Abstract

Tobacco smoking is the main risk factor associated with chronic destructive periodontal disease. No other known factor can match the strength of smoking in causing harm to the periodontium. The harmful effects manifest themselves by interfering with vascular and immunologic reactions, as well as by undermining the supportive functions of the periodontal tissues. The typical characteristic of smoking-associated periodontal disease is the destruction of the supporting tissues of the teeth, with the ensuing clinical symptoms of bone loss, attachment loss, pocket formation, and eventually tooth loss. A review of the international literature that has accumulated over the past 20 years offers convincing evidence that smokers exhibit greater bone loss and attachment loss, as well as more pronounced frequencies of periodontal pockets, than non-smokers do. In addition, tooth loss is more extensive in smokers. Smoking, thus, considerably increases the risk for destructive periodontal disease. Depending on the definition of disease and the exposure to smoking, the risk is 5- to 20-fold elevated for a smoker compared to a never-smoker. For a smoker exposed to heavy long-life smoking, the risk of attracting destructive periodontal disease is equivalent to that of attracting lung cancer. The outcome of periodontal treatment is less favorable or even unfavorable in smokers. Although long-term studies are rare, available studies unanimously agree that treatment failures and relapse of disease are predominantly seen in smokers. This contention is valid irrespective of treatment modality, suggesting that smoking will interfere with an expected normal outcome following commonplace periodontal therapies. The majority of available studies agree that the subgingival microflora of smokers and non-smokers are no different given other conditions. As a consequence, the elevated morbidity in smokers does not depend on particular microflora. The mechanisms behind the destructive effects of smoking on the periodontal tissues, however, are not well understood. It has been speculated that interference with vascular and inflammatory phenomena may be one potential mechanism. Nicotine and carbon monoxide in tobacco smoke negatively influence wound healing. Smoking research over the past two decades has brought new knowledge into the domains of periodontology. Even more so, it has called into question the prevailing paradigm that the disease is primarily related to intraoral factors such as supra- and subgingival infection. Smoking research has revealed that environmental and lifestyle factors are involved in the onset and progression of the disease. Being the result of smoking, destructive periodontal disease shares a common feature with some 40 other diseases or disorders. As a consequence, periodontal disease should be regarded as a systemic disease in the same way as heart disease or lung disease. Thus, chronic destructive periodontal disease in smokers is initiated and driven by smoking. Its progression may or may not be amplified by unavoidable microbial colonization.

Key words

Periodontal disease · Periodontal treatment · Risk factor · Smoking · Tobacco

Introduction

Tobacco smoking is the number one enemy of periodontal health. No other as yet identified factor can parallel the force of smoking as a destroyer of the periodontal tissues. The destructive force of smoking against the periodontal tissues clinically appears in several forms. On one hand, subtle signs of inflammation are suppressed by its interference with vascular and immune reactions and, on the other, the supporting functions are undermined by its destructive action on the bone tissue, the consequences being bone loss, pocket formation, and tooth loss. To describe this latter process, the term “(chronic) destructive periodontal disease” will be used in the following text. The present review provides an evaluation of the current literature on the
subject of tobacco smoking and periodontal health or
disease, with the objective being to yield a comprehensive
summary of available information and to serve as an evi-
dence base for clinical management. The review concerns
epidemiological and clinical observations with reference to
smoking and (1) periodontal inflammation, (2) destructive
periodontal disease, (3) risk for periodontal disease, (4)
periodontal therapy, (5) periodontal microflora, (6) poten-
tial mechanisms of action, and (7) periodontal health in the
future.

