A Journey Since 1899 To 2013 Finding Associations between Tobacco Smoking and Dental Caries.

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ABSTRACT

Dental caries remains one of the most common infectious diseases of mankind. Dental caries and its consequences together constitute a very real and personal problem for almost everyone. Several studies indicate an association with smokeless tobacco and caries but tobacco smoking and its relation to dental caries is a subject of many opinions. The present paper discusses various studies done in this regards to find the relation between dental caries and smoking using different parameters and emphasizes on studies, clinical trials and experiments needed to confirm the independent effect of smoking as one of the causes of dental caries.

INTRODUCTION

Cigarette smoking and other tobacco use imposes a public health problems globally. According to the WHO, in 2001; there were 1.1 billion smokers, 800 million of whom inhibit developing countries [¹]. This equates to approximately one third of the world’s entire population over 15 years of age and represents an enormous global health problem. Tobacco use is directly related to a variety of medical problems including cancer, low birth weight, pulmonary and cardiovascular diseases. It affects the oral cavity first, so it is evident that smoking has many negative influences on oral cavity too.

Effects Of Smoking On The Mouth (modified from Reibel) [²]

Teeth
- Dental caries
- Discolorations of teeth and restorations
- Increased failure rates for dental implants

Tongue
- Coated/hairy tongue
- Reduced ability to taste
- Oral candidosis

Gingiva, Buccal Mucosa and Palate
- Smokers’ melanosis
- Smokers’ palate
- Periodontal disease
- Smokers’ white patch/leukoplakia

Miscellaneous
- Oral cancer
Dental caries is defined as an irreversible microbial disease of the calcified tissues of the teeth, characterised by demineralisation of the inorganic portion and destruction of the organic substance of the tooth which often leads to cavitations.[3]

**Various Studies Done To Co-Relate Smoking and Dental Caries**

Hart AC, 1899[4]; Schmidt HJ, 1951[5] and Gibbs MD, 1952[6] reported that the increase in tobacco smoking was followed by a decrease in caries rate. Smoking increases thiocyanate level in saliva. Thiocyanate, a normal constituent of saliva, was found to have a possible caries inhibiting effect.

Ludwick W and Massler M in 1952 studied relation of dental caries experience and gingivitis to cigarette smoking in males of 17 to 21 years old and reported that those who smoked more than 15 cigarettes a day had significantly higher number of decayed, missing, and filled teeth[7].

In 1971, Ainamo J., found that increased smoking resulted in significantly higher number of decayed surfaces per dentition and also noted a trend toward more missing surfaces and fewer restored surfaces in subjects with a high consumption of cigarettes[8].

In 1980, Modeer, T. et al in a study found that 21.5% school children smoked regularly. Snuff was taken regularly by 11% of the boys, but none of the girls. Snuff was present in the oral cavity for an average of 3.5 hours every day. The variable number of cigarettes in this study was found to be a significant (p < 0.01) predictor to the dependent variable plaque after controlling for the predictors frequency of tooth-brushing and sex and relating the dental caries and smoking.[9]

In 1975, Ørstavik and Brandtzaeg reported that low titres of parotid S-IgA appeared to correspond with higher rates of dental caries. However, since the levels of lgA antibody rather than IgA immunoglobulin may be important, these studies were not definitive, in co relating the dental caries and secretory IgA.[10]. Thereafter, Challacombe SJ, in 1980 studied serum and salivary antibodies to Streptococcus mutans in relation to the development and treatment of dental caries and stated that salivary IgA is not directly related to protection against dental caries, but reflects a past exposure of the host to cariogenic microorganisms[11]. In 1981, McGehee and Michalek found that subjects with IgA deficiency fell into two groups in terms of oral antibody i.e. those with compensatory IgM antibodies against S. mutans in saliva and those without. Only in the group without compensatory IgM was the caries activity significantly higher than age sex matched controls. This study was indicating a co relation of secretory IgA and dental caries and smoking[12]. In 1982, Bolton, R.W. and G.L. Hlava examined children for caries activity and their salivary IgA were evaluated for reactivity to antigens of cariogenic bacteria by an enzyme-linked immunosorbent assay (ELISA). IgA levels to Streptococcus mutans were higher in children with no detectable caries. Analysis of lgA specific for Lactobacillus casei, teichoic acid, and glucan revealed no protective role for these specificities in children[13].

