

## Malnutrition and its Effects on Oral Tissues and Dentition

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### ABSTRACT

Childhood malnutrition is generally thought of as being restricted to developing countries, but, although most malnutrition occurs there, it is also present in developed nations. During early developmental years of the child, it appears that malnutrition has severe effects on the growth and differentiation of different tissues. Even oral tissues, including the teeth, are very sensitive to the changes in the nutritional supply at these particular developing stages, thus increasing the ill effects on the oral structures. Tooth defects provide a more cariogenic environment and less protection which increases the susceptibility of teeth to demineralization. The shielding mechanism of saliva is also distressed during malnutrition, thus drastically affecting the oral environment.

**Keywords:** Protein-energy malnutrition, dental caries, saliva, enamel hypoplasia

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### INTRODUCTION

Malnutrition refers to the syndrome of inadequate intake of protein, energy, and micronutrients, combined with frequent infections [1]. Malnutrition may take the form of either undernutrition or overnutrition. Good eating habits and food preferences are established early in childhood. Poor nutrition can eventually lead to poor health, obesity, tooth decay, and periodontal disease. Protein-energy malnutrition (PEM) occurs when there are deficiencies in protein-energy foods, or both, relative to the body's needs. Mild PEM has an acute course, the main deficiency being in energy; moderate PEM is chronic in nature and is mainly protein deficient in protein, while severe PEM is both chronic and acute, and is composed of deficiencies in both protein and energy [2]. PEM, while generally considered a health problem in developing countries, is not rare in developed countries. According to the reports by the United Nations in 2008, around 923 million people worldwide are suffering from malnutrition [3].

Nutritional status affects the teeth pre-eruptively, although this influence is much less important than the post-eruptive local effect of diet on the teeth. Deficiencies in vitamins D and A, and protein-energy malnutrition have been associated with enamel hypoplasia and salivary gland atrophy (which reduces the mouth's ability to buffer plaque acids) which

render the teeth more susceptible to decay. In developing countries, in the absence of dietary sugars, undernutrition is not associated with dental caries. Undernutrition coupled with a high intake of sugars may exacerbate the risk of caries [4]. The effects of malnutrition on the different oral tissues have been described as follows:

### Malnutrition and development of teeth

Malnutrition is undesirable for all tissues including enamel. Once the tooth erupts into the oral cavity, this tissue has no cellular mechanism to repair whatever developmental damage has taken place and, therefore, the lesion is, to a large extent, irreversible. It is true that the enamel surface is constantly exposed to reparative effects of saliva and its components, but this can be interfered with and completely neutralized by the destructive activity of plaque bacteria [5].

A protein deficient diet fed to experimental animals during the pre-eruptive tooth development period increased their caries susceptibility [6]. In humans, there is no direct evidence of a correlation between dental caries experience and dietary protein [7].

Alvarez et al observed that one malnutrition episode occurring during the first year of life is sufficient to cause a significant delay in the eruption of all primary teeth, even

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though some of the teeth erupt two years later [8].

Although, precise causes of this interruption in enamel deposition are multiple (Pindborg, 1982; Fraser and Nikiforuk, 1982; El- Najjar *et al.* 1978), much of the hypocalcemia leading to hypoplasia formation is generally assumed to reflect metabolic stress, temporarily disturbing amelogenesis due to malnutrition and/or infection [9].

### **Malnutrition and enamel hypoplasia**

If nutritional insults are imposed early in the formation of the organic matrix, the clinical expression may be hypoplasia of enamel characterized by pitting, furrowing, or absence of enamel. If the stress is imposed later, when the maturation process is the primary activity, the result may be hypocalcification, manifested by opaque or chalky areas surrounded by normal-looking enamel [10]. Both hypoplasia and hypocalcification can be induced by other environmental stresses such as febrile episodes, chemical intoxications, and genetic defects [11].

Linear enamel hypoplasia (LEH) of the deciduous maxillary incisors is commonly observed in undernourished children throughout the world [12-15]. The lesion is very rarely observed in the posterior dentition. Clinically, the lesion is characterized by a horizontal groove most frequently found on the labial surface of the middle third of the maxillary central incisors and on the incisal third of the maxillary lateral incisors.

Clinically, the progression of anterior caries from the hypoplastic defect is usually towards the cervical area of the tooth *i.e.* the crown portion developed subsequent to the formation of hypoplasia [16].

