Caries Experience in Asthmatic Children: A Review of Literature

Jihan Mohammed Turkistani* / Najat Farsi ** / Abdullah Almushayt *** / Sumer Alaki ****

Aim: *This review explores the discriminating factors involved for increased caries experience in asthmatic children.* ***Background:*** *Dental caries is the single most common chronic disease of childhood. Children with chronic medical conditions are considered high caries risk. Asthma is one of the most common chronic medical conditions in childhood.* ***Material:*** *27 studies examined the asthma-caries causative relationship. Most of them were cross sectional studies; only 5 longitudinal studies were reported. In the literature, there is a lack of consensus regarding the relationship between dental caries and asthma in a child population.* ***Conclusion:*** *Despite the fact that some relatively recent studies have provided little evidence for an asthma-caries causative relationship, the majority and the most recent reports have concluded that the individualistic nature of asthmatic condition, through either its disease status or its pharmacotherapy (different combinations of medicaments), or attempts to alleviate the condition's physiologic sequelea, carries several factors for an increased caries risk.*

Keywords: *Systematic review, asthma, caries, child*
J Clin Pediatr Dent 35(1): 1–8, 2010

INTRODUCTION

This review explores the discriminating factors involved for increased caries experience in asthmatic children. Electronic search of English scientific papers from 1979 to 2009 was accomplished using Pub Med search engine. The following search terms used were: caries, dental caries, asthma, bronchial asthma, children, inhalers, and streptoccocus mutans. About 80 articles and abstracts were reviewed as well as some references of selected articles. The clinical studies that examined the asthma-caries causative relationship were about 27 studies. Most of them were cross sectional studies; only 5 longitudinal studies were reported.

Dental caries is the single most common chronic disease of childhood. Some individuals are more likely to develop caries than others. Caries risk assessment is the determination of the likelihood of the incidence of caries during a certain time period.[1] In 2002, the American Academy of Pediatric Dentistry (AAPD) has developed the caries-risk assessment tool (CAT) based on a set of physical, environmental, and general health factors. One of the components of CAT is children with chronic medical conditions requiring long-term medication being at a risk of dental caries as a side effect.[2,3] Asthma is one of the most common chronic medical conditions in childhood.

An overview about asthma

Asthma is a chronic inflammatory disorder of the airways (bronchioles), characterized by bronchial hyper-reactivity to a nonspecific stimulus. The etiology of this condition remains poorly understood, however, multiple causative factors including genetic, infectious, allergenic, socioeconomic, psychosocial and environmental factors have been reported.[4] There is considerable concern that the prevalence of asthma is increasing in Western and developing countries, and seems to be part of a generalized trend of increasing prevalence of atopic sensitization and other allergic diseases such as eczema and rhinitis.[5] In the United States, data from the National Health Interview Survey showed that almost 6.7 million children (9%) have asthma.[6] The prevalence of asthma in the world displays large variations. In the mid 1990s, the International Study of Asthma and Allergies in Childhood[7] showed that the highest rates are found in Australia, Peru, New Zealand, Singapore, and the United Kingdom, whereas the lowest are in Albania and Russia. Prevalence rates tend to be highest in economically developed

* Jihan Mohammed Turkistani, BDS, MSc, Assistant Consultant at National Guard Health Affairs, King Abdulaziz Medical City – Jeddah, Saudi Arabia.
** Najat Farsi, BDS, MSc, Professor in Pediatric Dentistry Division, Preventive Dental Sciences Department at King Abdulaziz University, Jeddah, Saudi Arabia.
*** Abdullah Almushayt, BDS, MSc, PhD, Associate Professor and Chairman, Preventive Dental Sciences Department at King Abdulaziz University, Jeddah, Saudi Arabia.
**** Sumer Alaki, BDS, MSc, PhD, Assistant Professor in Pediatric Dentistry Division, Preventive Dental Sciences Department at King Abdulaziz University, Jeddah, Saudi Arabia.

Send all correspondence to: Prof. Najat M. Farsi, Pediatric Dentistry Division, Faculty of Dentistry, King Abdulaziz University, P.O. Box 80209, Jeddah 21589, Kingdom of Saudi Arabia

Email: n_farsi@yahoo.com

countries with a temperate climate, and low in rural subsistence and economically developing communities. This study was repeated after an interval of 5–10 years in 56 countries in children aged 13–14 years and in 37 countries in children aged 6–7 years.[8] The findings indicated that international differences in asthma prevalence have reduced, particularly in the 13–14-year age group. The percentage of children reported to have had asthma increased significantly, possibly reflecting greater awareness of this condition and/or changes in diagnostic practice. Although asthma affects people of all ages, most cases of asthma occur in childhood with peak prevalence between the ages of 6 and 11 years.[9] Until puberty, asthma is more common in boys than in girls, but after puberty the incidence is equal.[10]