In a longitudinal population study in Sweden performed by Ahlqwist et al in 1989; the differences in the number of teeth lost between female smokers and non-smokers were compared after a 12-year observation. During this period, the mean number of teeth lost was 3.5 among smokers and 2.1 among non-smokers, which was over 60% higher among smokers than among non-smokers.[14] Cigarette smoking influences the risk of oro-gastrointestinal disease in both protective (ulcerative colitis), and inductive (squamous tumours of the head, neck and oesophagus) roles.

In order to study the effects of smoking on mucosal immunity, salivary immunoglobulins were measured by Barton et al(1990), in pure parotid saliva from groups of healthy non-smokers, smokers, and ex smokers and from patients with epithelial head and neck tumours, both untreated and after radiotherapy. Of the healthy individuals, smokers had significantly lower Salivary IgA and higher IgM concentrations than did non-smokers. The effect on IgA was dose related, and reversible after cessation of smoking. Likewise, in patients with head and neck tumours (the majority being smokers), salivary IgA concentration was reduced and IgM increased when compared with non-smoking controls. Results were similar before and after radiotherapy. This study provided evidence of the effects of smoking on mucosal immunity as evaluated by parotid salivary immunoglobulins.[15] Zitterbart PA confirmed association between smoking and the prevalence of dental caries in adult males. Smokers had significantly higher DMFT (Decayed, Missing, and Filled Teeth) score, untreated decayed surfaces, and missing surfaces. He further correlated that more cigarettes consumed per day resulted in more missing tooth surfaces in a smoker’s mouth.[16]

In 1991, Camling have observed neither salivary lgA nor crevicular lgG, corresponds with colonization by cariogenic bacteria. According to him, crevicular lgG antibody is produced locally and appears to reflect caries experience rather than protection and these results did not mean that naturally-induced antibodies were unable to interrupt the caries process. One should maintain this perspective, although bacterial colonization was not impaired, the issue of bacterial metabolism and current caries activity was not addressed. Caries had been correlated with
elevated S-IgA antibodies and elevated serum IgM antibodies to S. mutans. This probably reflected the elevation of antibody which occurs during and after infections. [17]

In 1992 Hajishengallis et al., have shown that human parotid S-IgA antibodies against surface antigen I/II of S. mutans could block S. mutans adhesion to saliva-coated hydroxyapatite, suggesting that there is a mechanism of protection available to the host against certain cariogenic bacteria. Specific IgA antibodies against S. mutans had been measured and found to be significant in parotid saliva against all of the major serogroups of S. mutans. However, as yet, there had been no strong correlation between such antibodies in saliva and resistance to dental caries. [18] Locker D examined the relationship between smoking and oral health in older adults. The data were taken from a cross-sectional study of oral health and treatment needs among persons aged 50 years and over living independently in four Ontario communities from 907 subjects. Half of these reported a history of smoking and one fifth were current smokers. Current smokers were more likely to have lost all their natural teeth than those who had never smoked. Among those retaining one or more natural teeth, current smokers had fewer teeth, fewer functional units, more crown surfaces with decay and more decayed root surfaces. [19] Smith and Taubman stated that it is clear that infants and young children rapidly develop S-IgA antibodies against many oral antigens, presumably by the entero salivary pathway. However, an association between these S-IgA antibodies and resistance to dental infection by these pathogens has yet to be convincingly demonstrated. [20]