### **Malnutrition and development of caries**

Caries may be caused either by a quality defect in the matrix of the tooth enamel or equally important by alteration in the salivary gland. Biological factors of caries included cariogenic bacteria in a complex ecological system (plaque), fermentable carbohydrates, and host factors [17].

According to the current concept of caries etiology, initiation of caries needs presence of a cariogenic plaque bacteria, a susceptible host, and fermentable sugar [18].

Teeth with accentuated pits and fissures, enamel hypoplasia or mottled enamel are more susceptible to the caries process. One of the common causes for enamel hypoplasia is malnutrition. Base-producing bacteria and remineralizing activity may modify or counter the effects of the produced acid. However, more pertinent in terms of PEM are the host factors

associated with caries, specifically tooth defects and the salivary system [19].

Indirect evidence in support of this effect can be found in the studies which found a high prevalence of linear enamel hypoplasia and dental caries in the primary teeth of rural Guatemalan children and their association with early infectious episodes [16]. In humans, a cause-effect relationship between nutritional status and dental caries has not been directly demonstrated [5]. It would be expected that chronic malnutrition (stunting) would have a greater impact than acute malnutrition (wasting) on tooth eruption, suggesting that nutritional injury occurring early in the life of a child, when the primary teeth are being formed will increase susceptibility to dental caries three to four years later [5]. A prolonged malnutrition episode in infancy, which leads to both stunting and wasting, results in a significantly higher predisposition to caries by age four years [8].

### **Effect of malnutrition on physical and chemical properties of saliva**

Saliva is one of the most important factors which balances the oral environment. Human saliva not only lubricates the oral tissues, making oral functions such as speaking, eating, and swallowing possible, but also protects teeth and oral mucosal surfaces in different ways. The lubricating and antimicrobial functions of saliva are maintained mainly by resting saliva. Stimulation of saliva results in a flushing effect and the clearance of oral debris and noxious agents [20]. It plays a crucial role in the initiation, progress as well as inhibition of dental caries and periodontal diseases, thus can be correctly referred to as the double-edge sword. The hypofunction of the salivary glands can be defined as reduction in the salivary flow, buffer capacity and salivary constituents, especially proteins, which predisposes to a higher prevalence of dental caries [21]. Protein-energy malnutrition in rats has been shown to reduce salivary flow [22], affect salivary composition [22], alter the immune system [23], and increase the acid solubility of enamel [24].

Children with severe or moderate protein energy malnutrition have significantly stimulated whole saliva secretion and buffer capacity than the children with normal weight-for-age or mild protein-energy malnutrition. The stimulated secretion rate of saliva decreases as the severity of PEM (based on height-for-age) increases [25]. Johansson *et al* [25] studying the salivary composition in children with chronic malnutrition, once again found a reduction in the flow of stimulated salivary secretion and buffer capacity. According to Johansson, children with moderate or severe malnutrition had also decreased calcium ions and protein secretion in stimulated saliva. There was

damage to the immune system of the malnourished children, with reduced agglutination protective factors in unstimulated saliva [26].

Poster JW *et al* confirmed that stimulated and unstimulated salivary flow rates were reduced at statistically significant levels in subjects who had experienced severe malnutrition in their early childhood or who had continuing nutritional stress which resulted in delayed growth, as measured at ages 11–19 years. Salivary pH demonstrated little clinically meaningful variability between malnourished and non malnourished groups [27].

Secretion of antibacterial components from some exocrine glands is reduced by malnutrition. The volume of breast milk and the total amount of sIgA secreted per minute are reduced in malnourished women [28]. It has been reported that rats exposed to moderate protein deprivation from birth and in utero [28,29] were more caries than controls. These rats developed also reported to attain lower levels of DNA, RNA, and protein in the submandibular gland [22], as well as a reduction in saliva secretion rate and protein content [30].

Saliva provides considerable protection against dental caries. The Major changes in saliva composition observed during malnutrition raise the question as to whether such variations can be associated with enhanced susceptibility to caries. It, therefore, appeared opportune to determine the effect of malnutrition on some antibacterial components of saliva, on the development of caries, and the differential effects of starch versus sucrose when malnutrition is imposed on rats [31].