The typical symptoms of asthmatic episodes are wheezing, coughing, shortness of breath (dyspnea), and/or chest tightness.[11,12] The National Heart, Lung, and Blood Institute of the National Institutes of Health has released an advisory on asthma[13] that categorizes asthma as mild, moderate, or severe, based in part on the frequency and severity of daytime symptoms, exercise tolerance, and night-time symptoms. In addition, lung function tests or spirometry is an important tool for diagnosing and monitoring asthma. It measures the volume and flow of air that can be inhaled and exhaled before and after administration of short-acting bronchodilator.[14,15]

Pharmacological management of chronic childhood asthma involves two main categories of drugs: (a) anti-inflammatory agents (e.g. corticosteroids) to suppress the inflammatory response (preventer / controller medication) and (b) drugs used to reverse bronchospasm, bronchodilators, (reliever medication) e.g. β_2 agonist, anticholinergics, and thiophyllines.[16,17] Adequate management of asthma is often achieved through continuous pharmacotherapy. The complexity of asthma and asthmatic stimuli often leads to an individualized disease management plan for each patient.[11]

Asthma and caries

As the prevalence of asthma rises in the pediatric population, it is necessary to examine how this disease affects other areas of health care, most notably oral health.[18] Some asthmatic children lead a restricted lifestyle, missing so much school and not being able to participate in normal activities; these children may frequently consume sweets, leading to increase in caries levels. Also, due to increased attention given to their general asthmatic condition, parents may give little importance to oral hygiene procedures. Therefore, asthma is considered a risk factor for dental caries.[19,20] A link between dental caries and asthma (as a result of the medical condition per se or the physical and physiological effects of pharmacotherapy) has biological credibility.[21]

In spite of the increasing prevalence of asthma, only a limited number of studies have investigated caries experience in asthmatic children during the last 30 years, and the results are somewhat conflicting. Some authors have reported a correlation between childhood asthma and dental caries in children. These findings are mainly obtained from small scale studies on children and adolescents.[18,20,22-40] On the other hand, other studies have found no such connection between dental caries and asthma.[21,34,41-45] Table 1 summarizes the reported studies on the relation between asthma and dental caries in children according to chronologic sequence.

Two factors are largely responsible for this current lack of consensus. First, both asthma and caries are chronic conditions with variable presentations, complex etiologies, challenges in diagnosis, in addition to the different types of pharmacotherapy used during treatment which complicate attempts to study them. Second, lack of statistical power since most reported studies have been of cross-sectional design and have involved small, non-representative samples.[21,41] Moreover, there are some potential confounders that might affect the results of the opponent studies. These confounders are:

1. *Fluoride intake from water*. Milano[31] in 1999 reported significantly higher DMFT (decayed, missing, filled teeth) scores in asthmatic patients. Drinking-water fluoride in the study population was below the optimal level (0.7ppm). Whereas, another study showed no association between asthma and dental caries among the nationwide population based sample that have access to fluoridated water.[41] The difference in water fluoridation may be one of the factors influencing the results.[28]
2. *Use of anti-asthmatic medication*. In a study by Shulman *et al* [41]it was found that there is no association between childhood asthma and caries because not all the asthmatic children reported using anti-asthmatic inhalers or did not take their medication *per* their physicians' instructions. This suggests the possibility that many of the subjects were under-medicated compared to subjects in McDerra *et al* [30] study in 1998. All the asthmatic children used anti asthmatic inhalers, and statistical significant difference was found in caries scores between asthmatics and controls.
3. *Dental visits*. One of the reasons for not detecting differences in DMF scores in some studies is that the asthmatic group had increased access to preventive treatment during annual dental visit, such as office-applied topical fluorides, fissure sealants, diet counseling and early treatment of carious lesions.[36,41]
4. *Population characteristics*. The studies that have investigated caries experience in asthmatic children were carried out among different populations worldwide (e.g. Japan, USA, Finland, UK, Iran, India) with different geographic distribution, life style and asthma prevalence. These characteristics could have an impact on the results of those studies.

Therefore, the lack of consistency in the literature discussed above makes decision-making difficult in the clinical and public health fields. The clinically relevant issues are: (1) whether asthmatic children are at greater risk of caries than non-asthmatic children; (2) what is the evidence for

such a finding, and (3) what are the clinical and public health implications of the asthma-caries association.[41]

REVIEW RESULTS

Table 1 summarize the reported studies in the last 3 decades on the relationship between asthma and dental caries in children according to their chronological sequence. Sample size, study design, asthmatic drugs and the relation with caries are presented in the table. Five studies were longitudinal, while the rest were cross sectional. Only 8 out of 27 studies reported no significant relationship between dental caries and asthma.

Table 1. Summary of studies on the association between asthma and dental caries in children.

