Exposure To Second-hand Smoking And Dental Caries In Children

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Introduction

Exposure to second-hand smoke (SHS) in household and maternal smoking especially during antenatal period has long been implicated in a number of unwanted health outcomes in children. Low birth weight as well as poor compliance with breastfeeding has been reported to be one of the commonest adverse outcomes owing to maternal smoking. [1,2]. Further adverse outcomes associated with SHS are increased rates of asthma, otitis media and hence more missed day from school, increased rates of hospitalization, and malnutrition [3-6]. Another resent adverse outcome of interest simulating further research and debates is association of dental caries and SHS [7]. In this paper, we attempt to discuss the effect SHS on dental caries in children and possible mechanism by which SHS is implicated in causation of dental caries.

Literature in support of dental caries and exposure to second-hand smoking

The literature studying the association between smoking and dental caries suggest that household smoking increases risk of early childhood caries.while not many studies suggest that maternal smoking during pregnancy is a clear risk for dental caries.

Author, Year	Study design	Sample	Conclusion
Bolin, 1997 [8]	Cross-sectional data of school children in 8 European countries	N = 3200 Age = 5-12 years	Mother's smoking habits increased caries risk.
Williams, 2000[9]	Cross-sectional data from the UK National Diet and Nutrition Survey, (1995)	N = 749 Age = 3 to 4.5 years	Mother's smoking habits significantly increased caries risk.
Aligne, 2003[10]	Cross-sectional data from the National Health and Nutrition Examination Survey III (1988-1994) in the US	N = 3541 Age = 4 to 11 years	There is an association between environmental tobacco smoke and risk of caries among children. Reduction of passive smoking is important not only for the prevention of many medical problems, but also for the promotion of children's dental health.
Shenkin, 2004 [11]	Cross-sectional data from IFS in8 hospitals in the US	N = 637 Age = 4-7 year	Environmental tobacco smoke was associated with an increased risk of caries among children
Tanaka, 2006 [12]	Cross-sectional data from the NNSt, NSDD m (1999) in Japan	N = 925 Age = 1-14 years	smoking in the household was independently associated with an increased prevalence of decayed teeth
Ayo-Yusuf, 2007 [13]	Cross-sectional data in 21 high schools in South Africa	N = 1873	smoking may be a risk indicator for caries in adolescents' permanent teeth.
Hanioka, 2008 [14]	Cross-sectional data from records of health checkup at a public health facility in Japan	N = 711 Age = 36 months	results indicate the association of ECC with parental smoking, although the association with paternal smoking was weaker than with maternal smoking.
Leroy, 2008 [15]	Cross-sectional data from the Smile for Life project in 4 distinct geographical areas in Flanders, Belgium[23].	N = 1250 Age 3 yrs N= 1283 Age = 5 yrs	results of this study illustrate the existence of a significant association between parental smoking behavior and caries experience in 5-year-old children.
Tanaka, 2009 [16]	Cross-sectional data from the FCHSs in 7 public health centers in Japan	N = 2015 Age = 3 yrs	Both in utero exposure to maternal smoking and postnatal exposure to ETS may be associated with an increased prevalence of dental caries in young children.
Julihn, 2009 [17] associated factors	Cohort data from the SMBRo and SNR is available at Statistics Sweden	N = 18,142 Age = 13 yrs	Study demonstrates that the prenatal factors, maternal overweight, as well as smoking, are risk factors for approximal caries development in offspring during the teenage period.
Tanaka, 2010 [18]	Cross-sectional data from school records in Okinawa, Japan	N = 20,703 Age = 6 to 15 yrs	Findings suggested that household smoking might be associated with an increased prevalence of dental caries in children
Dilmyer, 2010 [19] associated factors	Cross-sectional data in schools in Nevada	N = 4000 Age = 12 to 19 yrs	Second hand smoke, insurance status, and tobacco use were significant, but to a lesser extent.

Possible Biologic Pathway

The most common risk marker for both SHS and dental caries is socioeconomic disparity. Rather, SHS has long been considered to be a marker of low (socioeconomic statues) SES [20] . In fact, low SES has been considered to be one of the strongest predictor for dental caries [21]. as well as enamel hypoplasia. Later has also been identified as a risk factor for development of dental caries [22]. Further, poorer SES has been found to be associated with unhealthy dietary choices and lifestyles. Unhealthy lifestyles of smoking parents might also be associated with increased sugar consumption which is again a cause factor for dental caries. [23, 24].

Recent papers have postulated several pathways for implicating SHS as a risk marker for dental caries. We further attempt to enlighten the readers with the underlying mechanisms which could explain modulation of existing etiology of dental caries by SHS.

Ameloblasts are extremely sensitive to environmental influences which may induce qualitative or quantitative defects in enamel formation. SHS during period of tooth formation may modify immediate environment of ameloblasts. This can cause defective mineralization and hence increase risk of development of dental caries. [25-28]. Environmental cadmium exposure may be independently associated with increased risk of early childhood caries [29].

In addition to inducing qualitative defects in enamel, immunologic pathway has also been implicated in dental caries. Suppression or modulation of body's immune function owing to SHS may result in increase colonization of cariogenic bacteria on rough tooth surfaces [30]. This has a direct bearing on increased dental caries.

Another factor that merits discussion here is decreased blood levels of vitamin C in smokers and children residing in environment with increased exposure to SHS. [31-33]. Decreased vitamin C levels have been reported to alter the oral ecosystem to favour growth of *S. mutans* [34].

One more postulate of interest in this regard is affect of SHS on salivary glands. Saliva has a protective role in dental caries by virtue of its buffering capacity. Active as well as passive smokers including children have been reported to have lower buffering capacity and thus increased salivary levels of lactobacilli and S. mutans. [35, 36, 37]. In addition to qualitative changes in saliva, quantitative alteration of salivary function has also been reported. Reduced salivary flow in smokers and children exposed to SHS may also enhance the colonization of cariogenic bacteria on rough tooth surfaces and interrupt clearance of fermentable carbohydrate from the mouth [38]. Another important mechanism might be decreased remineralization of white spot lesions because of altered salivary function.

Further, children whose mother smoke might have increased toxins in oral ecosystem delivered through breast milk [39]. Other reason for increased toxins in oral ecosystem might be high incidence nasal congestion in children. Nasal discomfort forces children to breathe through mouth and thus resulting in increased exposure to SHS [40]. Early colonization of *S. mutans* [41,42] have been reported in children residing with smoking parents.

The tentative effects of SHS regarding tooth formation and salivary gland function would be altered during pregnancy as well as after delivery. However, the association between maternal smoking and increased development of dental caries has not been supported by recent literature.

Conclusion

The available high quality evidence suggests that there is moderate to weak evidence between SHS and early childhood caries. However, the evidence to suggest an association of maternal smoking during pregnancy is scarce. Further research balancing for comprehensive confounding factors should be conducted to look for additional evidence for causal association between dental caries and SHS.

References

References are available on request at editor@healtalkht.com