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Effects of Smoking and Smoking Cessation and Smoking Cessation Intervention

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1. Introduction

The three- to four-decade lag between peak in smoking prevalence and subsequent peak in smoking-related mortality was a major factor affecting public awareness of the substantial health hazards of tobacco use in developed countries (Lopez et al., 1994). This factor may be applicable to periodontal disease if this disease is chronically affected by smoking epidemic. We searched the literature electronically and plotted the number of journal articles on association between smoking and periodontal disease with the trend in cigarette consumption (for example, in the USA) and expected trend in periodontal disease epidemic due to smoking by the year group (Fig. 1). Both peaks of expected trend of the disease and the number of journals stand closely in the 1990'.
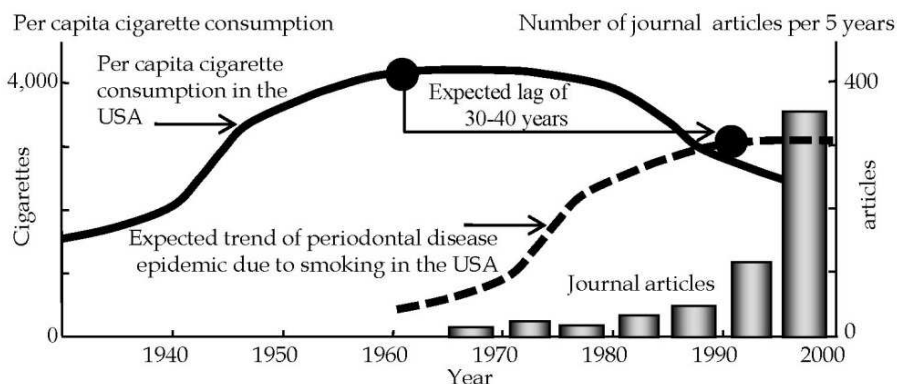

Fig. 1. Application of a descriptive model to the association of increase in smoking prevalence and smoking-related mortality with expected trends in smoking-attributable periodontal epidemic disease. The number of journal articles regarding smoking and periodontal disease followed the increase.

If this factor had been applied at an earlier stage in the series of periodontal research, practice of smoking cessation intervention in dental settings might have been more active.

The lag between the cigarette-smoking epidemic and epidemiological findings on the association of smoking with periodontal disease may have delayed public awareness of this association. Nevertheless, it is now well known that smoking is an independent risk factor of periodontal disease and influences the prognosis associated with periodontal treatments.

The validated association in the epidemiologic literature should be biologically plausible, since evidence supporting a causal association between smoking and periodontal disease has accumulated from clinical and basic studies over the past two decades. The underlying mechanism whereby smoking modulates components of the existing etiology of periodontal disease (Page & Kornman, 1997) has been largely clarified (Fig. 2). Though smokers are more susceptible to periodontal disease than non-smokers, bleeding on periodontal probing is less apparent in smokers than in non-smokers. The mechanisms underlying suppression of signs of clinical inflammation in smokers are under consideration for future studies.
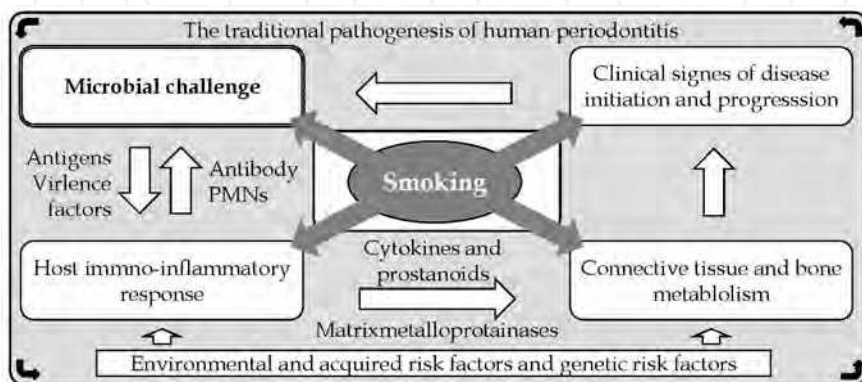

Fig. 2. Mechanisms by which smoking affects periodontal disease based on four components of the traditional pathogenesis of human periodontitis.