Authors Year	Sample size	Study design	Age (Year)	Asthma drugs	Results	p-value
Hyyppa and Paunio 1979 [44]	A = 30 C = 30	Cross sectional	10–12	Cromoglicate Corticosteroids	A:DMFS=10.0 C:DMFS=9.3	NS
Storhaug 1985 [33]	A = 47 C = 386	Cross sectional	1 - 6	75% used “saliva reducing drugs”	A: dmft=10.2 C: dmft=5.4	<0.01*
Bjerkebo-rn *et al* 1987 [43]	A: 61 C: 55	Cross sectional	5 – 18	Asthma-drugs in different combinations	A: DFS=7.8 C: DFS=6.9 A: dfs=3.8 C: dfs=4.1	NS
Ryberg *et al* 1987 [34]	A : 24 C : 24	Cross sectional	10–20	β 2-agonists	A: DFS=14.3 C: DFS=10.6	NS
Holbrook *et al* 1989 [40]	A : 49 C : 109	Cross sectional	4	Antiasthmatics	A: dmft=3.0 C: dmft=2.1	<0.05*
Ryberg *et al* 1991 [35]	A : 21 C : 21	Longitudinal	14–24	β 2-agonists A:DMFS=17.6	C:DMFS=11.9	<0.01*
Arnrup *et al* 1993 [24]	A : 25 C :244	Cross sectional	0 – 19	Not described	A: dmfs=3.5 C: dmfs=1.3	<0.01*
McDerra *et al* 1998 [30]	A : 100 C : 149	Cross sectional	4 – 10 11-16	All were using inhalers	A:DMFS=1.37 C:DMFS=0.37 A:DMFS=3.39 C:DMFS=1.79	<0.01* <0.05*
Kankaala *et al* 1998 [32]	A : 51 C : 102	Longitudinal Retrospective	> 3	Corticosteroids β 2-agonists as needed	Higher filling increments * and more extractions in asthmatic group	
Milano 1999 [31]	A : 179 C : 165	Cross sectional	2 – 12	Continous use of asthma drugs	A:dmfs=15.5 C:dmfs=11.7 A: DMFS=1.3 C: DMFS=0.6 <0.05*	<0.05*
Shulman *et al* 2001[41]	A : 1129 C : 5809	Cross sectional	4 – 16	Not all children used antiasthmatic inhalers	A:dfs=4.9 C: dfs=4.5 A:DMFS=0.24 C:DMFS=0.62	NS 0.01*
Meldrum *et al* 2001[21]	A : 39 C : 206	Longitudinal	15-18	antiasthmatic drugs	A: DFS=2.33 C: DFS=2.13	NS
Reddy *et al* 2003[20]	A:205	Cross sectional	3-18	Inhaler, syrup, tablet, combination	High caries prevalence, increased with severity of asthma and in those taking syrup medication	
Wogelius *et al* 2004 [36]	4920 A = 6% C=81.2%	Longitudinal	5 - 7	Corticosteroid β2-agonists	Increased caries risk in * permanent teeth (RR=1.45 95%CI: 0.99-2.11)	
Eloot *et al* 2004 [45]	A : 140	Cross sectional	3-17	Antiasthmatic drugs	dmft=1.99 dmfs=5.15 DMFT=1.1 DMFS=2.07	NS
Ghasempour *et al* 2005 [38]	A:75 C:75	Cross sectional	4-18	Inhaler, syrup, tablet, combination	A:DMFT=2.27 C:DMFT=0.8	< 0.05*
Bimstein *et al* 2006 [26]	A:50 C:127	Cross sectional	4	Not described	A:dmfs=13.2 C:dmfs=8.1	0.04*

Table 1. Summary of studies on the association between asthma and dental caries in children. *(continued)*

Authors Year	Sample size	Study design	Age (Year)	Asthma drugs	Results	p-value
Milano *et al* 2006[18]	A:165	Cross sectional	3-12	Albuetrol alone or combined with others	Children used medication > twice daily were significantly more likely to experience dental caries	
Wierchola *et al* 2006 [25]	A: 326 C: 326	Cross sectional	3-15	Corticosteroid β2-agonists	A:dmft=2.06 C:dmft=0.80 A:DMFT=6.76 C:DMFT=5.06	< 0.05*
Ersin *et al* 2006 [29]	A: 106 C: 100	Cross sectional	6-19	Corticosteroid β2-agonists leukotriene antagonists	A:dft=9.2 C:dft=5.7 A:DMFS=3.3 C:DMFS=1.5	< 0.05*
Khalilzadeh *et al* 2007 [28]	A: 45 C: 46	Cross sectional	5 - 15	anti-asthma inhalers	A:DMFT=4.30 C:DMFT=3.98	< 0.05*
Shashikiran *et al* 2007 [22]	A: 35 C: 106	Case control (follow-up)	6 - 14	β2-agonists (salbutamol inhaler)	A:dfs=1.23 C:dfs=0.14 A:DMFS=2.0 C:DMFS=0.43	<0.001*
Amaechi *et al* 2008 [37]	A: 117 C: 117	Cross sectional	2-17	Antiasthmatic medication	A:DMFS=13.3 C:DMFS=6.8 A:dfs=18.0 C:dfs=8.6	<0.001*
Mazzoleni *et al.* 2008[27]	A: 30 C: 30	Cross sectional	6-12	Corticosteroid β2-agonists	A:DMFT=1.2 C:DMFT=0.3	< 0.05*
Tanaka *et al.* 2008[42]	A: 21,792	Cross sectional	6-15	Not described		NS
Stensson *et al.* 2008 [23]	A : 127 C : 117	Cross sectional	3-6	Corticosteroid β2-agonists	3Y A:dfs=1.4 C:dfs=0.5 6Y A:dfs=2.5 C:dfs=1.8	<0.05* NS
Mehta *et a*l. 2009 [39]	A:80 C:80	Cross sectional	11-25	Corticosteroid β2-agonists	A:dfs=6.4 C:dfs=2.1	< 0.01*

A: asthmatic subjects
C: control subjects

* Significant association between asthma and dental caries
NS no significant association between asthma and dental caries

DISCUSSION

Since asthmatic children are a large and growing portion of pediatric dental patients, factors affecting their oral health are salient to the dental profession. There are several interrelated factors discriminating for caries in asthmatic children that will be discussed in the following section.