In 1994, Rose P.T. et al compared IgA antibody levels with S. mutans in whole and parotid saliva from 20 caries-susceptible (CS; DMFS > 5) and 20 caries-resistant (CR; DMFS < or = 1) children (aged 7-11 years). Whole salivary S. mutans numbers were significantly greater (P < or = 0.05) in the CS group (mean of 31.2% of total oral streptococci) than in the CR group (mean of 1.6% of total oral streptococci). Whole saliva, but not parotid saliva, from CR children had significantly higher (P < or = 0.05) levels of IgA antibodies to S. mutans than saliva from CS children. These results suggested that salivary IgA antibodies to S. mutans may play a role in natural protection from dental caries in children and that the source of increased salivary IgA antibody in CR children may be either the minor, submandibular, or sublingual salivary glands. [21] Gunnar Holm studied smoking as an additional risk for tooth loss. A total of 273 individuals were followed for 10 years, during which 93 individuals lost a total of 260 teeth. Younger individuals and especially males smoking more than 15 cigarettes a day were found to have the highest relative risk of losing teeth (4.55 and 3.18 respectively). In the younger age groups the proportional attributable risk was also highest; 78% for smokers smoking more than 15 cigarettes a day. The combination of a high plaque score and smoking was, together with age, the strongest predictor of tooth loss. The findings of this study suggest that smokers, especially those in the age group < 50 years, are a high risk group for tooth loss. [22] Kassirer B described smoking as a risk factor for gingival problems, periodontal problems and caries, in a similar study. [23]

In 1995, Telivuo M. et al., conducted a survey to study smokers' oral health behaviours and attitudes, and to determine if smokers were advised by their dentists to quit smoking. A random sample of 1,200 adults 15 to 64 years of age living in the province of North Karelia, Finland, was selected in each of two study years (1990 and 1991) and surveyed using a mail questionnaire. The 102-item questionnaire solicited information on smoking status, oral health behaviours, missing teeth, perceptions of tobacco's harmful effects on oral health, smoking status and quitting, and advice on smoking cessation provided by dentists. Variations in behaviours and opinions according to smoking status were analyzed. Nonsmokers reported more frequent healthy oral health behaviours than did daily smokers, with the exception that no difference in tooth brushing frequencies existed among women. Daily smoking was associated with increased use of sugar in tea or coffee, and with more frequent alcohol consumption. Daily smoking was correlated with the number of missing teeth in bivariate analyses, but not in multivariate analyses. Fewer daily smokers than nonsmokers considered smoking to have harmful effects on oral health. The majority of daily smokers, however, wanted to quit. Eight percent of daily smokers reported that they had been advised by their dentist to quit. [24]

In 1998, Axelsson P. et al; studied relationship between smoking and dental status in 35-, 50-, 65-, and 75-year-old individuals and concluded that smoking is a significant risk indicator for tooth loss, probing attachment loss and dental caries. [25]

In 1999, Griesel A G and Germishuys P J. studied to determine salivary immunoglobulin A levels in people who stopped smoking for at least 2 weeks. Salivary immunoglobulin A levels of each of 20 subjects were determined on 3 occasions: first, while the subject was still smoking; second, 7 days after cessation of smoking; third, on the 14th day after cessation. Two control groups (with 20 people in each group) were also used: the members of the first had never smoked, and the members of the second were current smokers. Seven days after cessation of the smoking habit, a transient decrease in salivary immunoglobulin A levels was observed. However, within 14 days the levels returned to normal. [26] Lennart Unell et al., did research on the explanatory models for clinically determined and symptom-reported caries indicators in an adult population. In general, the association pattern was as could be expected: immigrants, working class, low education, smoking, dissatisfaction with dental treatment and low utilization all appeared as risk factors for both the clinically determined caries indicators, but not
necessarily for subjective symptom reports. Only fear of dental treatment showed a consistent positive association with all the indicators. [27]

