium and phosphorus (Table 1). On a comparative basis with other animal sources, whole cow's milk is the richest source of calcium and vitamin D, considering beef meat and eggs, and is the cheapest source of protein, calcium, phosphorus, and vitamin D (Table 2) (2). A complete evaluation of the effects of cow's milk should take into account these aspects that represent a relevant contribution to the popularity of cow's milk. Indeed, cow's milk historically is the first choice for the prevention and treatment of moderate and severe malnutrition in children from low-income countries (3), and may represent the most accessible animal source of proteins with high biological value (4).

In spite of these considerations, the popularity of cow's milk has been the object of periodical reappraisals, not only in the lay press but even in paediatric journals and publications, according to the changing perspectives of treatment and prevention of paediatric disorders through the years (5,6). The classical complication associated with low consumption and/or avoidance of cow's milk during the lifespan has been recognised in lower intake and deposition of calcium within bones, thus negatively affecting bone mineral content and bone density (7), with a major predisposition to bone fractures (8). We should consider the following major critical points associated with the consumption of cow's milk: the risk of iron-deficiency anaemia in infants, lactase deficiency, allergy to milk proteins, ASDs, increased risk of type 1 diabetes mellitus, and possible associations with chronic degenerative, noncommunicable disorders such as metabolic syndrome and related complications and cancer.

IRON-DEFICIENCY ANAEMIA

Infants younger than 12 months may develop iron-deficiency anaemia when they are switched from maternal milk and not adequately complemented. In the Euro-Growth study involving 488 infants from 11 European centres, the prevalence of iron deficiency and iron-deficiency anaemia was 7.2% and 2.3%, respectively (9). Early introduction of cow's milk was the strongest negative determinant of iron status, with each month of cow's-milk feeding increasing the risk of iron deficiency by 39%. Feeding of iron-fortified formula was the main factor positively influencing iron status. Several different mechanisms may act synergistically (10):

1. The low iron content of cow's milk (Table 1).
2. Calcium and casein provided by cow's milk in high amounts because calcium and casein together inhibit the absorption of dietary nonheme iron.

Received July 26, 2011; accepted September 1, 2011.

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The authors report no conflicts of interest.

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DOI: 10.1097/MPG.0b013e318235b23e

TABLE 1. Whole cow's-milk composition (average values from various sources)

Nutrient	Value per 100 g
Energy, kcal	63
Carbohydrate (lactose), g	4.4
Fat, g	3.5
Protein, g	3.5
Calcium, mg	115
Phosphorus, mg	93
Iron, mg	0.04
Zinc, mg	0.43
Vitamin A, retinol equivalent	30
Vitamin D, µg	0.10
Folate, µg	9.0
Vitamin C, mg	1.2

- The resulting low iron bioavailability for absorption. Estimating <0.05 mg iron/100 g cow's milk, and an absorption rate of 10%, <5 µg/100 g intake should be absorbed (11).
- Occult intestinal blood loss in approximately 40% of normal infants during feeding of cow's milk.

The origins of the latter phenomenon are still largely unknown, and cannot be attributed to either the casein or whey fraction. The occult blood loss has been observed anecdotically also with fermented milk products in the first year of life, whereas it has been observed that babies exposed to modified cow's milk antigens from birth have a lower incidence of blood loss. Studies developed to define the size of the combined effect of poor iron supply and occult blood losses of cow's milk on iron status have found significant negative associations in the 9- to 12-month period when consumption was >460 mL/day (12). In any case, the loss of iron in the form of blood diminishes with age and ceases after 1 year of age.

