Diet, (Severe) Early Childhood Caries and Metabolic Diseases

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Abstract

Early childhood caries (ECC) is a symptom of a diet imbalance that can signal future diet related dental and systemic diseases. These diseases stem from diet related energy imbalances that can be strongly related to overweight, obesity, cardiovascular disease and cancer. Since the future of a child with ECC can hold such serious consequences, more attention to the long term effects of a high caloric diet should be given. A review of some of these systemic health related scenarios is presented.

Oral and Systemic Health

The relationship between oral health and systemic health is changing. Currently, there is momentum to show that periodontal disease affects general health. Periodontal disease is now considered a complex oral and systemic disease that needs to be addressed by an integrative coordinated care team that includes both dental and medical professionals. It appears that up to 50% of heart attacks are triggered by oral pathogens. *P. gingivalis,* an oral bacterium, raises risk for a heart attack by 13.6 times when present, which is twice that for a heavy smoker. Additionally, six oral spirochetes appear to be causal of Alzheimer's. [1]

These are arguments that oral periodontal pathogens cause or are related to systemic health. This article seeks to show that oral carious conditions are related to systemic health via diet. As stated by Gilbert 1999, dental caries is an obvious sign that the body's biologic systems are not functioning in an ideal manner.[2] Indeed, dental conditions maybe one of the first conditions that are noticed within the body and that if continued could lead to systemic diseases. How much more important it is to note in a young child and seek to make changes in the diet.

In the past, dental caries was shown to affect food selection and general health. Now the concept is that dental caries is a part of a spectrum of degenerative diseases that include: obesity, cardiovascular disease, diabetes and cancer.[3-5]

Early Childhood Caries (ECC)

The Vipeholm study (1954)[6] was a major study of 436 institutionalized individuals linking the effects of sugar intake, frequency of sugar intake and carbohydrate consumption to dental caries. Recently[7] 454 children with severe early childhood caries (S-EEC) and 439 caries-free children were studied using food frequency questionnaire. It was found that children with S-ECC consumed 3.2-4.8 fl oz more of sugar sweetened beverages than the caries free children. A similar finding that the amount of sugar intake was strongly correlated caries, was found in a study[8], where the weight of sugar intake appeared to be

more strongly correlated to caries than the frequency of intake; concentration of sugars in foods was positively related, and sugary snacks were more strongly related to related than total dietary sugars.

Diet seems to be have a stronger effect in caries development than the essential microbes, e.g. S. mutans positive children had higher food cariogenicity scores, higher food frequency, putative cariogenicity, and all were associated with S-ECC individually and in combination.[9] However, the differences in caries experience in children of Africa, Europe and North America cannot be explained by the prevailing mutans streptococci, but instead should be attributed to differences in the cariogenicity of the various diets.[10]

Additionally, from a world health perspective, it is noted that despite improved trends in levels of dental caries in developed countries, dental caries remains prevalent and is increasing in some developing countries undergoing nutrition transition. There is convincing evidence, collectively from human intervention studies, epidemiological studies, animal studies and experimental studies, for an association between the amount and frequency of free sugars intake and dental caries. Although other fermentable carbohydrates may not be totally blameless, epidemiological studies show that the consumption of starch staple foods and fresh fruit are associated with low levels of dental caries.[11]

Total intake of sugar-containing snacks and chips/crisps and chips intake with a sugar-containing drink to be associated with more caries.[12]

Thus our focus should change in the management of dental caries to diet. This in turn will have a positive effect in preventing other diet related diseases, e.g. overweight, obesity, type 2 diabetes, CVD and cancer. This change in focus and activity in the dental office and clinic can have profound effects in overall health.

Background

In 1978, the American Academy of Pedodontics released “Nursing Bottle Caries”, a joint statement with the American Academy of Pediatrics, to address a severe form of caries associated with bottle usage.[13] Initial policy recommendations were limited to feeding habits, concluding that nursing bottle caries could be avoided if bottle feedings were discontinued soon after the first birthday. An early policy revision added *ad libitium* breastfeeding as a causative factor. Over the next two decades, however, recognizing that this distinctive clinical presentation was not consistently associated with poor feeding practices and that caries was an infectious disease, AAPD adopted the term ECC to reflect its multifactorial etiology.[13]

Consequences of ECC include a higher risk of new carious lesions in both the primary and permanent dentitions.[11,12] hospitalizations and emergency room visits,[16,17] increased treatment costs[18], risk for delayed physical growth and development, [19,20] loss of school days and increased days with restricted activity, [21,22] diminished ability to learn,[20] and diminished oral health-related quality of life.[13,24]

This is the view of dentistry examining children with early dental caries. When viewing the same problem from a global aspect, it is possible to see that ECC is an early warning of a system disease and

not a local disease, e.g. risk for delayed physical growth and development. This paper will attempt to show that over consumption of sucrose and fructose can lead to a metabolic disease.[13]

Diseases Associated with Early Childhood Caries (ECC)

Determination of high risk children at an early age is a valuable tool to apply to the individual and/or community as a whole. It has been found that there is a relationship between early carious lesions on primary incisors and the future caries occurrence on both first permanent molars and all permanent dentition.[25] Alm et al.[26] found that the consumption of sweets at 1 year remained statistically significant with caries experience of DF > 4 and > 8. They concluded that early caries experience, consumption of sweets at an early age and mother's self-estimation of her oral health care as being less good to poor, are associated with approximal caries in adolescents. Thus, the first finding of ECC is a predictor of future caries in the permanent dentition.