### Periodontal effects of malnutrition

In health, the host uses a variety of restraints and defenses to maintain what amounts to a mutual nonaggression pact with potential periodontal pathogens. The host's defense mechanisms have specific (the immune system) and nonspecific components. The latter are either passive [e.g. anatomical barriers of the mucous membrane, and normal secretions of saliva and mucus [32,33] on active production of phagocytic cells as well as synthesis of the acute-phase reactants, lysozyme, and the cytokines [34,35]. Depleted nutritional reserves in the tissues are associated with a lowering of immunity, progressive damage to mucosa, as well as a diminished resistance to colonization and invasion by pathogens [36]. Malnutrition thus has a detrimental effect on the gingival and periodontal tissues leading to its breakdown.

### Malnutrition and oral microbial ecology

Anaerobic microorganisms most frequently isolated from malnourished children are *Prevotella melaninogenica*, *Porphyromonas gingivalis*, *Prevotella oralis*, *Prevotella ruminicola*, *Actinomyces israelii*, *Fusobacterium sp*, and the Spirochetes [37]. Increasing evidence points to the ability of the gram-negative anaerobic organisms to stimulate the host defense mechanisms that in turn may be responsible for tissue destruction in periodontitis [38,39,40].

### CONCLUSION

Nutritional status early in life appears to have a strong influence on tooth formation and emergence. Nutritional injuries early in life may affect tooth formation and result in increased susceptibility to dental caries. On the other hand, chronic malnutrition is associated with delayed tooth emergence and a shift of the curve for caries prevalence versus age. Malnutrition in children also has a drastic effect on the composition, viscosity, and pH of the saliva, which adjuncts the progression of dental caries as well as deteriorates the periodontal health. Complex interactions exist between nutritional status of the host, infections, and many other diseases that have an inflammatory component. The common types of periodontal diseases are inflammatory lesions elicited by specific pathogens in the dental plaque. In malnutrition, there is usually a variable degree of tissue depletion of key nutrients including the major antioxidant nutrients. Along with deficient immunity, colonization of specific micro-organisms favours destruction of the periodontal tissues.

Thus, proper nutrition during early development of the child has a major contribution in maintaining the general as well as oro-dental health.

### REFERENCES

1. Semba RD, Bloem MW. *Nutrition in Health in Developing Countries*. Totowa; Humana Press, 2001.
2. Shils ME, Olson JA, Moshe S. *Modern Nutrition in Health and Disease*. Philadelphia; Williams & Wilkins, Lippincott, 1999.
3. Food and Agriculture Organization Economic and Social Development Department. "The State of Food Insecurity in the World, 2008 : High food prices and food security - threats and opportunities". Food and Agriculture Organization of the United Nations, 2008;48.
4. World Health Organization recommendations for preventing dental diseases [cited 2010]. Available from: [http://www.who.int/nutrition/topics/5\\_population\\_nutrient/en/index18.html](http://www.who.int/nutrition/topics/5_population_nutrient/en/index18.html).