Twenty years ago, the Committee on Nutrition of the American Academy of Pediatrics recommended that whole cow's milk not be used during the first year of life (13). The Committee on Nutrition of European Society for Pediatric Gastroenterology, Hepatology, and Nutrition has considered the question of the introduction and the effects of cow's milk in infants' diet with 2 separate documents. In the first one, aimed at the prevention of iron-deficiency anaemia, the recommendation to postpone the introduction of whole cow's milk up to 12 months and, in case of breast-feeding failure, to substitute with an iron-enriched formula was clearly indicated (14). A few years later, when considering the issue of complementary feeding, the Committee suggested that recommendations on the age for introduction of cow's milk had to take into consideration tradition and feeding patterns in the population, especially the intake of complementary foods rich in iron and the volume of milk consumed. Accordingly, the Committee concluded that it is acceptable to add small volumes of cow's milk to

complementary foods, but it should not be used as the main drink before 12 months (15) and not to displace richer sources of iron. The so-called growing-up milks have a lower protein content than cow's milk and are supplemented with trace elements, including iron, vitamins, and essential fatty acids. Although the commercialisation of growing-up milks continues to increase in many countries worldwide, particularly in Europe (16,17), their benefits are still a matter of debate. This controversy arose because the possible nutritional risks associated with the use of cow's milk and the expected benefits from the use of growing-up milks have not been clearly demonstrated after the age of 1 year.

LACTASE DEFICIENCY

Lactose is the primary sugar of mammalian milk. Ingested lactose is hydrolysed by lactase, an enzyme of the microvillus membrane of the enterocytes, into its components glucose and galactose, which are absorbed. If lactase activity is low or absent, undigested lactose may induce the symptoms of lactose intolerance. Lactase deficiency or nonpersistence (adult-type hypolactasia) is caused by the downregulation of lactase enzyme activity during childhood. Lactase deficiency likely represents the most popular adverse status associated with disturbing symptoms ascribed to the consumption of cow's milk and some fresh dairy products (18).

The downregulation of lactase activity is genetically determined and occurs soon after weaning (starting at 24–36 months) in most ethnic groups, usually increasing from northern to southern Europe, being maximal in sub-Saharan countries. When symptomatic, it includes moderate-to-acute symptoms of excessive flatulence, bloating, abdominal pain, and diarrhoea. Besides large interindividual variability, even within single ethnic groups, it is also characterised by the weak association between symptoms and diagnosis, mostly based on breath test assessment of hydrogen produced by fermentation of undigested lactose by colonic bacteria (19,20). Consequently, most individuals presumed to be affected prefer to avoid milk and milk-containing products by self-selection, with a consequent low intake of calcium and the possible untoward consequences on bone health, starting even from adolescence (21,22). In revising the matter of the definition of lactose threshold in lactose tolerance, the European Food Safety Authority recently issued a document emphasising that lactose tolerance varies widely in individuals with (presumed or real) lactose maldigestion (23). A single threshold for all lactose-intolerant individuals cannot be determined because symptoms of lactose intolerance have been described even after intake of <6 g lactose, but most individuals diagnosed as having lactose intolerance or lactose maldigestion can tolerate 12 g of lactose as a single dose of milk (ie, approximately 250 mL) with no or minor symptoms (20). Higher doses may also be tolerated if distributed throughout the day. Individuals need to adapt their lactose consumption to their individual tolerance. Recent recommendations (22) to address lactose intolerance point out the beneficial effects of regular milk consumption that may adapt colon bacteria, thus facilitating the digestion of lactose; the consumption of yogurts and cheeses, mildly lower in lactose but displaying lactase activity (particularly yogurts and fermented products) at lower temperature, further aiding lactose digestion within the gastrointestinal tract; the consumption of dairy foods with meals to slow transit and maximise digestion; the use of milks with low lactose content, that is, with lactose already split by enzymatic intervention. The use of lactose-digestive aids has also been advocated, but they are expensive and their use is scarcely evidence based.