Smokers exhibit more periodontal tissue breakdown than non-smokers. These findings are based on the adjustment for confounding factors that are associated with periodontal disease and smoking. The underlying mechanisms include dysfunction of gingival fibroblasts, a decrease in microcirculatory function, and immune system deficiency. The more severe periodontal destruction in smokers than in non-smokers is attributable to impaired ability to repair damaged tissue rather than direct tissue damage.

Deeper understanding was provided by recent progress in molecular and genetic approaches (Ojima & Hanioka, 2010). Smokers exhibited overproduction of inflammatory molecules and suppression of anti-inflammatory molecules, thereby leading to inflammatory destruction of connective tissue and alveolar bone. Very recent studies using a novel method of bacterial identification revealed bacterial involvement in this process and provided an explanation of the connection between smoking and periodontal tissue breakdown in terms of pathogenic periodontal microorganisms.

The results of epidemiological and basic studies have led to periodontal disease now being considered a disease group in which there is sufficient evidence to infer its causal association with smoking. Special attention should be given to the treatment outcomes of periodontal disease in smokers. A negative response to periodontal treatment is consistently reported (Heasman et al., 2006). A more frequent recurrence of periodontal disease in

smokers than in non-smokers during periodontal maintenance was demonstrated (Carnevale et al., 2007). Evidence regarding the effects of smoking on periodontal disease and treatment indicates that smokers lose more tooth-supporting tissue than non-smokers. These effects lead to more rapid loss of tooth-supporting tissue in smokers than in non-smokers. An association between smoking and tooth loss during the periodontal maintenance period has recently been demonstrated (Chambrone et al., 2010). The number of journal articles on the association between smoking and tooth loss, as well as periodontal disease, has increased globally (Fig. 3), and evidence regarding the effect of smoking on tooth loss has accumulated. However, these reports are apparently limited to developed countries, possibly as a result of the lag between the smoking epidemic and occurrence of periodontal disease.
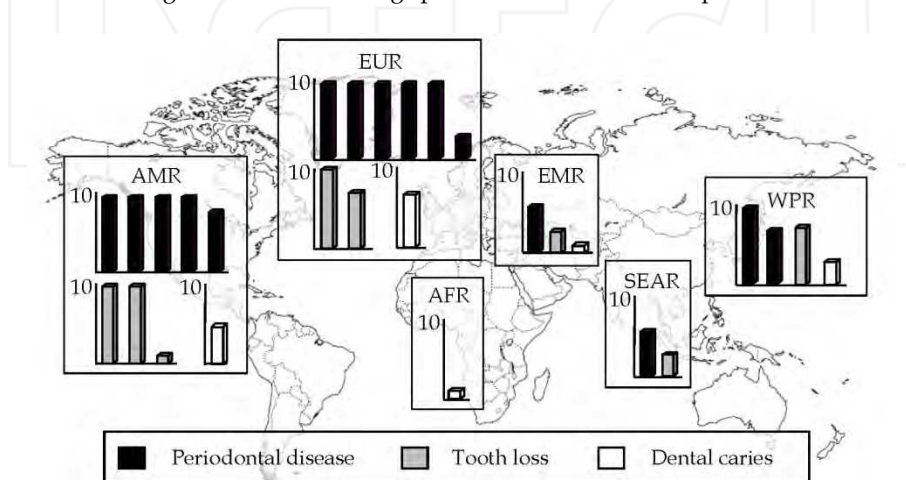

Fig. 3. Number of epidemiological articles addressing the association of smoking with periodontal disease, tooth loss, and dental caries in six WHO regions. The articles were extracted from MEDLINE in 2009 by searching for journal articles on periodontal disease, tooth loss, and dental caries by combining the key words "smoking" or "tobacco," and "periodontal disease" or "periodontitis," "tooth loss," and "dental caries," respectively.

A literature review of observational studies suggests that the evidence supporting a causal association between smoking and tooth loss is strong (Hanioka et al., 2011). Intervention for smoking cessation is an important practice not only for the prevention and treatment of periodontal disease but also for various important oral functions that may depend on the number of existing teeth. Several treatment modalities for tobacco dependence have been considered in the dental setting.