Severity and length of the disease

The relationship between asthma and caries experience in children is difficult to examine due to variation in its severity, and its pharmacotherapy often fluctuate over time according to the seasons.[23] Eloot *et al*[45] found that neither the time-span of the disease period, in 3-17-year-old children, nor the medication or the severity of asthma had a significant effect on the risk of developing caries or gingivitis. In line with this study, Stensson *et al* [23] and Ersin *et al* [29] were unable to demonstrate any correlation between the severities of asthma, the period of exposure to medications, and caries prevalence. Whereas, in a study by Milano *et al* [18] it was found that prolonged use of medication, in children with mixed dentition, was associated with decreased likelihood of caries experience. This may reflect better disease management.

Reddy *et al*[20] however, found a significant difference in both the primary ($P<0.05$) and mixed dentitions ($P<0.009$) of asthmatic children treated with various anti-asthma medications. The study also illustrated the statistically significant association between increasing prevalence of caries with increasing severity of asthma, most likely due to the increased dosage and frequency of medication required to treat more severe asthma.

Medication

Patients with bronchial asthma are affected by both the disease and the medication. It is difficult to dissociate the effects of the two, but there are indications that medication exerts a stronger effect.[30] The effect of medication can be discussed from several aspects:

Xerostomic effect. There are three medications commonly used by asthmatics with known xerostomic effects: anti-asthmatic inhalers, anti-histamines, and corticosteroids. The increase in caries risk associated with long-term use of these medications is due to reduced salivary gland function and reduced salivary flow.[41] When asthma is treated with β-adrenoceptor agonist, it promotes bronchial relaxation by stimulating the β-adrenergic receptors in the lungs. However, β-adrenergic receptors are also present in the secretory system of the salivary glands. A dose-response effect of the treatment with β_2 adrenoceptor agonists is the impairment of salivary secretion (xerostomia) and it also has an effect on its composition in asthmatic patients.[46-48] Several studies [34,35] showed that asthmatic children who were medicated with β_2 agonists had a decreased saliva secretion rate and increased levels of *lactobacilli* and *mutans streptococci*, in addition to more caries lesions, compared with healthy children.

Medication frequency and time of day. Children, who are medicated more frequently, therefore exposing their teeth to a higher dosage of sugar-containing drugs over time, were more likely to experience dental caries.[18,33] Medication taken in the evening, before bed, and without mouth rinsing could pose a greater risk for caries since salivation decreases at night, thereby diminishing the protection against the medication. This was reported by Reddy *et al* [20] who referred it to lack of instruction to the patient and parent and little to no oral hygiene was performed after the medication.

Medication type. Khalilzadeh *et al* [28] studied the effect of medication type on DMFT score. Children who received β_2-agonists alone had a higher prevalence of dental caries as compared to those using corticosteroids and β_2-agonists. This difference might be due to the fact that asthma is better managed in those receiving inhaled steroids. Therefore, the child's need for inhaled β_2-agonists (which are stronger xerostomic drugs compared to corticosteroids) will be decreased specially in the acute phase of disease.

Mode of delivery. The most common mode of administering the asthma medications is by inhalation using a metered dose inhaler (MDI). Inhaled medications are delivered in a carrier powder which may contain a sugar to assist the users in recognizing the delivery of their medication by taste. Corticosteroids inhalers as Becodisk (beclomethasone dipropionate) and Flixotide Rotadisk (fluticasone propionate) contain as much as 25 mg of large-particle lactose per dose.[49] While lactose is less cariogenic than other common sugars, it has caries-promoting potential when frequent oral inhalation is combined with reduced salivary flow rate.[33,50,51] Therefore, spacers are used to reduce the oral bioavailability of the medication and consequently less caries risk. It has been reported that children using the inhalers by spacers have lower DMFT scores.[28] Long term use of liquid oral medications containing sugars can also lead to an increased caries rate.[52-54]

pH of inhaled medication. Powdered versions of anti-asthmatic medications (such as beclomethasone dipropionate, fluticasone, salmeterol, and terbutaline sulphate powders) have a pH lower than 5.5 and are more acidic than aerosol versions.[49,55] Of the dose inhaled, 80% precipitates in the mouth. This low pH value of the inhaled medication, associated with a reduced salivary flow rate, may make asthmatics more susceptible to erosion[19,55] and demineralization.[17] Consumption of erosive drinks on a regular basis, will further complicates the problem of erosion.[56] McDerra *et al* [30] found that asthmatic children, who consumed erosive drinks regularly, have more tooth surface loss affecting predominantly the labial surfaces of anterior teeth and occlusal surfaces of posterior teeth.