Secondary lactase deficiency results from diseases of the small intestine that damage the intestinal epithelium, leading to subsequent lactose maldigestion of different degrees. Acute gastroenteritis, untreated coeliac disease, and chronic intestinal inflammation may

TABLE 2. Comparative data (Euros/quantity) (2)

	Milk	Beef meat	Eggs
Raw material/kg	1.4	15.5	5.7
Protein/100 g	43.2	72.8	46.0
Calcium/100 mg	1.2	454	11.2
Phosphorus/100 mg	1.5	9.4	2.7
Vitamin D/1 µg	19.5	—	1980

be associated with hypolactasia; however, when the epithelium heals, the activity of the lactase returns (18). In populations with a low prevalence of the adult type of hypolactasia, screening tests for coeliac disease could be considered in children presenting with chronic symptoms of lactose intolerance.

COW'S-MILK PROTEIN ALLERGY

Cow's-milk protein is the most frequently encountered dietary allergen in infancy. The prevalence of CMPA in childhood ranges between 2% and 7% (24), depending on the methods of recruitment, age distribution of populations studied, and diagnostic criteria. Caregivers should keep in mind that the prevalence of CMPA as perceived by the child's parents is higher than that of actual CMPA (25). The majority of affected infants acquire natural tolerance to cow's-milk protein before age 3 years. According to Høst et al (26), remission rates are 45% to 50% at 1 year, 60% to 75% at 2 years, and 85% to 90% at 3 years. Persistent cases of CMPA are characterised by the intensity of the family atopy history, a longer period between the consumption of the cow's-milk proteins and the onset of symptoms, a high frequency of multiple food allergies, and the coexistence of asthma and allergic rhinitis, as well as more of an allergy to casein than to soluble proteins. CMPA with early gastrointestinal symptoms has a better prognosis as opposed to CMPA with immunoglobulin E-mediated symptoms (27). The resolution of CMPA may be partial (28). Some children considered to be free of CMPA may retain a residual disease and may not be able to tolerate a normal intake of milk and dairy products. In a Finnish study, 45% of children, who were reliably diagnosed as having a CMPA during the first year of life and who were since considered free of that CMPA, complained at 10 years of gastrointestinal symptoms (diarrhoea, abdominal pain, and/or nausea) in relation to the ingestion of dairy products, compared with 10% in the control group (29). The nutritional effect of CMPA varies considerably in both expression and intensity and should be systematically evaluated (30). It depends not only on the extent of intestinal mucosal inflammation, which may induce malabsorption and/or protein-losing enteropathy, but also on the occurrence of skin protein losses, as in cases of atopic dermatitis (30). An inadequate energy intake in children with food (mainly milk) allergy on an elimination diet has been demonstrated in previous studies, together with low rates of growth concerning both length and weight, particularly in the first year of life (31). In an Italian study, for instance, we found an impairment in growth of infants with atopic dermatitis in the first year of life, beginning in the first months of life. The impairment was notably marked after the onset of disease, but for length it was evident even before the onset of disease (32). A proper diagnosis of CMPA is therefore crucial because restricted diets can reduce the quality of life and lead to serious detrimental effects, especially in infants and young children. The elimination diet prevents the allergic inflammation induced by the offending food, but may have deleterious effects on the child's nutritional status and growth pattern. Abnormal feeding behaviours induced by the use of restrictive diets and of therapeutic formulas may also play a role, together with an inefficient use of nutrients (higher needs, lower utilisation). Undernutrition may be the consequence of an uncontrolled, inappropriate, and/or excessively strict elimination diet (33,34). The nutritional risk is higher in cases of multiple food allergies: the elimination diet may easily result in deficiencies, especially if it includes multiple exclusions. In a study conducted by Christie et al (35), children with at least 2 food allergies were slightly shorter (height for age percentile) than those with a single food allergy. Also, >25% of children consumed less than two-thirds of the dietary reference intakes for calcium, vitamin D, and vitamin

E. The low calcium intake was especially marked in children with CMPA or multiple allergies.

All of these data suggest that children with milk allergy need nutritional counselling that considers not only the total energy needs of the patients but also the peculiarities deriving from the use of particular exclusion diets involving foods, not just single components. Because the atopic status itself may represent an "at-risk" condition of poor growth in the first year of life, "individually tailored" elimination diets may be required to sustain adequate growth in this population, while avoiding dietary imbalances. Maybe nutritional benefits will be shown from the recently introduced specific oral tolerance induction procedures, showing the major advantage of substantially reducing the risk of severe reactions after accidental ingestion of the allergen (36,37).