2. Epidemiological evidence

2.1 Periodontal disease and treatment

Effects of smoking and smoking cessation on periodontal disease and treatment responses were examined in observational studies. Data on the effects of adjunctive medications on treatment response in smokers were inconclusive. Benefits of smoking cessation in periodontal treatment were addressed recently.

2.1.1 Increased risk of periodontal disease due to smoking exposure

A comprehensive review in the Surgeon General's Report 2004 concluded that there is sufficient evidence to infer a causal relationship between smoking and periodontal disease (U.S. Department of Health and Human Services, 2004). In addition to studies in the review article, resent studies show a moderate to strong association (odds ratios ranging from 1.4 to 3.5, Warnakulasuriya et al., 2010). The effects on incidence and progression were also elucidated. Dose–response effects also demonstrated that heavy smokers had greater disease severity than light smokers in cross-sectional and cohort studies.

Representative populations were assessed in the USA, Japan, and Australia. The smoking-attributable fraction of periodontal disease ranged from 55.2% to 84% for current smokers and was 21.8% for former smokers. The population-attributable fraction ranged from 12.2% to 60% for current smokers and from 10.9% to 47% for former smokers (Tomar & Asma, 2000; Do et al., 2008). These variations may depend on different characteristics of the population, diversity of surrogate markers of periodontal disease, and confounding variables. An association has also been suggested in developing countries e.g., China, Thailand, and Brazil in terms of the cigarette-smoking epidemic. The consequences of the cigarette-smoking epidemic for oral health extend worldwide.

The effects of smoking on the young population are inconsistent. Smoking was significantly and independently associated with periodontal disease in the young population. A greater apparent association, with an odds ratio of 3.1, was shown in heavy smokers aged 14–29 years (Susin & Albandar, 2005) and in long-term smokers in a birth cohort study, which maintained a high follow-up rate (96%) and had high statistical power with a high incident odds ratio (5.2, Thomson et al., 2007). In contrast, association of smoking with periodontal diseases was not detected possibly due to lack of a sensitive marker such as attachment loss (Ojima et al., 2006).

Second-hand smoke inhalation potentiated bone loss during experimental periodontitis in rats. Data from the National Health and Nutrition Examination Survey (NHANES) III in the USA indicated that individuals exposed to second-hand smoke had greater odds (1.6 times) of having periodontal disease compared to individuals not exposed in the home and workplace (Arbes et al., 2001). Passive smokers, who were identified by salivary cotinine levels, showed a greater number of teeth with clinical attachment loss and higher levels of interleukin-1β, albumin, and aspartate aminotransferase in saliva than counterparts not exposed to passive smoke (Yamamoto et al., 2005). These findings were further confirmed by the research for dose-response relationship (Sanders et al., 2011). Smokeless tobacco users exhibited gingival recession and periodontal disease in the USA, Thailand, Bangladesh, and Sweden.

2.1.2 Decreased risk of periodontal disease due to smoking cessation

Decreased risk of periodontal disease due to smoking cessation is less clearly established than increased risk due to smoking exposure. Some studies suggest periodontal disease severity in former smokers falls between that of current and non-smokers. Very few studies demonstrate a dose–response relationship between risk reduction of periodontal disease and smoking cessation. Findings of the NHANES III revealed that the odds of periodontitis for former smokers who quit ≥11 years previously were indistinguishable from the odds for non-smokers (Tomar & Asma, 2000). In a study of senior employees and retired personnel of the electricity generating authority in Thailand, for light smokers, the odds for severe periodontitis reverted to the level of non-smokers when they had quit smoking for ≥10

years, and for moderate heavy smokers, the odds of having severe periodontitis did not differ from those of non-smokers when they had quit smoking for ≥20 years (Torrungruang et al., 2005).