In 2000, Williams SA. et al. studied Parental Smoking Practices and Caries Experience in Pre-School Children. Data analysis was confined to 749 children aged 3.0–4.5 years, to avoid confounding effects of unerupted teeth. Bivariate analysis indicated that the prevalence of maternal rather than paternal smoking was significantly related to caries and substantially attenuated social class differences. The reported number of cigarettes smoked was not important. It is concluded that maternal smoking is a significant factor to be considered as an additional risk indicator beyond social class when predicting caries risk in young children.[28] Benderli Y. et al. in the same year suggested that salivary S-IgA antibodies generated by the mucosal immune system play an important role in the immune response against dental caries. [29] Sgan-Cohen H.D. et al. conducted a study among a representative sample of 7139 21-year-old Israeli adults, upon release from compulsory military service. Data were collected between 1994 and 1997. The average DMFT level found was 8.49±4.95. Untreated caries (according to the D component of DMFT) was 2.25±2.90 and significantly higher among males. Untreated caries was also significantly associated with geographic origin: higher among subjects of African or former USSR descent; and with family size: higher among subjects with four or more siblings; with education: caries was higher among subjects with less than 12 years of schooling; and with smoking: caries was more extensive among those who smoked (P<0.0001 for all the associations). [30]

Pekka Ylöstalo et al. studied the relation of tobacco smoking to tooth loss among young adults. The data consisted of the 1966 birth cohort of Northern Finland, which was an unselected general population birth cohort (n = 12 058). The data were collected using a postal questionnaire in 1997–98 (n = 8690). Prevalence odds ratios and confidence intervals were estimated by applying a logistic regression model. It was found that smoking was associated with tooth loss in an exposure-dependent manner. [31]

In 2005, Bruno-Ambrosius K. et al. studied a sample consisting of a cohort of 162 girls under regular dental care, aged 12 years at baseline, who were followed for 3 years, from the sixth to the ninth grade. Eating, oral cleaning and smoking habits were self-reported three times per year through a questionnaire, and caries data at baseline and after 3 years were collected from dental records. The results showed significantly (P < 0.05) impaired eating habits during the study period and that adherence to regular main meals diminished. In the eighth grade, one-third of the girls skipped breakfast before school and only 50% had their free school lunch daily. The omission of breakfast and irregular main meals, as well as smoking were significantly associated with caries (decayed, missed and filled surfaces) increment in the eighth grade (odds ratio = 4.1-4.9, P < 0.05). [32] Kelbauskas E. et al. determined the prevalence of smoking among Lithuanian army recruits and how smoking and other factors affect oral health. The findings showed that poor education and living in the country, irregular tooth brushing, poor oral hygiene, and smoking were the most important factors related to a great number of untreated decayed tooth surfaces. [33]

In 2006, Heng C.K. et al. investigated the relationship of cigarette smoking to dental caries among female inmates of a federal correctional institution. Two-hundred inmates (age ranges 19-62) entering the institution were given an oral examination and a self-administered questionnaire. A high percentage, 64%, of inmates was current or former smokers. Although this study did not establish a causative relationship, cigarette smoking was shown to be associated with the experience of caries. [34] They investigated the association of active and passive smoking exposure with tooth loss in Japan: Study subjects were 1002 pregnant women. Tooth loss was defined as previous extraction of one or more teeth. Adjustment was made for age, gestation, parity, family income, education, and body mass index. The findings suggested that passive as well as active smoking may be associated with an increased prevalence of tooth loss in Japanese young adult women. [35] Y. Okamoto et al. studied the effects of smoking and drinking habits on the incidence of periodontal disease and tooth loss among Japanese males in a 4-year longitudinal study. The subjects were 1332 Japanese males, 30-59 yr of age, who were free from periodontal disease at the baseline check-up, and who underwent a second check-up 4 yr later. Periodontal disease was diagnosed using the community periodontal index score, based on the clinical probing of pocket depth (≥ 4 mm). Smoking and alcohol consumption patterns were evaluated using a self-administered questionnaire. Cigarette smoking was found to be an independent risk factor for periodontal disease and tooth loss. [36]

In 2007, Dietrich T. et al. studied the tobacco use and incidence of tooth loss among US male health professionals. They found that the data on the dose-dependent effects of smoking and smoking cessation on tooth loss are scarce. They hypothesized that smoking has both dose- and time-dependent effects on tooth loss incidence. [37]