AUTISM SPECTRUM DISORDERS

The mass media and easy access to the Internet have progressively increased the popularity of some beliefs, including that some observed and diagnosed functional disorders of the brain activity could be related to food components, including food additives or food protein components. Gastrointestinal disorders and associated symptoms are also commonly reported in individuals with ASDs, but key issues such as the prevalence and best treatment of these conditions are incompletely understood. More than 30 years ago, behavioural disorders induced by administration of low doses of morphine in rats were compared with some characteristics of autism in children, leading to the proposition that behavioural disturbances in autism can result from abnormal activation of the opioid system because of an excess of agonists in the brain (38). Gluten in cereals and casein in milk were identified as important sources of peptides with opioid activity (exorphins) (39). According to this hypothesis, some of the disorders in ASD can be related to excessive amounts of dietary exorphins, a theory that is still unconfirmed but is the main rationale for avoidance diets in these diseases.

This "mal-awareness" has resulted in the self-prescribed removal of gluten-containing cereals and cow's milk with milk-protein-containing foods that has become the most widely used method of alternative management in ASD in the United States (40). The risk in this situation of nutritional imbalances for a growing organism is high, considering that both treated children with coeliac disease and allergic patients (see previous section) may meet nutritional deficiencies even if regularly followed up. Despite the selective nature of their eating patterns, children with ASD seem to have growth similar to that of populations of children taken as a reference; however, there are no data available on the growth pattern or nutritional status of children with ASD following a gluten-free and casein-free diet. Therefore, it is not possible to confirm whether such a diet has no harmful effects in the short, medium, or long term. A consensus document was published in 2010 on the evaluation, diagnosis, and treatment of gastrointestinal disorders in individuals with ASD (41,42). Accordingly, "available research data do not support the use of a casein-free diet, gluten-free diet, or combined gluten-free, casein-free diet as a primary treatment for individuals with ASDs." Nevertheless, some parents take the decision of feeding their child with a restricted diet. In this situation, they need information to help plan a balanced diet within the restrictions imposed by the chosen diet. Given the real hardships associated with implementation of a strict gluten-free, casein-free diet, additional studies are needed to assess risk factors and possible markers that identify individuals who may benefit from these diets. CMPA should be considered in any case if specific gastrointestinal symptoms are present.

INCREASED RISK OF TYPE 1 DIABETES MELLITUS

Is there a biological plausibility for a role of cow's-milk proteins in type 1 diabetes mellitus development? In 1968, genetic differences were described for the first time in milk proteins between individual cattle and breeds (43). In northern Europe, A1 was the predominant form of β -casein in cow's milk (with a range of variation of the A1/ β ratio from 0.46 to 0.71). In other breeds, the ratio was lower up to 0.25 (44). The distinctive peptide formed mostly from A1 β -casein is β -casomorphin-7 (BCM7), considered the active ingredient (45). A clear direct relation was shown between the daily per capita consumption of A1 β -casein and the type 1 diabetes incidence per year in most Western countries (46). It was speculated that a higher A1/ β ratio may result, through immune-related biological processes, in a permanent change of the islet cells, making them prone to other factors or processes leading to apoptosis in later life. Truswell (47) critically reviewed the matter, pointing out that the β -casein A1 allele is unique in producing β -casomorphins with opioid properties. β -Casomorphins may inhibit human intestine lymphocyte proliferation, thus suppressing tolerance and/or defence mechanisms in the presence of other concurring episodes of infection, for instance. Even if it is possible that early feeding of cow's milk to infants who are genetically susceptible to type 1 diabetes may increase the risk of their developing diabetes, published case-control studies included nearly as many that showed no risk. Considering the meta-analysis published by Gerstein (48) and the main publications up to 2005, Truswell concluded that the relative risk was as little as >1.0 , ranging from about 1.5 for early cow's-milk exposure or <3 months' breast-feeding to 1.1 for non-breast-feeding infants, assessed with feeding records rather than mothers' recollections (49). In conclusion, there was no convincing or probable evidence that the A1 β -casein in cow's milk was a factor causing type 1 diabetes.

