



Influence of Smoking Habits on the Prevalence of Dental Caries: A Register-Based Cohort Study

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Abstract

Objective This study aimed to evaluate the influence of smoking habit on the prevalence of dental caries lesions in a follow-up study.

Materials and Methods A total of 3,675 patients (2,186 females and 1,489 males) with an average age of 51.4 years were included. Outcome measures were the incidence of dental caries defined as incipient noncavitated, microcavitated, or cavitated lesions which had been diagnosed through clinical observation with mouth mirror and probe examination evaluating change of texture, translucency, and color; radiographic examination through bitewing radiographs; or secondary caries through placement of a new restoration during the follow-up of the study.

Statistical Analysis Cumulative survival (time elapsed with absence of dental caries) was estimated through the Kaplan–Meier product limit estimator with comparison of survival curves (log-rank test). A multivariable Cox proportional hazards regression model was used to evaluate the effect of smoking on the incidence of dental caries lesions when controlled to age, gender, systemic status, frequency of dental hygiene appointments, and socioeconomic status. The significance level was set at 5%.

Results Eight hundred sixty-three patients developed caries (23.5% incidence rate). The cumulative survival estimation was 81.8% and 48% survival rate for nonsmokers and smokers, respectively ($p < 0.001$), with an average of 13.5 months between the healthy and diseased state diagnosis. Smokers registered a hazard ratio for dental caries lesions of 1.32 ($p = 0.001$) when controlled for the other variables of interest.

Conclusion Within the limitations of this study, it was concluded that smoking habit might be a predictor for dental caries.

Keywords

- dental caries
- smoking
- socioeconomic factors
- risk

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Introduction

Smoking represents one of the major risk factors for lung cancer and cardiovascular disease¹ with a well-established causal relationship. The relation between smoking and carcinoma of the lung is known since 1950 by Doll et al.² The impact of tobacco on mortality is severe, with 6 million persons dying every year according to the World Health Organization.³ Nevertheless, the consumption of tobacco remains high worldwide, with a 21% rate.⁴

Smoking habits also impact directly the oral cavity, with a wide range of effects extending from teeth/dental fillings staining, to periodontal disease, increased failure rates for dental implants and oral cancer.⁵ According to the International Agency for Research on Cancer, the estimated number of incident cases for lip and oral cavity cancer worldwide in 2012 was 300,000, with 145,000 estimated deaths.⁶

Systemic and oral diseases may share common risk factors. Consequently, a recent review suggested appropriate risk factor management procedures to be adopted in the dental setting⁷: smoking cessation, reduction of sugar consumption, and weight control were proposed for patients at risk of periodontal disease, caries, diabetes, heart disease, and certain cancers.

Dental caries is the most prevalent disease worldwide,⁷ with a majority of caries lesions being concentrated often in disadvantaged social groups: increased odds for dental caries (21–48%) were registered in patients with (own or parental) low educational/occupational background and income.⁸ According to a population-based study in Sweden between 1983 and 2003, the relatively unchanged status of tobacco consumption together with a significantly lower frequency of dental visits from smokers compared with non-smokers reveal a probable trend for an increase in the global burden of dental caries.⁹

The influence of smoking on dental caries is subject of further research, with some authors suggesting an association between an increased risk of dental caries with tobacco smoking.^{10–12} However, the authors of two of these studies (systematic reviews)^{10,11} indicated an overall poor quality of the studies included that prevented validating the association between smoking and dental caries.

The aim of this study was to evaluate the influence of tobacco smoking on the prevalence of dental caries lesions.

Materials and Methods

This register-based clinical study was written by following the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) guidelines.¹³ This clinical study was performed in a private clinic in Lisbon, Portugal between July 2012 and December 2014, and it was approved by an independent ethical committee (Ethical Committee for Health, Lisbon, Portugal; authorization no. 005/2012). All procedures in this investigation on the participants were conducted in accordance with the Declaration of Helsinki. All procedures were performed with the adequate and understanding and written consent from all participants included in the study.