In 2008, Sroisiri, T. et al. revealed that children with rampant caries had significantly higher levels of S-IgA in their oral cavities. Measurement of S-IgA was performed using an immunoassay kit. This finding tends to support the hypothesis that higher levels of salivary S-IgA may reflect a past exposure of the host to cariogenic microorganisms. [38]
Aguilar-Zinser V et al. studied among professional truck drivers in Mexico and their higher age, poorer oral hygiene, higher education and greater tobacco exposure were associated with higher caries experience (DMFT). It has also been shown that smoking is associated with poor oral hygiene and food habits, which could increase the caries risk. [39]

In 2009, T Yanagisawa et al. studied to determine the relationship of number of teeth with smoking and smoking cessation. Subjects included 547 males aged between 55 and 75 years. Oral examinations were conducted in 2005. Smoking status information was collected from questionnaire surveys conducted in 1990, 1995, 2000, and 2005. The relationship between having more than eight missing teeth and smoking status was estimated. They concluded that smoking had a positive association with the number of missing teeth and smoking cessation is beneficial for maintaining teeth. [40, Zinser V, et al. discussed that passive smoking was associated with a decrease in secretory immunoglobulin A (S-IgA) concentration in young children. [41] Roushdy M.M. studied the association of dental caries, streptococcus mutans counts and secretory IgA with tobacco smoking. Dental caries were significantly related to the presence of Streptococcus mutans as well as tobacco smoking. Furthermore, higher levels of microbial antigenic loads present in the oral cavity of the individuals under investigation probably increase the production of S-IgA. On the other hand the levels of S-IgA in saliva were significantly decreased tobacco smokers either with dental caries or without dental caries in comparison with non-smokers ones. [42]

In 2010, Tanaka K et al. did a cross-sectional study to examine the potential association between second hand smoke exposure at home and the prevalence of dental caries in children. The findings suggested that household smoking might be associated with an increased prevalence of dental caries in children. [43]

In 2011, Campus G et al. did a cross-sectional survey designed to clarify if smoking habit increases the caries risk in a sample of Italian adults attending a Military Academy. Clinical examinations including dental caries and presence of bleeding at probing were carried out following WHO criteria. Related socio-behavioural factors were collected. Heavy smokers attending a Military Academy showed a higher prevalence of caries, confirming a correlation between the disease and tobacco use. [44]

Table 1: Showing year wise studies done by different authors finding association between dental caries and smoking

<table>
<thead>
<tr>
<th>Serial No.</th>
<th>Author With Year</th>
<th>Association Between Dental Caries And Smoking</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>Hart AC, 1899</td>
<td>Negative</td>
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<tr>
<td>2.</td>
<td>Schmidt HJ, 1951</td>
<td>Negative</td>
</tr>
<tr>
<td>3.</td>
<td>Gibbs MD, 1952</td>
<td>Negative</td>
</tr>
<tr>
<td>4.</td>
<td>Ludwick W and Massier M in 1952</td>
<td>Positive</td>
</tr>
<tr>
<td>5.</td>
<td>Ainamo J. 1971</td>
<td>Positive</td>
</tr>
<tr>
<td>7.</td>
<td>McGhee and Michalek, 1981</td>
<td>Positive</td>
</tr>
<tr>
<td>8.</td>
<td>Ahlwist et al., 1989</td>
<td>Positive</td>
</tr>
<tr>
<td>10.</td>
<td>Locker D, 1992</td>
<td>Positive</td>
</tr>
<tr>
<td>11.</td>
<td>Gunnar Holm, 1994</td>
<td>Positive</td>
</tr>
<tr>
<td>15.</td>
<td>Lennart Unell et al. 1999</td>
<td>Positive</td>
</tr>
<tr>
<td>18.</td>
<td>Pekka Ylöstalo et al.,2004</td>
<td>Positive</td>
</tr>
<tr>
<td>20.</td>
<td>Kelbauskas E. et al,2005</td>
<td>Positive</td>
</tr>
<tr>
<td>21.</td>
<td>Heng C.K et al, 2006</td>
<td>Might Be</td>
</tr>
<tr>
<td>22.</td>
<td>Y.Okamoto et al.2006</td>
<td>Positive</td>
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<tr>
<td>23.</td>
<td>Dietrich T.et al., 2007</td>
<td>Positive</td>
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<td>24.</td>
<td>Aguilar-Zinser V et al., 2008</td>
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<td>25.</td>
<td>T Yanagisawa et al, 2009</td>
<td>Positive</td>
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<tr>
<td>27.</td>
<td>Tanaka K.et al., 2010</td>
<td>Might Be</td>
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<tr>
<td>28.</td>
<td>Campus G. et al, 2011</td>
<td>Positive</td>
</tr>
<tr>
<td>30.</td>
<td>L Golpasand et al, 2013</td>
<td>Positive</td>
</tr>
</tbody>
</table>