- eruption, and dental caries: a review. *Am J Clin Nutr* 1989; 49: 417-26.
6. Navia JM. Evaluation of nutritional and dietary factors that modify animal caries. *J Dent Res* 1970; 49: 1213-1227.
  7. Ayad M, Van Wuyckhuysse BC, Minaguchi K, et al. The association of basic proline-rich peptides from human parotid gland secretions with caries experience. *J Dent Res* 2000; 79: 976-982.
  8. Alvarez JO, Caceda J, Woolley TW, Carley KW, Baiocchi N, Caravedo L, Navia JM. A longitudinal study of dental caries in the primary teeth of children who suffered from infant malnutrition. *J Dent Res* 1993; 72(12): 1573-6.
  9. Corruccini RS, Handler JS, Jacobi KP. Chronological distribution of enamel hypoplasias and weaning in a Caribbean slave population. *Human Biol* 1985; 57(4): 699-711.
  10. Pindborg JJ. *Pathology of the dental hard tissues*. Philadelphia; WB Saunders, 1970.
  11. Molnar S, Ward SC. Mineral metabolism and microstructural defects in primate teeth. *Am J Phys Anthropol* 1974; 43: 3-9.
  12. Jelliffe DB, Jelliffe EFP. Linear enamel hypoplasia of deciduous incisor teeth in malnourished children. *Am J Clin Nutr* 1971; 24: 893.
  13. Jones MR, Larson NP, Prichard GP. Dental disease in Hawaii-I. Odontoclasia: a clinically unrecognized form of tooth decay in the pre-school child of Honolulu. *Dent Cosmos* 1930; 72: 439-450.
  14. Mayer J, Baume LJ. Pathologie de la melanodontie infantile, de l'odontoclasie et de la carie circulaire. *Rev Suisse Odontol* 1966; 76: 48-92.
  15. Sweeney EA, Saffir AJ, De Leon. R. Linear hypoplasia of deciduous incisor teeth in malnourished children. *Am J Clin Nutr* 1971; 24: 29-31.
  16. Infante PF, Gillespie GM. Enamel hypoplasia in relation to caries in Guatemalan children. *J Dent Res* 1977; 56: 493-98.
  17. Seow WK. Biological mechanisms of early childhood caries. *Community Dent Oral Epidemiol* 1998; 26: 8-27.
  18. Philip D Marsh. Dental plaque as a biofilm and a microbial community - implications for health and disease. *BMC Oral Health* 2006; 6(1): 14.
  19. Psoter WJ, Reid BC, and Katz RV. Malnutrition and dental caries: a review of the literature. *Caries Res* 2005; 39(6): 441-447.
  20. Lenander-Lumikari M., Loimaranta V. Saliva and
  21. Pereira DC, Miranda ACM, Bruno de Barros G, et al. Protein- energy malnutrition and early childhood caries. *Rev Nutr* 2010; 23(1): 119-126.
  22. Menaker L, Navia JM. Effect of undernutrition during the perinatal period on caries development in the rat. III: Effect of undernutrition on biochemical parameters in the developing mandibular salivary gland. *J Dent Res* 1973; 52: 688-91.
  23. Michalek SM, McGhee JR. Navia JM, Narkates AI. Effective immunity to dental caries: protection of malnourished rats by local infection of *S. mutans*. *Infect Immun* 1976; 13: 782-9.
  24. Aponte-Merced L, Navia JM. Pre-eruptive protein malnutrition and acid solubility of rat molar enamel surfaces. *Arch Oral Biol* 1990; 25: 701-5.
  25. Johansson I, Saellstrom AK, Rajan BP, Parameswaran A. Salivary flow and dental caries in Indian children suffering from chronic malnutrition. *Caries Res* 1992; 26(1 ): 38-43.
  26. Johansson I, Lenander-Lumikari M, Saellstrom AK. Saliva composition in Indian children with chronic protein-energy malnutrition. *J Dent Res* 1994; 73: 11-9.
  27. Psoter WJ, Spielman AL, Gebriand B, et al. Effect of childhood malnutrition on salivary flow and pH. *Arch Oral Bio* 2008; 53(3): 231-237.
  28. Navia JM, Di Orio LP, Menaker L and Miller S. (1970): Effect of undernutrition during the perinatal period on caries development in the Rat. *J Dent Res*, 49: 1091-1098.
  29. Menaker L and Navia JM. Effect of undernutrition during the perinatal period on caries development in the Rat. (II) caries susceptibility in underfed rats supplemented with protein or caloric additions during the suckling period. *J Dent Res* 1973(a); 52: 680-687.
  30. Menaker L and Navia JM. Effect of undernutrition during the perinatal period on caries development in the Rat (V) changes in whole saliva volume and protein content. *J Dent Res* 1974; 53: 592-597.
  31. Johansson I, Ericson T, Bowen W, Cole M. The effect of malnutrition on caries development and saliva composition in the Rat. *Dent Res* 1985; 64: 37.
  32. Mandel ID. The function of saliva. *J Dent Res* 1987; 66: 623-7.
  33. Sreebny L, Baum B, Edgar W, et al. Saliva: its role in health and diseases. *Int Dent J* 1992; 42: 291-304.
  34. Wan JMF, Haw MP, Blackburn GL. Nutrition, immune function, and inflammation: an overview. *Proc Nutr Soc* 1989; 48: 315-35.

- Rev* 1990; 3: 193-210.
36. Chandra RK. Nutrition and immunity: lessons from the past and new insights into the future. *Am J Clin Nutr* 1991; 53: 1087-101.
37. Sawyer DR, Nwoku AL, Rotimi VO, Hagen JC. Comparison of oral microflora between well-nourished Nigerian children. *J Dent Child* 1986; 439-43.
38. Lamster IB, Novak MJ. Host mediators in gingival crevicular fluid: implications for the pathogenesis of periodontal disease. *Crit Rev Oral Biol Med* 1992; 3: 31-60.
39. Genco Ri. Host responses in periodontal diseases: current concepts. *J Periodontol* 1992; 63: 338-55.
40. Genco Ri, VanDyke TE, Levine Mi et al. Molecular factors influencing neutrophil defects in periodontal disease. *J Dent Res* 1986; 65: 1379-91.