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Pregnancy, Child Nutrition, and Oral Health

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1. INTRODUCTION

Growth and development begin with pregnancy and continue through adolescence and early adulthood. Growth is defined as increases in cell size caused by processes of cell multiplication involving hyperplasia, hypertrophy, and accretion. Development involves a progressive maturation, differentiation, or specialization that results in a final physical, emotional, psychological, and cognitive biological state. The effects of nutrition are manifested throughout this process and in all tissues and structures in the body (1).

Both general nutrition and dietary intake of specific nutrients have been associated with oral growth and development. Dietary choices throughout life can have a primary effect on the tooth structure, whereas nutritional status exerts a systemic effect on the integrity and maintenance of other oral tissue (2). However, research in this area has been limited to animal studies and a few preliminary human investigations. Lack of an extensive body of evidence to identify associations between specific nutrients and optimum oral growth and development has resulted in limited interpretations of the precise interactions.

The purpose of this chapter is to examine the synergy between oral health development and nutritional status from conception to adolescence and to review past, present, and future research in this area according to stages of development that include pregnancy, infancy, early childhood, and school-aged children. Finally, this chapter identifies issues in managing oral health and promoting nutrition for special-needs individuals during pregnancy and childhood.

2. PRENATAL AND PERINATAL NUTRITION AND TOOTH DEVELOPMENT

The nutritional intake of the pregnant woman has specific and global effects on the dentition of her child. The specific effects are related to the formation of the enamel and dentin of the primary and permanent teeth during fetal development. The global effects are related to the amount of weight she gains, her diet’s overall nutritional quality, how much her infant weighs at birth, and the gestational age of her infant—that is, whether
the infant is delivered at term or preterm. Primary teeth begin to form at 6 wk of gestational age when cells in the fetal oral cavity begin to differentiate and form tooth buds. The dentin layer forms first and then the enamel layer is deposited (3). Mineralization begins at 4 mo in utero, and central incisors (the teeth that erupt first) have 83% of their enamel formed by the time of birth (3). Insults from teratogens or lack of crucial nutrients during pregnancy can have significant impact on the nearly developed primary teeth and the beginnings of the permanent teeth. Infants are born with all the primary teeth and many permanent unerupted teeth in varying stages of development (4,5). A review of early childhood caries (ECC) and hypoplasia in infants and children in developing countries revealed that these conditions were most closely associated with a general underlying nutritional deficiency state (malnutrition or undernutrition) in the perinatal period (6). A clear relationship has been found between specific dietary nutrients during critical periods of calcification and poorly calcified teeth, which reduces caries resistance (7).

2.1. Maternal Protein Energy Malnutrition

Maternal malnutrition, specifically protein energy malnutrition (PEM), has global effects. A pregnant woman needs to gain enough weight to support the fetus, placenta, and associated gain in maternal tissues to support pregnancy and lactation. Generally, a weight gain of 27.5 lbs (12.5 kg) is considered adequate, but the recommended weight gain for a specific woman depends on her prepregnancy body mass index (BMI) (8,9). The requirement for protein increases by 20%, to a total of approx 60 g per day (10) depending on body weight and age. A woman who enters pregnancy underweight with low protein stores and who does not gain sufficient weight during pregnancy to support the fetus and her increased metabolism is at risk of delivering a low birth weight (<2500 g) and/or preterm infant (11).