2.1.3 Effects of smoking on treatment response

The effects of smoking on the response to periodontal treatment have been extensively reviewed (Heasman et al., 2006). A negative effect of smoking on the outcome of several periodontal treatment modalities has been demonstrated in recent studies, and the width of keratinized gingiva for gingival recession therapy, radiographic bone defect, subgingival microbial changes, inflammatory markers, and gingival blood flow in addition to the pocket probing depth, clinical attachment level, and bleeding on probing are used to examine treatment outcome. No significant difference was detected in the 10-year periodontal stability in recession defects of patients receiving guided tissue regeneration therapy and an immediate effect of instrumentation on the subgingival microflora between smokers and non-smokers. Smokers more frequently experienced a recurrence of periodontal disease than non-smokers during supportive periodontal therapy. Tooth loss is a tangible outcome of periodontal treatment and also reflects the recurrence of periodontal disease.

2.1.4 Effects of adjunctive medications on treatment response in smokers

Clinicians are required to use adjunct antimicrobial or host-modulation therapy for smokers. Adjunctive local medications were effective in reducing *Porphyromonas gingivalis*, the attachment level gain reduced with doxycycline, and red or orange-complex bacteria in current smokers and C-reactive protein concentration improved with minocycline. The effects of adjunctive systemic medications, however, are inconclusive. Low-dose doxycycline administration was shown to be effective on analysis of a smoking subgroup (Preshaw et al., 2005a), while no additional benefit was shown in smokers when a stricter analytical method with a multilevel model was used (Needleman et al., 2007). Adjunctive administration did not show an additional benefit compared to non-surgical treatment for azithromycin and surgical treatment for flurbiprofen, while adjunctive azithromycin administration adjunct to scaling and root planing contributed to treatment outcomes in smokers. These findings suggest inconclusive effects of adjunctive medications for smokers, indicating the importance of emphasizing the benefit of smoking cessation.

2.1.5 Benefits of smoking cessation in periodontal treatment

Observational studies comparing periodontal health between current, former, and non-smokers after periodontal treatment suggested that quitting smoking is beneficial to patients with periodontal diseases. Some studies showed that responses to treatment in ex-smokers were similar to those in people who had never smoked. However, there are limited data from long-term longitudinal clinical trials to demonstrate unequivocally the periodontal benefit of smoking cessation. An intervention study investigated longitudinally (12 months) the effect of quitting smoking on periodontal status when combined with non-surgical periodontal therapy in smokers with chronic periodontitis (Preshaw et al., 2005b). A new culture-independent assay for bacterial profiling quantifies the effect on subgingival pathogens. This method revealed an effect on subgingival microbial recolonization after smoking cessation.

Theoretical modeling of the cost-effectiveness of smoking cessation was described. The model revealed that a 10% increase in the number of cigarettes smoked per day increased the treatment costs of periodontal diseases by 0.7% and 0.2% for men and women, respectively (Sintonen & Tuominen, 1989). Adding smoking cessation to the concept of periodontitis prevention will enable significant cost savings to be made.

2.2 Tooth loss

Ten cross-sectional and five prospective cohort studies regarding smoking and tooth loss were selected for the evaluation of methodological quality among 496 citations obtained by a literature search and screening the database. Methodological quality of studies was assessed using a standardized scale; eight studies (six for cross-sectional and two for prospective cohort studies) were classified as high quality.

Three elements—the strength of association, experiment, and the dose–response relationship—were assessed in terms of consistency to allow the synthesis of evidence for each element. The evidence of association was evaluated for each element with respect to consistency through studies that examined 58,755 subjects in four countries; Germany, Italy, Japan, and the USA (Fig. 4). The association between current smoking and tooth loss was significant in all studies. The effect size in cross-sectional studies (odds ratio) varied from 1.69 to 4.04 and that in cohort studies (hazard ratio) was 2.1 and 2.3.
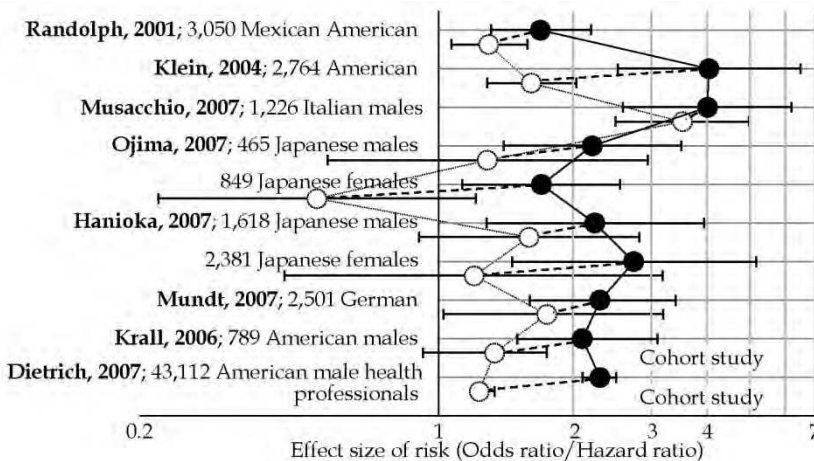