The inclusion criteria were patients that presented permanent teeth without active carious lesions at the first diagnostic appointment. There was a total of 3,675 patients included in this study (2,186 females and 1,489 males; average age = 51.4 years; range = 18–97 years). The clinical examinations were performed by a team trained and calibrated clinicians (with interexaminer reliability results of 0.87 to 0.89 considering the weighted kappa scores in the 3 years of data collection). The patients were part of an epidemiological surveillance study carried to assess the incidence rate of oral diseases. The patient's attendance pattern was characterized by regular check-ups, with patient enrolment in a maintenance program that included dental hygiene and lifestyle instructions (dietary and smoking cessation) and maintenance procedures (scaling, polishing of teeth surfaces, and topical fluoride). The outcomes were recorded at each visit in an anonymized database. For the present study, the information of each patient was retrieved from a register-based database. The information collected from the sample is outlined in ► **Table 1** and included demographic variables (age and gender), socioeconomic status evaluated from the occupation of each patient according to Goldthorpe classification (socioeconomic status 1: higher managerial, administrative, and professional occupations; socioeconomic status 2: intermediate occupations; socioeconomic status 3: routine and manual occupations),¹⁴ recall regimen (average frequency of 6 months between dental hygiene appointments), systemic compromise status (absence, *n* = 2,692 patients; presence, *n* = 983 patients: hepatitis, *n* = 26 patients; cardiovascular, *n* = 636 patients; thyroid, *n* = 128 patients; diabetes, *n* = 104 patients; rheumatological, *n* = 174 patients; HIV+, *n* = 6 patients; oncological, *n* = 40 patients; inflammatory, *n* = 10 patients; neurological, *n* = 13 patients; autoimmune, *n* = 5 patients; renal, *n* = 3 patients; genetic, *n* = 3 patients; Sjögren syndrome, *n* = 1 patient; more than one condition: *n* = 157 patients), smoking status (nonsmoker; smoker), and dental caries status (absence or presence).

Outcome measures were the incidence of active dental caries considering noncavitated, microcavitated, or cavitated lesions,^{15–18} which diagnosed by (1) clinical observation with mouth mirror and probe examination (blunt probe, Hu-Friedy, Chicago, Illinois, United States) evaluating change of texture, translucency, and color; (2) radiographic examination through bitewing radiographs (Kodak film bitewing DF42, Eastman Kodak Company, Rochester, New York, United States; Kwik-bite, Kerr, Bioggio, Switzerland); or (3) secondary caries through the placement of a new restoration. Survival was defined as the absence of dental caries during the follow-up of the study.

The onset of dental caries was defined as the time period elapsed between the healthy state and the diagnosis of dental caries during the follow-up of the study.

Statistical Analysis

The data was analyzed by using SPSS version 17.0 (IBM, Rochester, New York, United States). Descriptive statistics were computed for the variables age, gender, socioeconomic status, systemic compromise status (hepatitis, cardiovascular condition, thyroid condition, diabetes, rheumatologic condition, HIV+, oncological condition, inflammatory condition,

Table 1 Sample characteristics

Variables	Total (n = 3675 patients)	Dental caries (n = 670 patients)	Healthy (n = 3005 patients)
Age mean (standard deviation)	51.4 (14.8) y	50.3 (15.3) y	51.8 (14.7) y
Gender			
Female	2,186 (100%)	505 (23.1%)	1,681 (76.9%)
Male	1,489 (100%)	358 (24%)	1,131 (76%)
Smoking habits			
Nonsmoker	3,005 (100%)	675 (22.5%)	2,330 (77.5%)
Smoker	670 (100%)	188 (28.1%)	482 (71.9%)
Systemic compromise			
Healthy	2,692 (100%)	621 (23.1%)	2,071 (76.9%)
Systemic compromised	983 (100%)	242 (24.6%)	741 (75.4%)
Frequency of dental hygiene appointments: mean (standard deviation)	6.3 (2.6) mo	6.7 (3.4) mo	6.2 (2.3) mo
Socioeconomic status			
Status 1	2,261 (100%)	535 (23.7%)	1,726 (76.3%)
Status 2	660 (100%)	152 (23%)	508 (77%)
Status 3	395 (100%)	95 (24.1%)	300 (75.9%)

Note: Socioeconomic status classification according to Goldthorpe¹⁴:

Status 1: Higher managerial, administrative, and professional occupations.