Low birth weight and preterm delivery are associated with enamel hypoplasia of the primary and permanent teeth. Nutritional deficiencies during pregnancy can affect tooth size, timing of tooth eruption, defects in enamel mineralization, and salivary gland formation and can create increased susceptibility to caries (12,13). Furthermore, developmental defects of enamel and enamel hypoplasia are seen as a result of malnutrition during pregnancy and early childhood (14–16). When nutritional deficiencies or toxicities occur during “critical periods” of oral tissue development, consequences can be permanent and irreversible (17). Turnover time for oral soft tissue is between 3 and 7 d. This is more rapid than in other tissue and may increase oral tissue needs for nutrients beyond those in tissue with longer turnover rates (3). Female rats fed a low-protein diet gave birth to rats with smaller molars, impaired salivary gland function (which affects caries resistance), and delayed eruption of the first and second molars (7). When female rats consumed protein- and calorie-deficient diets, the molars and incisors of the offspring weighed less than those of the normally fed control group (18). These defects can be permanent and irreversible because of the absence of enamel and dentin regenerations once tooth eruption occurs (19,20). Eruption of primary teeth has been delayed in longitudinal studies of stunted infants compared to healthy infants and associated with a risk of caries later in life (21).

2.2. Maternal Micronutrient Malnutrition

Deficiencies in micronutrients such as vitamins A, C, and D and minerals such as calcium, phosphorus, fluoride, iron, and iodine have an effect on developing dentition (22).
Vitamin A deficiency is widespread and often accompanies protein–calorie malnutrition. It is especially prevalent when dairy product and fresh fruit and vegetable intakes are limited (23). It can exert its effect on dentition through the global effect of malnutrition as well as from a specific deficiency. Vitamin A deficiency can increase the risk of maternal mortality and is associated with preterm birth, intrauterine growth retardation, and low birth weight (24). A vitamin A deficiency that occurs during gestation results in decreased epithelial tissue development, tooth morphogenesis dysfunction, and decreased odontoblast differentiation in fetal development (3). It is thought that a lack of vitamin A produces chemical changes in the dentin that reduce the extent of mineralization (25). However, vitamin A can be teratogenic when ingested in the form of retinoic acid analogs, given to treat severe cases of acne vulgaris. The β-carotene form of vitamin A found in fruits and vegetables is not teratogenic (26).

Vitamin C plays a role in the integrity of osteoblasts, fibroblasts, chondroblasts, and odontoblasts (27–29). It is necessary for synthesis of collagen, which contributes to the organic matrix for the deposition of calcium phosphate crystals that occurs during bone mineralization (27,30). In vitro odontogenesis is constrained by vitamin C deficiency caused by dedifferentiation of odontoblasts, which leads to a cessation of dentin production (27,30). A positive correlation \( r = 0.245, p = 0.033 \) was reported between higher intakes of vitamin C and changes in bone density in 76 subjects at 10–20 wk gestation (31). Ogawara (30) studied vitamin C deficiency in rats and concluded that it causes a marked reduction in dentin formation. The recommended dietary intake (RDI) for vitamin C is increased by 67% during pregnancy and requires daily ingestion because it is a water-soluble vitamin. Many fresh fruits and vegetables provide excellent sources. A long-term deficiency of vitamin C leads to scurvy, which can cause swollen, bleeding gums and loss of teeth. Vitamin C also works in concert with vitamin A, as collagen and calbindin (a product of vitamin A), to promote mineralization and development of teeth (27).

Vitamin D deficiency most often occurs when the dietary intake of vitamin D–fortified milk is deficient or when pregnant women choose clothing that inhibits exposure to sunlight (32). Effects of vitamin D deficiency by itself are difficult to separate from the effects of calcium and phosphorus deficiency because vitamin D causes the effect of hypomineralization through lowering plasma calcium levels (3,33). The effects of calcium, phosphorus, and vitamin D deficiency result in lowered levels of plasma calcium, hypoplastic effects/hypomineralization, compromised tooth integrity, and a delay in tooth eruption. Permanent as well as primary teeth are affected. Likewise, animal and human studies indicate that excess vitamin D can result in disturbances in tooth calcification and hypoplasia that are not reversible (17).

The demands of calcification of the fetal skeleton and mineralization of the primary teeth result in an increased need for calcium during gestation. The permanent teeth begin mineralization just before fetal maturity is reached (7). The RDI for calcium increases to 1000 mg per day for pregnant women (33). Yet, many women do not ingest this amount, even though calcium is found in all dairy foods, many fruits and vegetables, and some dietary sources of protein (34). Hypomineralization of primary teeth from deficiencies of vitamin D, calcium, and phosphorus can contribute to increased susceptibility to ECC (35,36).