Systemic effects of corticosteroids. As it is known that corticosteroids influence the calcium metabolism of bones, a more apparent explanation could be that long-term use of asthma-medications during the time of tooth formation has influenced the mineralization of the permanent teeth.[57] Further, frequent use of inhaled corticosteroids in recommended doses in asthma treatment causes initial growth retardation, but no decrease in adult height.[58] This could lead to the speculation that inhaled corticosteroids can cause disturbances in tooth development, e.g. hypomineralizations, which in contrast to the effect on bone demineralization are irreversible disturbances. Therefore, in a study by Wogelius *et al* [36] when comparing children with asthma-medication use with children without asthma-medication use, they found no increased risk of dental caries in the primary teeth, while in the newly erupted permanent teeth, caries risk increased.

Microbiological factors

Some studies have proved a positive relationship between duration of asthma and the salivary levels of *Streptococcus mutans*. Inhaled β_2-agonists can provide an optimal environment for the growth and proliferation of microorganisms responsible for dental caries (*Streptococcus mutans* and *Lactobacilli*) through decreasing salivary secretion and pH.[36,41] The results from Mazzoleni *et al* [27] study supported the hypothesis that asthma may increase the risk of caries among children undergoing treatment with short-acting $ß_2$-agonists. They registered a higher DMFT score, lower buffer capacity and higher cariogenic bacteria counts in the saliva of the asthmatic children, in spite of the results of better oral hygiene and lower plaque index registered among the asthmatic group compared with the controls. Similarly, Khalilzadeh *et al*[28] demonstrated that the number of *Streptococcus mutans* colonies was significantly higher in the asthmatic group compared to control group (p=0.0001). Another study[59] evaluated the levels of *Streptococcus mutans* and *Lactobacilli* in the saliva of asthmatic children, from 3 to 15 years old. No differences were observed in caries prevalence between asthma and control group in 3–6 and in 7–10 years old children. However, in 11–15 years old children, higher caries prevalence was observed in asthma and control

groups. There was an increase on salivary levels of *Streptococcus mutans* in asthma group, however, no differences were observed for Lactobacilli. The higher caries prevalence in 11-15 years old patients, as well as the higher salivary levels of *Streptococcus mutans* in asthmatic children, suggests that these children need more dental preventive attention.

However, Stensson *et al* [23] found no connection between caries prevalence and microbiological factors in 6-year-old asthmatic children. No *Streptococcus mutans* or *lactobacilli* were detected in about 60% of the children in both asthma and control groups.

Salivary factors

Saliva is one of the defensive mechanisms against dental caries. The presence of immunoglobulins and other glycoproteins in the saliva and also its viscosity play an important role in decay prevention.[60] Any changes in saliva amount or quality may alter the oral health status.[61]

Salivary flow rate. The effect of reduced salivary flow has been elucidated in several studies.[24,34,35,61,62] Ryberg *et al*[34,35,63] reported a link between increased caries incidence and regular use of inhaled ß$_2$-agonists. These studies suggested that asthmatics may have altered salivary composition and flow rates due to the use of ß$_2$-agonists as has been explained earlier. The secretion rates of whole and parotid saliva is decreased by 26% and 36% respectively in asthmatic patients when compared with healthy control group. The values for pH and buffer capacity did not differ between the groups. The concentrations of total protein and amylase in stimulated parotid saliva were significantly lower in the asthmatic group. Thus, these data further indicate that the changes in saliva secretion and synthesis of salivary proteins are caused by the medications treatment.

As reduced salivary flow is accompanied by concomitant increase in Lactobacilli and *Streptococcus mutans* in the oral cavity, it may be one of the major contributing factors for increase in caries rate.

Salivary pH. In a study by Kargul *et al* [64] that utilized the touch electrode technique to investigate the effect of two MDIs with spacer device, on salivary pH and plaque pH in 30 asthmatic children between the ages of 6 and 14 years. A significant decrease in both plaque and saliva pH was observed over 30 min following inhaler use. This significant decrease in pH was below the critical value of 5.5 for enamel demineralization. Another study of Tootla *et al* [17] evaluated the acidogenic potential of asthma inhalers, metered dose inhaler and dry powder inhaler formulations. Although none of the inhalers were able to demonstrate an acidogenic response below the critical pH, the substantial pH drops observed with the lactose-based dry powder inhalers may be an important consideration for enamel demineralization.

On the other hand, Ersin *et al*[29] demonstrated that the salivary flow rate and pH value were statistically lower in the asthmatic group, however, there was a significant negative correlation between the salivary flow rate and caries development (P= 0.023). Although the salivary pH was statistically lower in asthmatics than non-asthmatics, it was not below the critical pH.