Fig. 4. Effect size (95% confidence interval) of risk of tooth loss in current (closed circles) and former smokers (open circles) relative to non-smokers.

The element of 'experiment' was evaluated by comparing the strength of association between former and current smokers relative to non-smokers, because interventional studies are difficult to conduct in humans. This surrogate element was named "natural experiment." The association between former smoking and tooth loss was not significant in four studies. Although another four studies reported a significant association, the effect size was consistently smaller for former smokers than for current smokers in all studies. The evidence from natural experiments for evaluating the association between smoking cessation and tooth loss was strong with respect to consistency. Two cohort studies with

observational periods of 16 and 36 years on populations in the USA reported decreases in hazard ratios on the basis of years of abstinence (data not shown).
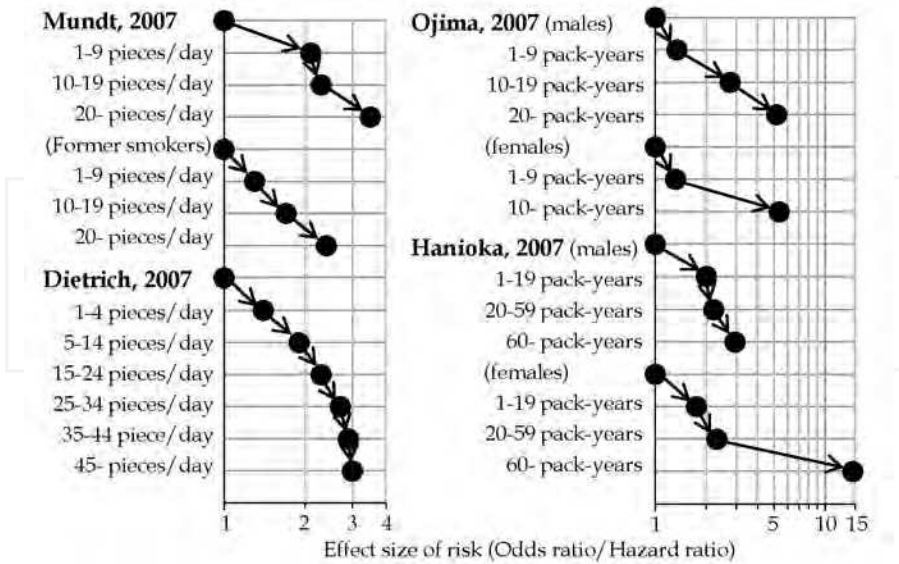

Fig. 5. Relationship between dose of exposure to smoking and effect size.

The dose–response relationship was reported in four high-quality studies, including one cohort study (Fig. 5). These studies examined 50,926 subjects in three countries; Germany, Japan, and the USA. One study in Germany examined the relationship in former smokers. The trend of the relationship between the level of exposure and effect size, i.e., odds ratio or hazard ratio, was obvious in all studies. Therefore, the evidence for a dose–response relationship between smoking and tooth loss was also strong with respect to consistency.

The results from the assessment of each element suggested that the evidence was strong in terms of consistency. This interpretation was based on consistent results with little or no evidence to the contrary in six cross-sectional and two prospective cohort studies. The inclusion of cohort studies indicates more convincing evidence for a causal association. Based on the consistent evidence from each element in the evaluation of this causal association with existing biological plausibility, the evidence supporting a causal association between smoking and tooth loss appears to be strong.