Fluoride increases the resistance of teeth to decay by increasing mineralization. Fluoride supplementation does not seem to benefit the developing fetus (37) even though it diffuses
across the placental barrier and is incorporated into fetal bones and teeth. The uptake of fluoride into calcified teeth is most dramatic during infancy but decreases with age (3).

Iron and iodine are minerals closely associated with promoting oral health because of their effect on the development of fetal dentition (22,38). They are crucial for the health of the developing fetus. Iron-deficiency anemia is a serious condition for a pregnant woman (11). It can result in reduced oxygen-carrying capacity in the mother, leading to hypoxia for developing fetal tissues and reduced iron deposition in these tissues. Iodine is critical for the formation of thyroid hormones, and its deficiency can cause mental retardation in a fetus. Because it may be difficult for a pregnant woman to meet the dietary reference intake for iron exclusively from foods, iron supplementation of 30–60 mg is recommended (11). Iodine consumption can be easily accomplished through inclusion of iodized salt in the diet. However, for women on salt-restricted diets because of hypertension, elemental iodine is available in selected prenatal supplements.

Table 1 lists vitamin and mineral recommendations for promotion of fetal and infant health (39,40). Dietary sources are listed, and recommendations for supplementation are noted. A prenatal vitamin and mineral supplement has become part of routine prenatal care for women planning a pregnancy.

3. INFANT AND EARLY CHILDHOOD FEEDING PRACTICES AND ORAL HEALTH

Rate of growth during both fetal life and infancy has been associated with long-term consequences for bone health as well as cardiovascular risk and brain development in infants (41). Dietary and feeding practices have a primary influence on infant growth. The World Health Organization (WHO) recommended exclusive breastfeeding up to or beyond 2 yr of age (42). Recommendations were not given on introduction of solid foods for formula-fed infants even though there is evidence that introduction before 4 mo of age may be associated with increased body fat, higher BMI, increased incidence of respiratory illness in later childhood, and increased risk for ECC. A report including several cohorts of greater than 2000 infants born in the United Kingdom in the mid-1990s noted that weaning occurred on average 4 mo earlier than recommendations from the Department of Health and that this earlier weaning was associated with less positive health behaviors and significantly higher infant body weights at 6 mo of age (41). A significant relationship was identified between reported weaning practices and ECC in more than 1000 Hispanic children of women participating in the Women, Infants, and Children (WIC) Supplemental Feeding Program in south Texas, which suggests that feeding practices may modulate the incidence of ECC (43).

The fluoride content of breast milk is low (3,44). Fluoride inhibits demineralization, encourages remineralization, and increases the stability of the tooth enamel (3). Fluoride is most effective when ingested during infancy beginning at 6 mo of age through supplemented community water or through the use of infant formula constituted with fluoridated water that contains less than allowable optimal amounts of fluoride (0.3 ppm). Fluoride supplementation (in the form of drops) is not recommended during the first 6 mo of life (45). In the absence of access to fluoridated water, the infant’s or child’s physician may prescribe fluoride-containing vitamin supplements. Guidelines for fluoride supplementation have been established for children between 6 mo and 16 yr of age, based on the fluoride ion level in the drinking water and the use of other fluoride sources (46).
Table 1