Buffering capacity. The results of saliva analysis[17,27] revealed lower buffer capacity and an increased bacteria load in the saliva of the asthmatic children compared with the control group, thereby supporting the hypothesis that asthma may increase the risk of caries. The alteration in the saliva buffer capacity and increase of Streptococcus mutans and Lactobacilli counts in the saliva of the asthmatic children undergoing treatment with short-acting ß$_2$-agonists could be attributed to the diminished salivary production and secretion associated with the prolonged use of ß$_2$-agonists. In contrast, Stensson *et al* [23] found that the distribution of buffer capacity classes according to Dentobuff was about the same in both asthma and control groups.

Salivary composition. Some studies[61,65,66] indicated that, in asthmatic patients, salivary low buffering capacity, low calcium and phosphate, and low specific sIgA have less pronounced link to increased caries. In addition, the concentrations of total protein and amylase in stimulated whole saliva were found lower in the asthmatic children, but it was not statistically significant.[29]

Physiologic sequalea of asthmatic condition

Some authors reported that another reason for the increased caries rate in asthmatics may be an increased frequency of consumption of cariogenic drinks. Asthmatics may use erosive cariogenic drinks to alleviate the desiccating effects of mouth breathing, alleviate the increased thirst related to the reduction in saliva flow caused by β_2 agonists, or eliminate the residual taste of medication that remains in oropharynx. Parents of asthmatic children often commented that their asthmatic children were thirstier than their non asthmatic siblings; this was merely an anecdotal observation.[30,67] Stensson *et al*[23] found that there was a general trend for asthmatic children to have a somewhat higher prevalence of mouth breathing than children without asthma, although it was not significant for 6-year-old children. In 3-year-old children with asthma and frequent mouth breathing, the mean dfs was 3.1 ± 5.0 compared with 0.8 ± 1.9 in asthmatic children without mouth breathing (P< 0.05). Also, they experienced significantly more bleeding on probing (gingivitis).

CONCLUSIONS

Despite the fact that some relatively recent studies have provided little evidence for an asthma-caries causative relationship, the majority and the most recent reports have concluded that the individualistic nature of asthmatic condition, through either its disease status or its pharmacotherapy (different combinations of medicaments), or attempts to alleviate the condition's physiologic sequalea, carries several factors for an increased caries risk.

CLINICAL SIGNIFICANCE

It is essential to develop individualized intensive preventive dental programs for asthmatic children according

to their age, since they are at higher risk for dental caries. This program may include: (1) brushing after medication use with fluoridated tooth paste; (2) daily fluoride rinses; (3) dietary advice; (4) drinking water instead of erosive drinks when thirsty (5) more frequent recall visits and regular topical fluoride treatments.

The parents of children with asthma should be educated and informed about the nature of this medical condition and how it increases their susceptibility to dental caries. It is important to develop collaboration between dental and medical care-givers in relation to children with asthma.