The European Food Safety Authority was asked for an opinion on the potential health effects of β -casomorphins and related peptides, and concluded in 2009 that even if autoantibodies found in type 1 diabetes have not been proven to be directly involved in disease progression, the development of type 1 diabetes is the result of a combination of genetic predisposition and environmental risk factors (50). Although some ecological studies have linked the intake of BCM7 with type 1 diabetes, a cause-effect relation between the oral intake of BCM7 or related peptides and noncommunicable diseases cannot be established.

The recent publication of the 10-year observations from the second trial of the Trial to Reduce Insulin-dependent Diabetes Mellitus in Genetically at Risk study (Trigger), inclusive only of Finnish participants, has raised again the discussion on the possible role of cow's-milk proteins in the pathogenesis of type 1 diabetes (51). The tested hypothesis was that supplementing breast milk with greatly hydrolysed cow's-milk formula would decrease the cumulative incidence of diabetes-associated autoantibodies in children with genetic susceptibility (ie, human leukocyte antigen-conferred susceptibility to type 1 diabetes and at least 1 family member with type 1 diabetes). Accordingly, 230 infants were randomised to receive either a casein (100%) hydrolysate formula or a conventional, cow's-milk-based formula (control: 80% protein made up of whey and casein, 20% hydrolysed casein) whenever breast milk was not available during the first 6 to 8 months of life. At 10 years, the positivity for 1 or more autoantibodies was 46% lower for the casein hydrolysate group. Therefore, dietary intervention decreasing exposure to intact cow's-milk proteins during infancy appears to have a long-lasting effect on markers of β -cell autoimmunity, which may reflect an autoimmune process playing a role in the

occurrence of type 1 diabetes. Development to type 1 diabetes was similar in the 2 groups, and therefore the clinical meaning is still debated. Other studies have highlighted the possible interaction between the genetic susceptibility to type 1 diabetes and dietary exposure in the development of the disease, showing that high consumption of cow's milk during childhood (≥ 3 glasses/day, equivalent to 540 mL) can be diabetogenic in siblings of children with type 1 diabetes (52).

METABOLIC SYNDROME AND CHRONIC DEGENERATIVE DISORDERS

The question of the possible association between cow's-milk consumption and the facilitated development of metabolic syndrome and chronic degenerative disorders later on is sensitive because it is strictly connected to the main functional effects of cow's milk, that is, its growth-promoting effect. There is a general consensus that the growth-promoting effect of cow's milk is mainly mediated by insulin-like growth factor-1 (IGF-1). Indeed, through an elegant series of experiments, a group in Copenhagen has shown, step by step, that IGF-1 levels in blood are associated with the consumption of animal versus vegetable proteins and milk versus meat (with increase in milk intake from 200 to 600 mL leading to a 30% increase in IGF-1 level) (53), skim milk versus lean meat (54), and casein versus whey (55). Milk consumption is also associated with increased insulin secretion and resistance in the short term when compared with meat (56), and whey has been shown to increase insulin levels versus casein (55). This observation could explain the dissociation between the glycaemic and the insulinemic curves when whole cow's milk and skim milk are tested against glucose. Although the glycaemic index is lower than for glucose and similar for both whole and skim cow's milk, the insulinemic response is the opposite, being higher for both types of milk compared with glucose (57). Maybe additive components such as lactose or some amino acids could stimulate insulin secretion with a resulting anabolic effect without altering the glucose response. The growth-stimulating effect of milk has been recognised since 1928, from the effects on height with intervention diets similar as far as energy supply from school lunch but differing in milk content (58), up to the more recent observations of recovery and catch-up length and weight in young children on a vegan diet, wherein a minimum supplement with fish per week and fat plus milk per day was effective (59).