Daily Vitamin and Mineral Recommendations During Pregnancy

<table>
<thead>
<tr>
<th>Vitamin</th>
<th>Recommended intake</th>
<th>Quantity in standard prenatal supplement</th>
<th>Some recommended food sources</th>
<th>Supplementation recommended?</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>770 RE</td>
<td></td>
<td>Fish oils, dark-green vegetables, and deeply colored fruits</td>
<td>No</td>
</tr>
<tr>
<td>B1</td>
<td>1.4 mg</td>
<td></td>
<td>Green leafy vegetables, lean pork, soy milk, enriched whole grain breads and cereals</td>
<td>No</td>
</tr>
<tr>
<td>B2</td>
<td>1.4 mg</td>
<td></td>
<td>Green vegetables, eggs, milk, meats</td>
<td>No</td>
</tr>
<tr>
<td>B6</td>
<td>1.9 mg</td>
<td>2 mg</td>
<td>Wheat germ, pork, cereals, legumes</td>
<td>No</td>
</tr>
<tr>
<td>B12</td>
<td>2.6 μg</td>
<td>50 mg</td>
<td>Meats, poultry, fish, shellfish, milk, eggs, cheese</td>
<td>No</td>
</tr>
<tr>
<td>C</td>
<td>85 mg</td>
<td>50 mg</td>
<td>Dark-green vegetables, citrus fruits</td>
<td>No</td>
</tr>
<tr>
<td>D</td>
<td>5 μg</td>
<td>5 μg</td>
<td>Fortified milk, egg yolks, fatty fish</td>
<td>No</td>
</tr>
<tr>
<td>E</td>
<td>15 mg</td>
<td></td>
<td>Polyunsaturated plant oils, wheat germ, tofu, avocado, sweet potatoes</td>
<td>No</td>
</tr>
<tr>
<td>K</td>
<td>90 mg</td>
<td></td>
<td>Leafy green vegetables, cabbage, cheese</td>
<td>No</td>
</tr>
<tr>
<td>Folate</td>
<td>600 μg</td>
<td>400–600 μg</td>
<td>Dark-green leafy vegetables, beans, peas, lentils</td>
<td>Yes</td>
</tr>
<tr>
<td>Niacin</td>
<td>18 mg NE</td>
<td></td>
<td>Peanut butter, lean ground beef, chicken, tuna, shrimp</td>
<td>No</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Mineral</th>
<th>Recommended intake</th>
<th>Quantity in standard prenatal supplement</th>
<th>Some recommended food sources</th>
<th>Supplementation recommended?</th>
</tr>
</thead>
<tbody>
<tr>
<td>Iron</td>
<td>27 mg</td>
<td>30 mg</td>
<td>Spinach, broccoli, tofu, shrimp, iron-fortified cereals</td>
<td>Yes</td>
</tr>
<tr>
<td>Calcium</td>
<td>1000–1300 mg</td>
<td>250 mg</td>
<td>Dairy products including milk, yogurt and cheese; leafy green vegetables; almonds; calcium-fortified foods</td>
<td>No</td>
</tr>
<tr>
<td>Phosphorus</td>
<td>700–1250 mg</td>
<td></td>
<td>All animal foods (meats, fish, poultry, eggs, milk)</td>
<td>No</td>
</tr>
<tr>
<td>Zinc</td>
<td>11–13 mg</td>
<td>15 mg</td>
<td>Lentils, shrimp, crab, turkey, pork, lean ground beef, eggs, tofu</td>
<td>No</td>
</tr>
</tbody>
</table>

*a Source: Institute of Medicine, refs. 33,40.

*b Supplementation is recommended if foods high in calcium are not consumed.

*c Both iron and copper compete with zinc at absorption sites; therefore, zinc supplementation is recommended when elemental iron supplementation exceeds 60 mg/d. RE, retinol equivalents; NE, niacin equivalents.
3.1. Health Consequences of Early Childhood Caries (ECC)

The simultaneous presence of cariogenic microorganisms, fermentable carbohydrate, and a susceptible tooth and host initiate the infectious and transmissible disease known as dental caries in adults and ECC in children. At a microscopic level, the biology of caries is the same for adults and children. However, in children this disease can have a negative impact on a child’s diet, nutritional status, sleep patterns, psychological status, and, later on, school attendance. Children have been reported to experience pain with ECC that adults would not endure. ECC has been described as a virulent form of caries that starts soon after teeth erupt, and it proceeds rapidly to involve primary molars and canines (6).