REFERENCES

1. Reich E, Lussi A, Newbrun E. Caries-risk assessment. Int Dent J, 49: 15–26, 1999.
2. American Academy of Pediatric Dentistry. Policy on use of a caries-risk assessment tool (CAT) for infants, children, and adolescents. Pediatr Dent, 30: 29–33, 2008.
3. Hobson P. Sugar based medicines and dental disease. Community Dent Health, 2: 57–62, 1985.
4. Weiss KB, Gergen PJ, Wagener DK. Breathing better or wheezing worse? The changing epidemiology of asthma morbidity and mortality. Annu Rev Public Health, 14: 491–513, 1993.
5. Tattersfield AE, Knox AJ, Britton JR, Hall IP. Asthma. Lancet, 360: 1313–1322, 2002.
6. Bloom B, Cohen RA, Freeman G. Summary health statistics for U.S. children: National Health Interview Survey, 2007. Vital Health Stat, 10: 1–80, 2009.
7. The International Study of Asthma and Allergies in Childhood (ISAAC) Steering Committee. Worldwide variations in the prevalence of asthma symptoms: the International Study of Asthma and Allergies in Childhood (ISAAC). Eur Respir J, 12: 315–335, 1998.
8. Pearce N, Ait-Khaled N, Beasley R, et al. Worldwide trends in the prevalence of asthma symptoms: phase III of the International Study of Asthma and Allergies in Childhood (ISAAC). Thorax, 62: 758–766, 2007.
9. Taylor WR, Newacheck PW. Impact of childhood asthma on health. Pediatrics, 90: 657–662, 1992.
10. Nicolai T, Pereszlenyiova-Bliznakova L, Illi S, Reinhardt D, von Mutius E. Longitudinal follow-up of the changing gender ratio in asthma from childhood to adulthood: role of delayed manifestation in girls. Pediatr Allergy Immunol, 14: 280–283, 2003.
11. Steinbacher DM, Glick M. The dental patient with asthma. An update and oral health considerations. J Am Dent Assoc, 132: 1229–1239, 2001.
12. Casey KR, Winterbauer RH. Acute severe asthma. How to recognize and respond to a life-threatening attack. Postgrad Med, 97: 71–78, 1995.
13. Guidelines for the diagnosis and management of asthma. National Heart, Lung, and Blood Institute. National Asthma Education Program. Expert Panel Report. J Allergy Clin Immunol, 88: 425–534, 1991.
14. Urbano FL. Review of the NAEPP 2007 Expert Panel Report (EPR-3) on Asthma Diagnosis and Treatment Guidelines. J Manag Care Pharm, 14: 41–49, 2008.
15. Pulmonary disease In: Little J, Palace D, Miller C, Rhodus N eds, Dental Management of the Medically Compromised Patient. 6th ed. St. Louis, Missouri: Mosby; 125–146, 2002.
16. Zhu JF, Hidalgo HA, Holmgreen WC, et al. Dental management of children with asthma. Pediatr Dent, 18: 363–370, 1996.
17. Tootla R, Toumba KJ, Duggal MS. An evaluation of the acidogenic potential of asthma inhalers. Arch Oral Biol, 49: 275–283, 2004.
18. Milano M, Lee JY, Donovan K, Chen JW. A cross-sectional study of medication-related factors and caries experience in asthmatic children. Pediatr Dent, 28: 415–419, 2006.
19. Al-Dlaigan YH, Shaw L, Smith AJ. Is there a relationship between asthma and dental erosion? A case control study. Int J Paediatr Dent, 12: 189–200, 2002.
20. Reddy DK, Hegde AM, Munshi AK. Dental caries status of children with bronchial asthma. J Clin Pediatr Dent, 27: 293–295, 2003.
21. Meldrum AM, Thomson WM, Drummond BK, Sears MR. Is asthma a risk factor for dental caries? Finding from a cohort study. Caries Res, 35: 235–239, 2001.
22. Shashikiran ND, Reddy VV, Raju PK. Effect of antiasthmatic medication on dental disease: dental caries and periodontal disease. J Indian Soc Pedod Prev Dent, 25: 65–68, 2007.
23. Stensson M, Wendt LK, Koch G, Oldaeus G, Birkhed D. Oral health in preschool children with asthma. Int J Paediatr Dent, 18: 243–250, 2008.
24. Arnrup K, Lundin SA, Dahllof G. Analysis of paediatric dental services provided at a regional hospital in Sweden. Dental treatment need in medically compromised children referred for dental consultation. Swed Dent J, 17: 255–259, 1993.
25. Wierchola B, Emerich K, Adamowicz-Klepalska B. The association between bronchial asthma and dental caries in children of the developmental age. Eur J Paediatr Dent, 7: 142–145, 2006.
26. Bimstein E, Wilson J, Guelmann M, Primosch RE. The relationship between oral and demographic characteristics of children with asthma. J Clin Pediatr Dent, 31: 86–89, 2006.
27. Mazzoleni S, Stellini E, Cavaleri E, et al. Dental caries in children with asthma undergoing treatment with short-acting beta2-agonists. Eur J Paediatr Dent, 9: 132–138, 2008.
28. Khalilzadeh S, Salamzadeh J, Salem F, Salem K, Vala M. Dental Caries-Associated Microorganisms in Asthmatic Children. Tanaffos, 6: 42–46, 2007.
29. Ersin NK, Gulen F, Eronat N, et al. Oral and dental manifestations of young asthmatics related to medication, severity and duration of condition. Pediatr Int, 48: 549–554, 2006.
30. McDerra EJ, Pollard MA, Curzon ME. The dental status of asthmatic British school children. Pediatr Dent, 20: 281–287, 1998.
31. Milano M. Increased risk for dental caries in asthmatic children. Tex Dent J, 116: 35–42, 1999.
32. Kankaala TM, Virtanen JI, Larmas MA. Timing of first fillings in the primary dentition and permanent first molars of asthmatic children. Acta Odontol Scand, 56: 20–24, 1998.
33. Storhaug K. Caries experience in disabled pre-school children. Acta Odontol Scand, 43: 241–248, 1985.
34. Ryberg M, Moller C, Ericson T. Effect of beta 2-adrenoceptor agonists on saliva proteins and dental caries in asthmatic children. J Dent Res, 66: 1404–1406, 1987.
35. Ryberg M, Moller C, Ericson T. Saliva composition and caries development in asthmatic patients treated with beta 2-adrenoceptor agonists: a 4-year follow-up study. Scand J Dent Res, 99: 212–218, 1991.
36. Wogelius P, Poulsen S, Sorensen HT. Use of asthma-drugs and risk of dental caries among 5 to 7 year old Danish children: a cohort study. Community Dent Health, 21: 207–211, 2004.
37. Amaechi B, Lozano-Pineda J. Prevalence of caries among siblings with and without asthma. J Dent Res, AADR 37th Annual Meeting 2008; 87.
38. Ghasempour M, Mohammadzade I, Hosaininia K. Dental health status in asthmatic children. Journal of Isfahan Dental School, 1: 41–46, 2005.
39. Mehta A, Sequeira PS, Sahoo RC. Bronchial asthma and dental caries risk: results from a case control study. J Contemp Dent Pract, 10: 59–66, 2009.
40. Holbrook WP, Kristinsson MJ, Gunnarsdottir S, Briem B. Caries prevalence, Streptococcus mutans and sugar intake among 4-year-old urban children in Iceland. Community Dent Oral Epidemiol, 17: 292–295, 1989.
41. Shulman JD, Taylor SE, Nunn ME. The association between asthma and dental caries in children and adolescents: A population-based case-control study. Caries Res, 35: 240–246, 2001.
42. Tanaka K, Miyake Y, Arakawa M, Sasaki S, Ohya Y. Dental caries and allergic disorders in Japanese children: the Ryukyus Child Health Study. J Asthma, 45: 795–799, 2008.
43. Bjerkeborn K, Dahllof G, Hedlin G, Lindell M, Modeer T. Effect of disease severity and pharmacotherapy of asthma on oral health in asthmatic children. Scand J Dent Res, 95: 159–164, 1987.