**Smoking and periodontal inflammation**

The suppression of hemorrhagic responsiveness is the ear-
est and probably the least noticed effect of smoking on the
periodontal tissues. This effect is subtle; it often escapes clin-
dical detection and may even be recognized as something
positive and healthy. However, the non-bleeding or inade-
quately bleeding gingiva of a smoker may in fact be far
from healthy. The tendency of an inadequate inflammatory
response among smoker patients was first described in
the early 1980s. Studies during later years have confirmed
these observations and contributed additional evidence
that the gingival inflammatory response is altered by
smoking. The anti-hemorrhagic and anti-inflammatory
effects imply that typical symptoms of inflammation, such
as redness, bleeding, and edema appear comparably late in
the disease process, if at all, in the average smoker patient.
Interestingly, in smokers undergoing smoking cessation pro-
grams, gingival bleeding and gingival blood flow, as well as
gingival crevicular fluid flow, increase and normalize toward
non-smoker levels after quitting.

**Smoking and destructive periodontal disease**

Smoking is associated with excessive destruction of the sup-
porting periodontal tissues, resulting in bone loss, pocket
formation, and premature tooth loss. It is well documented
in the literature that bone loss and attachment loss are sig-
ificantly more pronounced in smokers compared to
non-smokers. The same conclusion holds true for peri-
dontal pocketing. Almost all studies that have utilized
periodontal probing or probing depth as a surrogate endpoint
for destructive disease report a significantly greater pocket
frequency and/or probing depth among smokers compared
to non-smokers. Moreover, smokers suffer a greater rate of
tooth loss than non-smokers. Tooth loss can, indeed, be the
result of other circumstances than periodontal disease. How-
ever, when the confounding effects of other factors, such as
caries, have been controlled for, it becomes evident that excess tooth loss in the average smoker is asso-
ciated with the periodontal destruction. It is unrelenting
logic in this gradually increasing interference of smoking:
from suppression of the inflammatory defense reaction via
destruction of the supporting bone to loss of the teeth.

The results of the large body of investigations sum-
marized above are remarkably straightforward, in spite
of a great variety among the separate studies with respect
to populations studied, endpoint selection, and strategy of
performance. As may be expected, cross-sectional studies
constitute the predominant type of study design. Cross-
sectional studies make possible the disclosure of associa-
tions between potential risk factors such as smoking and the
occurrence of disease. However, they do not permit con-
clusions about causal relations. For such a purpose, con-
secutive observations over time are necessary. Longitudinal
or cohort studies may pick up changes that occur over time
and, therefore, they possess a potential for disclosing causal
effects. Because clinical trials, for ethical reasons, cannot be
performed with respect to smoking, cohort studies are the
most powerful means of investigating possible causative
effects from smoking. Results are currently available from
a limited number of cohort studies where smokers and non-
smokers have been compared with reference to the pre-
valence or severity of periodontal disease. All of these unani-
mosly indicate that periodontal disease more frequently affects
and is more severe in smokers than in non-smokers. Intriguingly, studies including former
smokers who have given up smoking in the past conclude that former smokers are less frequently affected than
smokers or that they exhibit less severe disease. This is an
important observation, suggesting that quitting smoking is
beneficial from a periodontal health point of view.

Investigations comparing smokers who have varying
levels of exposure are of great value to test the existence of
dose-response relation. Where increased levels of expo-
sure correspond to increased severity or elevated risk, the
plausibility of a causal relation is strengthened. To date,
a fairly large number of studies have explored the relation
between various exposure levels and the periodontal health
condition. Taken together, these studies support the conten-
tion that periodontal disease morbidity increases with increasing exposure to smoking, i.e., the
greater the daily consumption and/or the longer the dura-
tion of the smoking habit, the greater the severity of the
disease. This has an important clinical and practical impli-
cation: if the patient’s smoking behavior can be modified
towards lower exposure levels, the periodontal tissues will
benefit. The results of the cohort studies, in particular those
that have evaluated the dose-response effects, offer con-
vincing evidence that the relation between smoking and
destructive periodontal disease is causal, i.e., smoking can
cause the disease.

**Smoking and risk for destructive periodontal disease**

Risk assessment is a powerful statistical means of estimat-
ing the potential influence of various population-based
factors on disease occurrence and severity and may be
applied to cross-sectional and case-control, as well as cohort
studies. The concept of risk was introduced into periodon-
tology several years ago and has now been applied in
several studies to estimate the risk associated with smoking.