Children with S-ECC appear to be at significantly greater odds of having low ferritin status compared with caries-free children and also appear to have significantly lower hemoglobin levels than the caries-free control group. Children with S-ECC also appear to be at significantly greater odds for iron-deficiency anemia than cavity-free children.[27]

Malnutrition has been consistently associated with caries in primary teeth. In 2003, 121 children aged 7-9 were randomly selected from nine underserved communities in Lima, Peru. Parents provided demographic information and a food diary for their children. Clinical examinations included assessments of height, weight, oral hygiene and dental caries. Stunting was defined using the 2000 CDC and 2007 WHO standards. In 2006, 83 children were re-examined and the 3.5 year net DMFS increment was calculated. It was concluded that stunting was a significant risk factor for caries increment in permanent teeth over a 3.5 year period, independent of other well-known risk factors for caries development.[28]

Components of ECC Diet

Frequent consumption of simple carbohydrate, primarily in the form of dietary sugars, is significantly associated with increased dental caries risk. Malnutrition (under-nutrition or over-nutrition) in children is often a consequence of inappropriate infant and childhood feeding practices and dietary behaviors associated with limited access to fresh, nutrient dense foods, substituting instead high energy, low cost, nutrient poor sugary and fatty foods.[29]

Evidence suggests that risk for early childhood caries (ECCs), the most common chronic infectious disease in childhood, is increased by specific eating behaviors. These same behaviors are also related to systemic diseases like obesity, atherosclerosis, and cardiovascular diseases in children.

A cross sectional study examined whether consumption of added sugars, sugar-sweetened beverages (SSBs) and 100% fruit juice, as well as eating frequency, are associated with severe ECCs, cross-sectional data collected from a sample of low-income, racially diverse children aged 2 to 6 years were used. It was found after control for many factors that children with the highest SSB intake were 2.0 to 4.6 times more likely to have severe ECCs compared with those with the lowest SBB intake.[30]

There is speculation that the cariogenicity of S. mutans in the presence of high-fructose corn syrup (HFCS) may differ compared to cariogenicity in laboratory studies as the change in pH was greater in HFCS than in the sucrose media biofilm.[31]

A discussion on the poor metabolic outcomes of fructose metabolism is found in later sections of this paper, but it is noteworthy that SBB are a primary cause of ECC.

Conversely, Marthaler[32] showed a very low caries prevalence in patients with hereditary fructose intolerance, a condition involving an inborn deficiency in the liver enzyme system essential for the metabolism of fructose and sucrose.

To change dietary habits is difficult and it is best to prevent the need for change. To accomplish this, mothers of children less than one year should be guided in proper nutrition for their children. This will have system effects as well as dental. Feldens et al. found that a nutritional program involving home visits during the first year of life reduced the incidence of early childhood caries.[33]

What is fructose metabolism and what diseases occur?

It has been noted previously that children with the highest sugar sweetened beverages intake were 2.0 to 4.6 times more likely to have severe ECCs compared with those with the lowest sugar sweetened beverages intake.[8] Fructose is a common component of the sweeteners and as such deserves a discussion as to its effects on the rest of the body.

Fructose is half of the sucrose molecule and as such, it is interesting to note the amount of sucrose intake of the average American, which is about 152 pounds per year. Two hundred years ago the average American ate only 2 pounds per year! Today we eat 3 pounds of sugar in one week![34,36] Also of interest is that the average grain consumption was 45% higher in 2000 than in the 1970s.[36]

Fructose metabolism and diseases

Fructose has become ubiquitous in our food supply (see Table 1), with the highest consumption being teens and young adults. Thus, understanding the consequences of fructose ingestion and its role in (dental caries and other) chronic disease development is of critical importance.[34]

Most of the fructose, fructose syrup and sugar are not consumed in their pure forms. Rather, they are added to foods and drinks, either by self, or food and drink producers. Products ranging from baby nutrition to breakfast cereals, yogurts, sausages, ready-made meals and soft drinks are loaded with fructose.[38,39]

If the liver is full of glycogen, then when fructose is ingested, the fructose is converted to liver fat.[40-43]

If this becomes a chronic condition, then fatty liver disease can result.[39,43] Fatty liver is a major risk factor for heart disease.[39] The liver metabolizes fructose to uric acid, which at low levels is a protective factor in the blood stream. However, at high levels, such as those now found, it can result in joint symptoms like gout and kidney disease.[38,45]