Warnings about the excess of proteins given in early childhood and the risk of a negative effect on growth stimulation started with the observation of Rolland-Cachera et al (60) of an earlier adiposity rebound in association with a higher intake of proteins. According to a few other studies, when average protein intakes at early ages are particularly high ($\geq 15\%$ energy), early adiposity rebound and associations with measures of adiposity later on are observed (61). A stimulatory effect of IGF-1 on preadipocytes is possible, but the precise mechanisms supporting a full biological plausibility are still unclear (62). In any case, it should be noted that the association is with the general protein intake, and no direct involvement of cow's milk is described.

The possible double face of cow's milk growth-promoting effects, from a virtuous circle to a vicious circle, has been extensively reviewed by Hoppe et al (63). Cow's milk contains nutrients directly acting on body composition (calcium and vitamins, for instance) and bioactive peptides with indirect action on hepatic synthesis and pituitary secretion of growth-promoting agents. Both of these components may affect biomechanisms, potentially influencing the expression of noncommunicable diseases, according to the genetic background, on a theoretical basis.

As a matter of fact, results from observational studies are not univocal. An inverse relation between protein intake and blood pressure levels has been found in 2.5-year-old Danish children (64). Because children in the study received 33% of their protein from milk or dairy products, the effect of protein could be related to some bioactive components in milk. Within the Framingham Children's Study, 99 children, 6 years at baseline, were followed up to adolescence (65). The lowest sex-specific tertiles of dairy intake in preschool (<1.25 servings/day in girls and <1.70 in boys) had greater gains in body fat in childhood (≥ 3 additional millimetres of subcutaneous fat per year in the sum of 4 skinfold measurements). Consistent with these findings, the Coronary Artery Risk Development in Young Adults (CARDIA) study considered the 10-year cumulative incidence of insulin resistance syndrome components (practically all of the major components of the metabolic syndrome) by categories of total dairy intake with stratification by baseline overweight status in young adults. Dairy consumption was inversely associated with the incidence of all of the insulin resistance syndrome components among individuals who were overweight at baseline, leading the authors to conclude that dietary patterns characterised by increased dairy consumption have a strong inverse association with insulin resistance syndrome among overweight adults and may reduce the risk of type 2 diabetes mellitus and cardiovascular disease (66).

It can be concluded from the previous observations that there is no available evidence that cow's-milk consumption in childhood plays a deleterious role in the later risk of chronic degenerative, noncommunicable disorders.

CANCER

The observations on the associations between intake of milk or dairy products and some types of cancer are not univocal. The 2007 Report of the World Cancer Research Fund/American Institute for Cancer Research (WCRF/AICR) on food, nutrition, physical activity, and the prevention of cancer concluded that "the evidence on the relationship between milk and dairy products, and also diets high in calcium, and the risk of cancer, points in different directions. Milk probably protects against colorectal cancer. Diets high in calcium are a probable cause of prostate cancer; there is limited evidence suggesting that high consumption of milk and dairy products is a cause of prostate cancer" (67). Among the possible causes are a downregulation of 1,25-(OH)₂ vitamin D, a greater intake of conjugated linoleic acid (LA), exposure to contaminants such as polychlorinated biphenyls, as well as the IGF-1-stimulating effect (68). Few data are available on the role of childhood dairy or milk intake and cancer risk in adulthood. A study from the British Boyd-Orr cohort found a near-tripling in the odds of colorectal cancer (odds ratio [OR] 2.90; 95% confidence interval 1.26–6.65) in the highest versus the lowest quartile range of childhood dairy intake, independent of meat, fruit, and vegetable intakes and socioeconomic indicators (69). Childhood milk intake showed a similar association with colorectal cancer risk. A recent study from New Zealand showed that participation in school milk programs from 1937 to 1967 was associated with a reduced OR for colorectal cancer (OR 0.70; 95% confidence interval 0.51–0.96), with a 2.1% reduction in the OR for every 100 half-pint bottles drunk (1 half-pint bottle = 284 mL) (70).