Status 2: Intermediate occupations.

Status 3: Routine and manual occupations.

neurologic condition, autoimmune condition, renal condition, and Sjögren syndrome), smoking status, dental caries status, and onset of dental caries. Cumulative survival was estimated through the Kaplan–Meier product limit estimator and by using the patient as the unit of statistical analysis (first incidence of a dental caries lesion was considered as event). Differences between survival curves were determined by using the log-rank test. The multivariable Cox proportional hazards regression model was used to evaluate the effect of smoking on the incidence of dental caries lesions controlled for the presence of other covariates/confounders. The covariates inserted in the model were age, gender, smoking status, systemic compromise status, frequency of dental hygiene appointments, and socioeconomic status. Regression coefficients (hazard ratios [HR]) were estimated with corresponding 95% confidence intervals (95% CI). The survival function was tested for prediction by using the area under the curve statistic (AUC) and illustrated by the receiver operating characteristics curve (ROC curve). The level of significance was set at 5%.

Results

From 4,179 patients eligible for follow-up, 504 (12%) were lost to follow-up and excluded from analysis, with analysis performed on the data of the remaining 3,672 patients. During the follow-up of the study, 2,812 patients had no incidence of dental caries while 863 patients developed dental caries (incidence rate of 23.5%). The average follow-up for each patient was 2 years (range = 1–2.5 years). The average time of onset for dental caries was 13.5 months 95% CI (13.0–13.9).

The distributions of survival rate estimates (Kaplan–Meier) were the following: estimated cumulative survival rates for females and males were 59.5 and 59.8%, respectively, with a nonsignificant difference between both groups ($p = 0.433$); estimated cumulative survival rates for nonsmokers and smokers was 81.8 and 48.0%, respectively, with a significant difference between both groups ($p < 0.001$; **Fig. 1**); estimated cumulative survival rates for healthy and systemic compromised patients was 59.1 and 60.7%, respectively, with a nonsignificant difference between both groups ($p = 0.504$); and estimated cumulative survival rates for socioeconomic

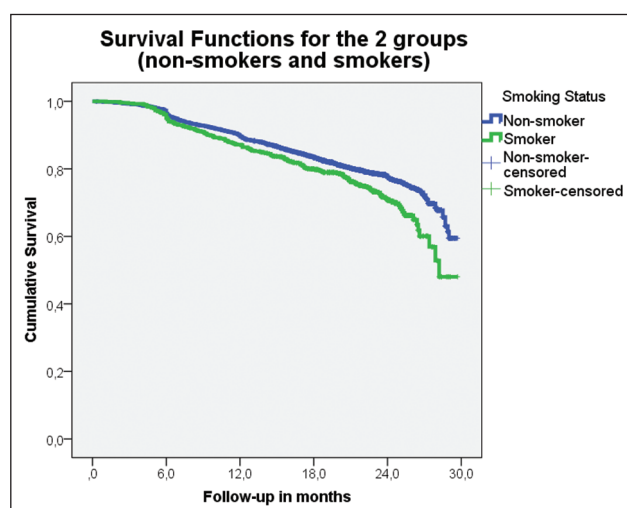


Fig. 1 Kaplan–Meier survival curves for dental caries lesions in non-smoker and smoker patients; $p < 0.001$, hazard ratio = 1.316, 95% confidence interval = 1.114–1.555.

status 1, 2, and 3 were 58.5, 53.3, and 70.8%, respectively, with a nonsignificant difference between both groups ($p = 0.943$).