44. Hyyppa T, Paunio K. Oral health and salivary factors in children with asthma. Proc Finn Dent Soc, 75: 7–10, 1979.
45. Eloot AK, Vanobbergen JN, De Baets F, Martens LC. Oral health and habits in children with asthma related to severity and duration of condition. Eur J Paediatr Dent, 5: 210–215, 2004.
46. Baum BJ. Neurotransmitter control of secretion. J Dent Res, 66 Spec No: 628–632, 1987.
47. Venter JC, Fraser CM, Harrison LC. Autoantibodies to beta 2-adrenergic receptors: a possible cause of adrenergic hyporesponsiveness in allergic rhinitis and asthma. Science, 207: 1361–1363, 1980.
48. Fraser CM, Venter JC, Kaliner M. Autonomic abnormalities and autoantibodies to beta-adrenergic receptors. N Engl J Med, 305: 1165–1170, 1981.
49. O'Sullivan EA, Curzon MEJ. Drug treatments for asthma may cause erosive tooth damage. BMJ, 317: 820, 1998.
50. Pearce EI, Sissons CH. On the cariogenicity of lactose. N Z Dent J, 83: 32–36, 1987.
51. Makinen KK, Philosophy L. The role of sucrose and other sugars in the development of dental caries; a review. Int Dent J, 22: 363–386, 1972.
52. Maguire A, Rugg-Gunn AJ, Butler TJ. Dental health of children taking antimicrobial and non-antimicrobial liquid oral medication long-term. Caries Res, 30: 16–21, 1996.
53. Siu AS, Chu FC, Yip HK. Cough syrup addiction and rampant caries: a report of two cases. Prim Dent Care, 9: 27–30, 2002.
54. Tredwin CJ, Scully C, Bagan-Sebastian JV. Drug-induced disorders of teeth. J Dent Res, 84: 596–602, 2005.
55. Shaw L, al-Dlaigan YH, Smith A. Childhood asthma and dental erosion. ASDC J Dent Child, 67: 102–106, 182, 2000.
56. Shaw L, Smith A. Erosion in children: an increasing clinical problem? Dent Update, 21: 103–106, 1994.
57. Williams DM. Clinical considerations in the use of inhaled corticosteroids for asthma. Pharmacotherapy, 21: 38S–48S, 2001.
58. Agertoft L, Pedersen S. Effect of long-term treatment with inhaled budesonide on adult height in children with asthma. N Engl J Med, 343: 1064–1069, 2000.
59. Ferreira F, Botelho M, Fernandes K, et al. Levels of cariogenic microorganisms and oral conditions of asthmatic children. J Dent Res IADR/CADR 86th General Session 2008; 87
60. Hegde PP, Ashok Kumar BR, Ankola VA. Dental caries experience and salivary levels of Streptococcus mutans and Lactobacilli in 13-15 years old children of Belgaum city, Karnataka. J Indian Soc Pedod Prev Dent, 23: 23–26, 2005.
61. Lenander-Lumikari M, Laurikainen K, Kuusisto P, Vilja P. Stimulated salivary flow rate and composition in asthmatic and non-asthmatic adults. Arch Oral Biol, 43: 151–156, 1998.
62. Laurikainen K, Kuusisto P. Comparison of the oral health status and salivary flow rate of asthmatic patients with those of nonasthmatic adults—results of a pilot study. Allergy, 53: 316–319, 1998.
63. Ryberg M, Moller C, Ericson T. Saliva composition in asthmatic patients after treatment with two dose levels of a beta 2-adrenoceptor agonist. Arch Oral Biol, 35: 945–948, 1990.
64. Kargul B, Tanboga I, Ergeneli S, Karakoc F, Dagli E. Inhaler medicament effects on saliva and plaque pH in asthmatic children. J Clin Pediatr Dent, 22: 137–140, 1998.
65. Leone CW, Oppenheim FG. Physical and chemical aspects of saliva as indicators of risk for dental caries in humans. J Dent Educ, 65: 1054–1062, 2001.
66. Sag C, Ozden FO, Acikgoz G, Anlar FY. The effects of combination treatment with a long-acting beta2-agonist and a corticosteroid on salivary flow rate, secretory immunoglobulin A, and oral health in children and adolescents with moderate asthma: a 1-month, single-blind clinical study. Clin Ther, 29: 2236–2242, 2007.
67. Pedersen S. Inhalers and nebulizers: which to choose and why. Respir Med, 90: 69–77, 1996.