Because the avoidance of milk and dairy products should lead to the necessity of supplementing diet with integrators, we should do well to remember that, in absence of a clear cause–effect relation, the guidelines of the major cancer societies recommend meeting nutritional needs through natural foods and not supplements, according to strategies consistent with general public health guidelines. Randomised clinical trials have produced strong evidence that high-dose supplements of some nutrients increase cancer risk. Therefore, the 2007 WCRF/AICR report also concluded that

from an individual point of view, dietary supplements are not recommended for cancer prevention (67).

IS MILKFAT THE CAUSE?

One of the milk components often discussed for the possible negative consequences on long-term health is fat. Fat composition of cow's milk is not 1-way. It is true that cow's milk is mainly made up of saturated fats, with a contribution of 65% to 70% from myristic (14:0) to stearic (18:0) acid, but it includes also short-, medium-, and intermediate-chain fatty acids. LA (18:2n-6) is low, around 2%, and α -linolenic acid (18:3n-3) is slightly lower but extremely variable, 0.2% to 1.2%. The resulting LA/ α -linolenic acid ratio is variable, around 4 to 10:1, thus allowing for a more favourable predisposition towards the individual synthesis of the derivatives longer-chain n-3 polyunsaturated fatty acids, especially docosahexaenoic acid (22:6n-3) even if whole cow's milkfats include small amounts of arachidonic acid (20:4n-6). Thus, infants fed cow's milkfat show a long-chain polyunsaturated fatty acid status ranging between those fed formulas enriched with vegetable oils and those fed breast milk (71). It is even possible to raise levels of DHA with breeding techniques that are in any case difficult to maintain (72). Finally, milkfat also contains fatty acids originating from the elaborate digestive processes, such as *trans*-vaccenic and conjugated LA. Overall, increased consumption of full-fat dairy products and naturally derived *trans* fatty acids seems not to cause significant changes in cardiovascular disease risk variables, as may be expected on the basis of the present health recommendations (73), even if excess consumption should be avoided (74). Beyond composition, cow's milkfat represents a rich source of energy for the first years of life. The Committee on Nutrition of the European Society for Pediatric Gastroenterology, Hepatology, and Nutrition recommended that the fat content of the diet should be above, not below, 25% of total energy intake and that lowfat milks (1.5%–2%) should be recommended from >2 to 3 years onward, depending on the growth curve and family history of chronic degenerative disorders. Lowfat milk may limit energy intake and thereby growth (15); however, with the present obesity epidemic that affects both preschool children and older children, the potential beneficial effects of lowfat milk on energy intake and later preferences should also be taken into account.

CONCLUSIONS

Cow's milk represents a major source of protein of high nutritional quality and calcium. After briefly reviewing all of the major issues connected with the potential of cow's milk to be harmful to children's diet, we summarise:

1. Negative effects of cow's-milk consumption on iron status are possible up to 9 to 12 months; then no negative effects are observed, provided that cow's milk, limited to an optimal daily intake of 500 mL, is adequately complemented with iron-enriched foods and other relevant nutrients.
2. Lactose intolerance can be easily managed. There is no need for eliminating dairy foods and milk that could be consumed up to 250 mL/day.
3. Allergy to cow's-milk proteins is usually transient. Atopic children may independently be at risk for poor growth, and the contribution of dairy nutrients to their diet should be considered.
4. The connection between cow's milk and ASDs is lacking.
5. A cause–effect relation with type 1 diabetes mellitus has not been established, and many factors may concur.
6. Cow's milk stimulates IGF-1 and may affect linear growth, but association with chronic degenerative, noncommunicable diseases has not been established.
7. Reduced-fat milks should be considered after 24 to 36 months.

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