The multivariable model identified smoking habits (HR = 1.32) as a predictor for dental caries lesions and age as a protective effect (HR = 0.99) when controlled for gender, systemic compromised status, frequency of dental hygiene appointments, and socioeconomic status (►Table 2). The survival function retrieved from the model by using the six predictors registered an AUC of 0.835 and 95% CI (0.815–0.855; ►Fig. 2).

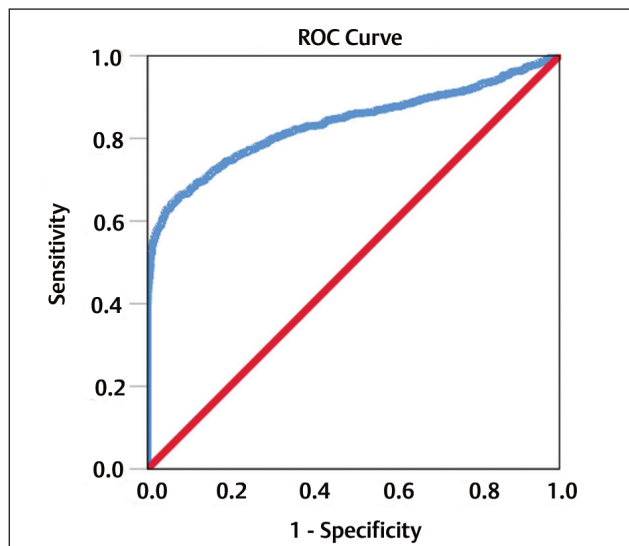


Fig. 2 Receiver operating characteristic curve illustrating the performance of the regression model in prediction of dental caries lesions. The model accuracy according to the C-statistic (95% confidence intervals) was area under the curve = 0.835 (0.815–0.855).

Discussion

This study registered a significant association between smoking habits and dental caries prevalence, with a 32% increase in the hazard ratio for developing dental caries for smokers compared with nonsmokers. This result is in line with the literature^{10,11,19} using a register-based study design and considering the control for other variables of interest. A systematic review¹⁰ evaluated the effect of tobacco smoking on dental caries in adult smokers and disclosed a significant association between the exposure to smoking and an increased risk of dental caries. Bernabé et al¹² in a study that included 955 adult patients, assessed the impact of daily smoking on the 4-year net increment in the numbers of decayed, filled, and missing teeth also reported an increased incidence of dental caries in smokers. In the referred study, the authors concluded that smoking was independently related to caries development with a 70% increment on the incidence rate ratio of decayed teeth for smokers when compared with nonsmokers.

Smoking might potentially influence the incidence of dental caries through the negative impact on the patients' saliva. The saliva properties of smokers include a higher count of bacteria (*Streptococcus mutans*)¹⁸ and a decreased buffering effect that may increase the patient's susceptibility to dental caries,^{18,20} accounting for a 25% of the variability of caries risk.²⁰ Furthermore, smoking was previously associated with lower salivary secretory immunoglobulin A (IgA) concentrations.²¹ The specificity of IgA action represents the main specific defense mechanism in the oral cavity.²² In conjunction with other antimicrobial substances, the IgA action limits microbial adherence to epithelial and tooth surfaces helping to maintain the oral cavity stable.^{23–25} Smoking

Table 2 Multivariate Cox regression model for risk factors for dental caries lesions (total $n = 3,675$ patients)

Factor	Hazard ratio	95% confidence interval	p-Value
Age	0.991	(0.986–0.996)	0.001
Gender			
Female	1.0		
Male	1.036	(0.897–1.196)	0.635
Smoking habits			
Nonsmoker	1.0		
Smoker	1.316	(1.114–1.554)	0.001
Systemic compromise			
Healthy	1.0		
Systemic compromised	1.184	(0.995–1.409)	0.057
Frequency of dental hygiene appointments	1.002	(0.975–1.030)	0.879
Socioeconomic status			0.735
Status 1	1.0		
Status 2	0.984	(0.820–1.181)	0.864
Status 3	1.085	(0.868–1.356)	0.473