In 2012, Hugoson A et al. did epidemiological studies to study dental caries in relation to smoking and the use of Swedish snus: covering 20 years (1983–2003). The study was done to evaluate some intra-oral caries-
associated variables and tobacco use on dental caries. The participants were randomly recruited from three cross-sectional studies in Jönköping, Sweden, in 1983, 1993 and 2003. Each study consisted of 130 individuals in each of the 20, 30, 40, 50, 60 and 70-year age groups. Of these, 550, 552 and 523 dentate individuals attended respective year of examination. They were all examined both clinically and radiographically. A questionnaire was completed in conjunction with the examination. The results of these epidemiological studies, indicate that daily smoking or snus use does not increase the risk of dental caries [45].

In 2013, L Golpasand et al studied the association of dental caries and salivary S-IgA with tobacco smoking. A total of 70 healthy subjects were selected and classified into four groups according to dental caries and tobacco smoking habits: smoking with caries (Group 1, n = 15); smoking without caries (Group 2, n = 15); non-smoking with caries (Group 3, n = 15); and non-smoking without caries (Group 4, n = 25). Salivary S-IgA was measured using ELISA. The fissure and proximal caries were examined clinically and radiographically. Caries status was determined according to the decay surface index. Smokers showed a higher number of caries and the lowest concentration of S-IgA. The findings indicate that low concentrations of salivary S-IgA are correlated with a higher prevalence of dental caries in smokers. [46] Farhang A. A and Fikry A. Q investigated the effect of cigarette smoking on some immunological and hematological parameters in Erbil city. The study was carried out on fifty male smokers, who smoked at least 10 cigarettes per day for at least 10 years. Depending on the age of the smokers, they were divided into two groups. The first group included smokers with age range between 25-35 years, and the second group included smokers with age range between 36-45 years. Two control (non-smokers) groups were collected with the same range of age for statistical comparison. A significant decrease of the total immunoglobulin A (IgA) level was recorded in both age groups when compared with their control. [47]

**Figure 1 : Summary of the studies associating dental caries and smoking**

While previously there has been no clear consensus on the aetiological relation between smoking and dental caries, there are now studies pointing to contributing factors that show smoking is a risk factor for increased caries activity. Smoker saliva has a decreased buffering effect and a higher number of lactobacilli and Streptococci mutans which in turn increase susceptibility to caries. Smoking is associated with lower salivary cystatin activity. This product contributes to oral health by inhibiting certain proteolytic enzymes. Additional risk factors in smokers include poorer oral hygiene, different eating habits (presumably consuming higher amount of sugar), poorer brushing habits and reduced frequency of dental recall than non-smokers. Studies also indicate a higher incidence of dental caries in conjunction with the lowest concentration of salivary S-IgA in smokers. Smoking influences the salivary immunoglobulin content and besides the immunosuppressive properties of smoking, the presence of some components such as nicotine can promote the growth of cariogenic bacteria (e.g. the Streptococci mutans) in smokers and passive smokers. Several studies on immunoglobulins seem to be promising in finding the relation or association between dental caries and smoking. But the exact association of dental caries with tobacco smoking still remains unknown although smoking seems to be a risk factor for increased dental caries. Further studies, clinical trials and experiments are needed to confirm the independent effect of smoking as one of the causes of dental caries.

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