Children who develop ECC are more likely to experience delayed development. Several ECC interventions have been identified in the literature as important for the amelioration of a commonly coexisting condition of ECC and the condition of growth faltering (45,47,48). Acs et al. (49) found that correcting ECC could lead to an accelerated velocity of weight gain, resulting in the improvement of the growth faltering. Long-term effects of reversed growth faltering on health status or, conversely, prevention of childhood obesity are unknown. Because ECC has been closely associated with underlying nutritional deficiencies in the perinatal period (6), it is likely, as the disease progresses, that developmental eating behaviors and nutritional status are threatened.

3.2. Recommended Infant and Early Childhood Feeding Practices to Promote Oral Health

Nutrient- and energy-dense liquids are supplemented and partially replaced with solid foods in the infant’s diet when developmental readiness occurs. Thus, introduction of pureed and solid foods is recommended to parallel physiological, emotional, and cognitive development. Guidelines to ensure maintenance of adequate nutrient intake to support growth, development, and oral health are in sync. Introduction of foods to the diet to diminish the transmission of oral bacteria from the caregiver to the child and to include a variety of textures and consistency may be important to craniofacial and occlusal development. Animal studies have shown that animals fed only soft diets had smaller maxillary and mandibular arch widths and lengths than those fed hard diets (50). A variety of foods further supports nutrient needs and enhances mastication, which leads to salivary production beneficial in decreasing caries risk. Table 2 outlines recommendations for oral health promotion among infants and young children based on policy statements of the American Academy of Pediatric Dentists and the American Academy of Pediatrics (51,52).

4. SCHOOL-AGE CHILDREN AND ORAL HEALTH

Approximately 50% of children have experienced dental decay or caries by the age of 8 despite the fact that oral health has improved in the past few decades. This diet-dependent infectious bacterial disease remains one of the most common diseases of childhood. According to the Surgeon General’s Report on Oral Health in America, it is five times as common as asthma and seven times as common as hay fever. Some racial or ethnic minorities, homeless and migrant populations, children with disabilities, and those with human immunodeficiency virus (HIV) infection often present with the most advanced forms of caries. If those with dental caries remain untreated, serious general
health problems can result. It has been reported that among low-income children, almost 50% of caries remain untreated. Thus, these children may experience pain, inability to chew well and eat, failure to gain weight, and embarrassment because of discolored and damaged teeth. These circumstances can reduce a child’s capacity to succeed in school and can alter self-esteem and health behavioral self-efficacy. Dental-related illness results in more than 51 million lost school hours each year. Likewise, head, neck, and mouth trauma resulting from violence, sports, falls, and motor vehicle crashes create additional oral health problems for children.

4.1. Food Choices and Frequency of Dietary Intake

Caries risk throughout childhood remains dependent on the synergy existing among oral bacterial count, general health status, fluoride exposure, and dietary patterns. Diet is one of the significant factors in modulating the direction of the dynamic demineralization and remineralization process of the tooth enamel. Diet cannot alter the percentage of acidogenic bacteria in dental plaque, but the dietary composition and frequency of exposure can alter the pH of the environment adjacent to the tooth as well as promote saliva production. Saliva buffers dental plaque pH and promotes tooth remineralization and oral clearance. When plaque pH is measured following the consumption of a fermentable food or beverage, it can drop from neutral (pH 7.0) to a critical level below 5.7 within a 5-min time frame. It takes approx 40–60 min and a maximum of 120 min to return to neutral, particularly if interventions such as oral hygiene or xylitol/sorbitol sugar-free gums are not introduced. Thus, food choices and frequency of dietary intake are major determinants of caries risk.
4.2. Snacking and School Environments