Note: Socioeconomic status classification according to Goldthorpe¹⁴:
 Status 1: Higher managerial-administrative and professional occupations.
 Status 2: Intermediate occupations.
 Status 3: Routine and manual occupations.

was also associated to lower salivary cystatin activity and cystatin C²⁶ that contributes to oral health by inhibiting certain proteolytic enzymes suggested to have a contributory effect on caries lesion formation when in interaction with acid demineralization.²⁷

Age had a protective effect in our study (HR = 0.99), a result that was also registered in a previous study¹² and that can be explained by the exposure time. With increased age, there is a higher probability of patients with missing or filled teeth and therefore a lower conditional probability of incident dental caries lesions. The variable socioeconomic status did not yield statistical significance, opposing the results from other study.⁸ Schwendicke et al⁸ registered a 29% increase in the odds ratio of caries experience for low income compared with high income in their meta-analysis, despite grading the included studies between low and very low evidence levels due to risk of bias. A probable reason for the different result may reside in the study setting, with the present study performed on a private practice where patients attended a recall regimen in comparison to the Schwendicke et al meta-analysis,⁸ where population-based studies were included. Nonetheless, the short-term development of dental caries had a similar distribution between the three socioeconomic classes, which accounts for the importance of patient clinical monitoring in preventing the incidence of dental caries lesions.

The multivariable model had an accuracy of 0.835 according to the AUC statistic. This result is considered excellent when evaluating the performance of prediction models.²⁸ The high accuracy translates into a high proportion of true positives and true negatives among the total number of patients when predicting the probability of experiencing dental caries. This model may be valid for screening patients with high risk of developing dental caries lesions and may be used as an aid in the decision process for implementing more strict methodologies of dental caries prevention and smoking cessation.

Dental caries lesions consist in a dynamic process resulting from an imbalance between demineralization and remineralization of the dental surface.²⁹ The time of onset for dental caries registered in the present study was 13.5 months. According to the authors' knowledge, this is the first time such statistic has been computed. Despite the methodology implemented in patients' data collection and monitoring system, this statistic data should be interpreted with caution as it was dependent on recall appointments. It needs to be further refined to retrieve more precise measurements due to the possibility of an earlier diagnosis.

The clinical implications of the time of onset for dental caries is three partied. First, concerning the recall regimen, the results of the present study suggest reducing the recall periods for patients who smoke due to the higher probability of developing dental caries registered in the study; second, a reduced recall regimen would potentially increase the frequency of diagnostic measures; and third, it would enable monitoring the patient adherence to dietary, smoking and oral hygiene habits.

The limitations of this study include the short-term follow-up that could impact the prevalence rate of carious

lesions and the lack of control for dietary and oral hygiene habits (considering it was a register-based study with no intervention on patients) that could have provided a more precise estimation of the effect of smoking on dental caries. Nevertheless, the study sample consisted in patients enrolled in a maintenance program, part of an epidemiological surveillance study, where all patients received information on good dietary and oral hygiene habits and smoking cessation as per protocol on the dental hygiene appointments. Furthermore, the results were controlled for the recall regimen (frequency of dental hygiene appointments). It was previously stated in the literature, that smoking may be associated with decreased oral hygiene habits with increased plaque accumulation³⁰⁻³² either related to decreased toothbrushing and flossing frequency³² or due to elevated salivary lipid levels.³³ Further investigations should focus on controlled longer follow-up studies to refine the estimates of both the association of smoking and dental caries and the time of onset for dental caries lesions.

In conclusion and considering the limitations of this study, smoking habits might be a predictor for dental caries while age had a protective effect when adjusted for gender, systemic compromise, socioeconomic status, and frequency of dental hygiene appointments.

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None.

Conflict of Interest

None declared.

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