Additionally, uric acid in the kidneys blocks activation of vitamin D, which can result in poor calcium absorption and osteoporosis.[43,46-48]

Uric acid impairs the effectiveness of insulin,[41,49,50] which can lead to insulin-resistance, putting additional stress on the cardiovascular system.[45]

Fructose enhances protein synthesis and appears to promote a more aggressive cancer phenotype. Fructose intake is associated with increased risk of pancreatic and small intestinal cancers and possibly others. Fructose promotes flux through the pentose phosphate, which in enhances protein synthesis and may indirectly increase tumor growth. Fructose has become ubiquitous in our food supply, with the highest consumers being teens and young adults.[37]

Fructose induces transketolase which can promote pancreatic cancer growth. Fructose provides an alternative substrate to induce pancreatic cancer cell proliferation. Fructose induces thiamine-dependent transketolase flux and is preferentially metabolized via nonoxidative pentose phosphate pathways to synthesize nucleic acids and increase uric acid production. Liu et al.[51] showed that cancer cells can readily metabolize fructose to increase proliferation. This finding is of major significance for cancer patients, with dietary refined fructose consumption, and indicates that efforts to reduce refined fructose intake or inhibit fructose-mediated actions may disrupt cancer growth.[51]

Others have observed that cancer cells readily utilize fructose to support proliferation and preferentially use fructose for nucleic acid synthesis.[51-55]

Metabolic syndrome

Metabolic syndrome is a group of risk factors that raises your risk for heart diseases and other health problems such as diabetes and stroke. It is a disorder of energy utilization (diet) and storage, diagnosed by a co-occurrence of the following medical conditions: abdominal obesity, elevated blood pressure, elevated fasting plasma glucose, high serum triglycerides, and low high-density cholesterol (HDL) levels.[56]

Metabolic syndrome is found in the over-weight and obese, inactive life style and those with insulin resistance. Researchers continue to study conditions that may play a role in metabolic syndrome, such as:

*fatty liver

*polycystic ovarian syndrome

*gall stones

*breathing problems during, sleep such as sleep apnea.[56]

The main focus of treating metabolic syndrome is managing the risk factors that are within your control such as overweight or obesity, an unhealthy diet and inactive lifestyle.[56]

Childhood obesity can be harmful in a variety of ways. Obese children are more likely to have:[57]

*High blood pressure and high cholesterol, which are risk factors for cardiovascular disease. In one study, 70% of obese children had at least one cardiovascular risk factor, and 39% had two or more.[58]

*Increased risk of impaired glucose tolerance, insulin resistance and type 2 diabestes.[59]

*Breathing problems, such as sleep apnea, and asthma.[60,61]

*Joint problems and musculoskeletal discomfort.[50,62]

*Fatty liver disease, gallstones, and gastro-esophageal reflux.[59,60]

*Greater risk of social and psychological problems, such as discrimination and poor self-esteem, which can continue into adulthood.[56,60,61]

The purpose of this article is focus on the diet. Early signs of improper sugar ingestion can be found in young children with ECC. This should alter the clinician that additional health consequences will occur to the child if changes are not made to the diet.

ECC and Metabolic Diseases

It is known that children with the highest SSB intake were 2.0 to 4.6 times more likely to have severe ECCs compared with those with the lowest SBB intake.[30] It has been demonstrated that SBB can be related to over-weight, obesity, cardiovascular disease and cancer. It seems reasonable that children with ECC should be given more diet counseling and emphasizing the life time effects of this diet on system disease as well as dental health. ECC is usually found in children older than one year, this may actually be late in trying to change the diet tastes of the child. It is better, if the child has minimal encounters with sugar and all its forms, rather than trying to change a diet preference.

Summary

The relationship of diet to dental caries, including ECC, has been known for a long time. What is new is the perspective of a child with ECC may well be exhibiting the first signs of systemic diseases. It is urged to have diet consultations with mothers and care takers prior to the first year of life.

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Table 1[35]

America's sweet tooth increased 39% between 1950-59 and 2000, while the use of corn sweeteners increased 800%

	Annual Averages					
Item	**1950-59**	**1960-69**	**1970-79**	**1980-89**	**1900-00**	**2000**
Pounds per capita, dry weight						
Total caloric sweeteners	109.6	114.4	123.7	126.5	145.9	152.4
Cane and beet sugar	96.7	98.0	96.0	68.4	64.7	65.6
Corn sweeteners	11.0	14.9	26.3	56.8	79.9	85.3
High fructose corn syrup	.0	.0	5.5	37.3	56.8	63.8
Glucose	7.4	10.9	16.6	16.0	19.3	18.1
Dextrose	3.5	4.1	4.3	3.5	3.8	3.4
Other caloric sweeteners	2.0	1.5	1.4	1.3	1.3	1.5

Note: Totals may not add due to rounding

Source: USDA's Economic Research Service and Agriculture Fact Book, chapter 2