3. Biological plausibility

3.1 Molecular and genetic aspects
3.1.1 Microflora
The effect of smoking on the severity of periodontal disease with respect to the prevalence of specific periodontal pathogens is a controversial issue: some studies have shown differences in the microbial flora between smokers and non-smokers, but several other studies have not been able to demonstrate relevant differences. Differences in periodontal pathogen detection techniques, specimen sampling, and disease definition may explain

these conflicting findings. DNA-based techniques have been employed for the detection of specific periodontal pathogens. The polymerase chain reaction (PCR) is a more sensitive and specific method for the detection of bacteria than conventional culture-based methods.

A series of recent studies (Preshaw et al., 2005b; Delima et al., 2010; Shchipkova et al., 2010) revealed a bacteriological mechanism by using a novel method for bacterial identification. The microbial profile of disease-associated and health-compatible organisms in smoking-associated periodontitis patients was significantly different from that in non-smokers. After non-surgical periodontal therapy and smoking cessation counseling, those who continued smoking had a microbial profile similar to that at baseline, while the subgingival microbiome in those who stopped smoking exhibited a healthy profile. These findings explain the connection between smoking and periodontal tissue breakdown by pathogenic periodontal microorganisms.

Another series of studies (Bagaitkar et al., 2009, 2010; Budneli et al., 2011) addressed the involvement of anaerobic bacterial periodontopathogens in the mechanism of suppression of the clinical inflammatory response in periodontal disease in smokers. As an environmental factor, the stress of cigarette smoke upregulates *P. gingivalis* fimbrial antigens and creates conditions that promote biofilm formation, though the proinflammatory response to the pathogen is inhibited. An reduced inflammatory response potential of oral microflora was indicated by alteration of fatty acid profiles in the saliva of smokers with chronic periodontitis.

3.1.2 Smoking-associated pathophysiological changes

Destructive effects of smoking on periodontal tissue are categorized with respect to vascular, immune, and inflammatory responses (Fig. 6). Smoking modulates the destruction of periodontal tissue through various responses; adverse vascular changes and suppression of host immune systems, and disorder of inflammation (Ojima & Hanioka, 2010).

Repeated vasoconstrictive attacks and impairment of revascularization due to cigarette smoking can influence immune function and the subsequent inflammatory reaction in the gingiva. In the inflamed gingival tissues of smokers, significantly fewer vessels were observed compared to non-smokers. Microcirculatory changes may be related to impairment of oxygen delivery to gingival tissue. Gingival blood flow increased after quitting smoking. Expression of intercellular adhesion molecule-1 (ICAM-1), a marker of endothelial dysfunction leading to damaging vascular disorders, was higher in smokers than in age-matched non-smoking controls. These vascular alterations due to cigarette smoking may contribute to disruption of the immune response and delay in the healing response.