The school environment provides a venue for a significant portion of a child’s experiences with lifestyle behaviors that affect health. Using well-defined protocols to distinguish snack and meal consumption patterns, nationally representative data from three large surveys conducted between 1977 and 1996 described increased snacking prevalence in US children (55). Groups of foods eaten within a 15-min time span were counted as one snack. Children between the ages of 2 and 5 yr and 6 and 11 yr reported significant increases in the number of snacks eaten daily as well as the kilocalories from those snacks. In 1996, 90% of children snacked, whereas only 80% did so in 1977. Total daily energy intake from snacks rose from about 18% to 25% during this time frame. Snack intake reflected an increased consumption of soft drinks, potato and corn chips, and other kinds of salty snacks and a simultaneous drop in fruit, vegetable, and milk consumption. Energy consumption from meals remained constant over time, and children between 6 and 11 yr decreased their intake of calcium and protein; however, there was no change in vitamin A and fiber intake from these foods. Although it was reported that the average size of snacks and the energy per snack remained relatively constant, the number of snacking occasions had increased significantly. These trends are related to both childhood obesity and dental health status (55).

A smaller study conducted in Finland reported that children who had candy and juice more than once a week when they were 3 yr old generally consumed more sucrose 3 yr later and had more visible plaque, leading to a higher incidence of carious lesions by the age of 6 yr (56). Those who had sweets once a week or less had less plaque and fewer lesions at 6 yr of age.

Snacking is essential to the dietary pattern of children for adequate nutritional support. Because both the food and beverage snack choices and the frequency of their consumption have implications for caries risk, guidelines for nutrition educators can augment and enhance their role in the promotion of children’s overall health and well-being. Table 3 (2,20,55–61) suggests specific guidelines for children between the ages of 3 and 10 yr.

5. SPECIAL NEEDS CHILDREN, NUTRITION, AND ORAL HEALTH

5.1. Cleft Lip and Palate

Studies suggest that the development of cleft lip and palate are related to a gene–environment interaction (62). Studies have shown that maternal multivitamin supplement use protects against cleft lip and palate (63,64). In a Danish population, higher levels of vitamin A intake from multivitamins and liver sources also seemed to protect against cleft lip and palate (65). Further studies have suggested that the protection was not entirely explained by multivitamin use, indicating that adequate levels of vitamin A may be required for normal development of the primary palate (62). Other authors have identified other risk factors, including cigarette smoking and alcohol consumption, use of anticonvulsant drugs, and exposure to organic solvents. A protective effect has been shown for supplementation with folic acid (66). Maternal diabetes has also been implicated as an etiologic agent in cleft lip and palate. Mothers with diabetes were found to be 1.352 times (95% confidence interval, 1.004–1.821; \( p < 0.05 \)) more likely than nondiabetic mothers to have a newborn with cleft lip or palate (67). Early glycemic control by expectant mothers may be an important factor in decreasing the incidence of this congenital anomaly.
Table 3  
Oral Health Nutrition Messages for 3- to 10-Yr-Old Children and Their Caregivers  

<table>
<thead>
<tr>
<th>Message</th>
<th>Rationale</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Starchy, sticky, or sugary foods should be eaten with nonsugary foods.</td>
<td>The pH will rise if a nonsugary item that stimulates saliva is eaten immediately before, during, or after a challenge.</td>
<td>55</td>
</tr>
<tr>
<td>Combine dairy products with a meal or snack.</td>
<td>Dairy products (nonfat milk, yogurt) enhance remineralization and contain calcium.</td>
<td>56</td>
</tr>
<tr>
<td>Combine chewy foods such as fresh fruits and vegetables with</td>
<td>Chewy, fibrous foods induce saliva production and buffering capacity.</td>
<td>2</td>
</tr>
<tr>
<td>fermentable carbohydrates.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Space eating occasions at least 2 h apart and limit snack time to</td>
<td>Fermentable carbohydrates eaten sequentially one after another promote demineralization.</td>
<td>20</td>
</tr>
<tr>
<td>15 min.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Limit bedtime snacks</td>
<td>Saliva production declines during sleep.</td>
<td>57</td>
</tr>
<tr>
<td>Limit consumption of acidic foods such as sports drinks, juices, and</td>
<td>Acidic foods promote tooth erosion that increases risk for caries.</td>
<td>58</td>
</tr>
<tr>
<td>sodas.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Combine proteins with carbohydrates in snacks.</td>
<td>Proteins act as buffers and are cariostatic.</td>
<td>2,60</td>
</tr>
<tr>
<td>Examples: tuna and crackers, yogurt and berries, apples and cheese,</td>
<td></td>
<td></td>
</tr>
<tr>
<td>cookies and milk.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Combine raw and cooked or processed foods in a snack.</td>
<td>Raw foods encourage mastication and saliva production, whereas cooked or processed foods may be more available for bacterial metabolism if eaten alone.</td>
<td>2,60</td>
</tr>
<tr>
<td>Encourage use of xylitol/sorbitol-based chewing gum and candies</td>
<td>5 min exposure is effective in increasing saliva production and dental plaque pH. Excessive use may cause gastrointestinal distress.</td>
<td>59,61</td>
</tr>
<tr>
<td>immediately following a meal or snack.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sugar-free chewable vitamin–mineral supplements and syrup-based</td>
<td>Sugar-free varieties are available and should be suggested for high-caries risk groups.</td>
<td></td>
</tr>
<tr>
<td>medication should be recommended.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Encourage children with pediatric GERD to adhere to dietary guidelines.</td>
<td>GERD increases risk for dental erosion.</td>
<td></td>
</tr>
</tbody>
</table>

*Not recommended for children under the age of 4 yr. GERD, gastroesophageal reflux disease.*
All of the above studies of possible etiology reveal that adequate preconceptual and prenatal nutrition has an important role in the primary prevention of cleft lip and palate. Dietary recommendations for infants born with a cleft are no different from those for other infants. The delivery method of breast milk or formula can be altered to ensure adequate nutrient intake in preparation for successful future surgical outcomes (69). Chappelle and Nunn (68) showed that only 34% of 12-yr-old children with cleft lip and palate were free of caries. All the 4-yr-olds examined in their investigation had evidence of erosion of enamel in the primary teeth (incisors and first molars) and 56% of the 12-yr-olds had erosion of permanent teeth (incisors and first permanent molars). Lin and Tsai (70) found that children with clefts who took a bottle to bed showed an increased risk of developing ECC. The parents or caretakers of bottle-fed children allowed use of the bottle beyond 1 yr of age and also showed a lack of motivation to perform regular preventive dental home care for their children. This suggests that oral health promotion programs should begin in infancy for children with clefts and their parents, teaching them to wean their children from a bottle by age 1 yr, avoid nocturnal nursing bottles, and practice good oral hygiene from infancy.

5.2. Neural Tube Defects

The etiology of neural tube defects (NTDs) is unknown, although it is thought to be the result of a genetic predisposition manifested by the environment. It results from the disruption of the orderly formation of the vertebral arches and spinal cord that occurs between the fourth and sixth week of embryogenesis. According to recent evidence, NTDs may be related to the interaction of a genetic propensity for the defect and a deficiency of folic acid or poor metabolism of folic acid. Multivitamins containing folic acid taken during the first 6 wk of pregnancy can prevent more than 50% of NTD occurrence (71).

There are two common forms of NTDs: spina bifida occulta and myelomeningocele (meningomyelocele) (72). The form of NTD expressed as myelomeningocele has many associated problems, such as hydrocephalus, paralysis, orthopedic deformities, and genitourinary abnormalities. Children with closed spina bifida present with skin covering the area. The defect occurs in the vertebral column with tissue protrusion through a bony cleft. It usually is observed as a tuft of hair, a dimple, or a pigmented area above a defect. Sometimes with growth, children with this condition may develop foot weakness or bowel and bladder sphincter disturbances (73).

Children with NTDs often are on long-term liquid medications to manage these conditions. When these medications have high sucrose content, caries may result (74). Children with spina bifida have associated circumstances that make them prime candidates for dental caries. Many of these children tend to be overweight, which can be attributed to frequent snacking and impaired mobility because of the nature of their disability (73). Caloric intake for these children often exceeds energy expended and is coupled with a lack of stimulation from physical activities. Caregivers may use food as a demonstration of affection or as a reward. Without good oral hygiene, this creates a perfect environment for oral bacteria, making the teeth susceptible to decay. These children often are seen for extensive dental rehabilitation (75).

Few reliable statistics on how many children with spina bifida experience dental caries can be found. These children, however, are at greater risk for periodontal disease than the general population because of their disabilities and potential defects in the developmental formation of their enamel (76).
5.3. Mentally and Physically Handicapped

Children with mental retardation may be prone to childhood dental caries because their neurological impairment can make dental care difficult or impossible. Often, children with this disability need assistance with eating, clothing, and toileting from caregivers. Given all of these needs, diligent oral hygiene may be considered less important and thus may be neglected (71). Dental health in children with special needs is as essential as immunizations, regular physical examinations, and attention to injury prevention. Children with mental retardation may acquire dental caries because of poor dental hygiene, poor nutritional intake, and long-term medication therapy (77).

The dental status and treatment needs of Israeli children and adults with mental retardation were studied in a random sample of 387 subjects. Findings confirm high dental morbidity such as decayed and missing teeth and significant oral health differences by level of retardation, age, and dental clinic status. Specialized training designed to address treatment issues with handicapped populations is recommended for dental providers and the staffs of institutions to enhance oral health outcomes (78).

Parents and caregivers of handicapped children should be educated to clean the gums and mouths of children who do not have teeth yet with moist gauze. Children with disabilities need an appropriately sized toothbrush and require assistance with brushing. The use of an electric toothbrush can be beneficial for children who have poor plaque removal technique because of their lack of manual dexterity (79).

6. SUMMARY

Nutrition and diet are significant determinants of oral health status during pregnancy, infancy, and childhood. Adequate nutrition of the fetus during pregnancy that is extended into childhood increases the likelihood of establishing health behaviors that promote positive outcomes. Conversely, malnutrition can contribute to low birthweight, preterm delivery, failure to thrive, obesity, and altered growth patterns associated with increased risks for oral diseases. Dental professionals should address oral health issues specific to stages of growth and development and promote parenting behaviors in keeping with achieving good health.

Guidelines for Practice

<table>
<thead>
<tr>
<th>Oral Health Professional</th>
<th>Nutrition Professional</th>
</tr>
</thead>
<tbody>
<tr>
<td>Prevention</td>
<td></td>
</tr>
<tr>
<td>• Educate women who are planning pregnancies, are pregnant, or are parenting about the role of nutrition in the oral health of infants and children</td>
<td>• Include oral health screening in routine prenatal, infant, and child dietary assessment activities</td>
</tr>
<tr>
<td>Intervention</td>
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<tr>
<td>• Provide guidelines that promote oral health and support dietary changes necessary to decrease risk for ECC</td>
<td>• Provide messaging to parents about the synergy between nutrition and risk for oral disease</td>
</tr>
<tr>
<td>• Develop a referral protocol to a nutrition professional for comprehensive dietary counseling and monitoring</td>
<td>• Conduct dietary assessment and nutrition education with dental patients/clients who are parenting</td>
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<td>• Tailor dietary counseling to include guidelines to promote optimum oral health and disease risk reduction</td>
</tr>
<tr>
<td></td>
<td>• Develop a referral protocol to a dental professional for oral health maintenance</td>
</tr>
</tbody>
</table>
REFERENCES


43. Garcia-Godoy F, Mobley CC, Jones DL. Caries and Feeding Patterns in South Texas Preschool Children. San Antonio, TX: University of Texas Health Science Center at San Antonio, 1995


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