Smoking may depress host immune responses, although there are some conflicting results. The number of neutrophils in gingival crevicular fluid (GCF) was lower or remained constant in smokers compared to non-smokers, while that in blood was higher in a dose-dependent manner. Adverse effects of smoking on the function of polymorphonuclear neutrophils, e.g., reduced viability and phagocytosis, were observed in periodontally healthy smokers. Smoking may influence lymphocyte numbers and antibody production. The serum level of Immunoglobulin G_2 (IgG_2), which was an important antibody against gram-negative periodontal pathogens, decreased in patients with periodontitis. Smoking may decrease the proliferative capacity of T cells or T-cell-dependent antibody responses that affect B-cell function and antibody generation.
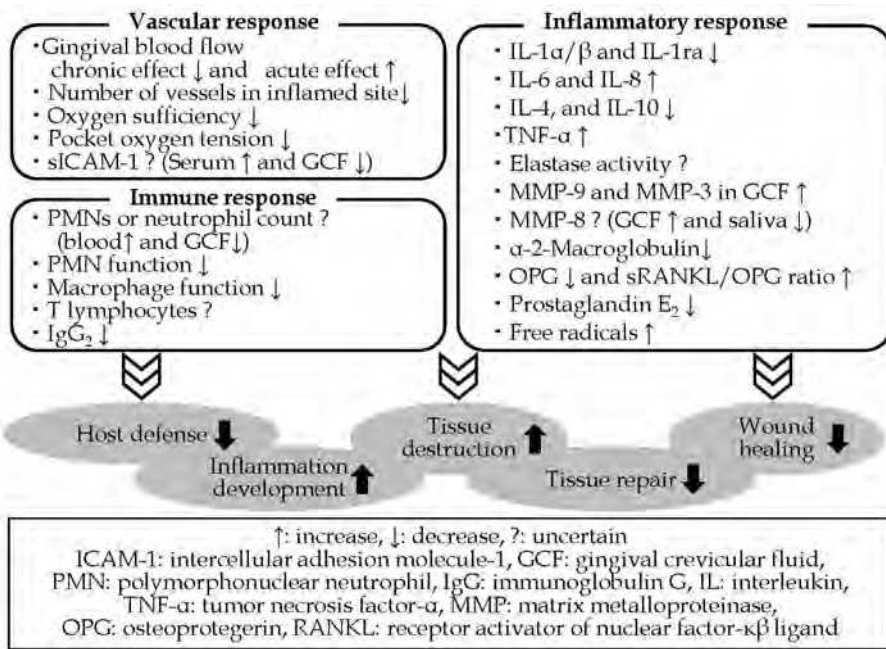
Fig. 6. Destructive effects of smoking on periodontal tissue.

Among several cytokines associated with periodontal disease, levels of interleukin (IL)-1 in GCF have been extensively compared between smokers and non-smokers. Smokers exhibited significantly lower concentrations of IL-1a and IL-1ra in GCF than nonsmokers. Smokers tend to exhibit excess production of inflammatory molecules, such as IL-6, IL-8, and tumor necrosis factor-α, and suppression of anti-inflammatory molecules, such as IL-4, IL-10, and IL-1ra; however, these findings are to some extent inconsistent. Findings regarding the effects of smoking on the level of neutrophil-derived proteolytic enzymes in oral specimens are inconsistent; however, smoking may increase their level in the systemic circulation.

Matrix metalloproteinase-9 (MMP-9) in plasma was higher in smokers than in non-smokers. Smokers had the higher level of elastase and MMP-3 in GCF, and MMP-8 expression in periodontal tissue than non-smokers, while the salivary MMP-8 level was significantly lower in current smokers than in former smokers. Smokers showed a significantly lower concentration of α-2-macroglobulin in GCF as well as total amounts of α-2- macroglobulin and α-1-antitrypsin than non-smokers. Smoking seems to disturb the balance between proteolytic and anti-proteolytic activities in periodontal tissue.

IL-1, IL-6, and TNF-α stimulated the expression of the receptor activator of nuclear factor-κβ ligand (RANKL) and the inhibitor protein osteoprotegerin (OPG), which are dominant regulators of bone resorption and remodeling. The OPG concentration was significantly lower and the sRANKL/OPG ratio was higher in smokers compared with non-smokers, in saliva as well as serum, explaining the greater potential for alveolar bone loss in smokers.

IL-1 and IL-6 induce production of prostaglandin E_2 (PGE_2) by neutrophils and macrophages, which could also accelerate alveolar bone resorption, although the level of PGE_2 in GCF and saliva in smokers was similar to that in non-smokers or even lower than that in non-smokers. The level of free oxygen radicals in periodontal tissues, which induces tissue damage by injuring cells such as fibroblasts, was higher in smokers than in non-smokers. Impairment of fibroblasts by smoking possibly leads to delay in tissue repair and wound healing in periodontal disease.

Most findings support the idea that smokers exhibit a greater burden of inflammatory responses to microbial challenges compared to non-smokers. However, limited evidence is available regarding the effects of quitting smoking on pathophysiological changes in periodontal tissue.

3.1.3 Gene-smoking relationship

Relationships between smoking and genetic susceptibility to periodontal diseases have been investigated with respect to genotypes associated with cytokines (IL-1, IL-6, and IL-10), the immune system (Fcγ receptor), bone metabolism (vitamin D receptor), and xenobiotics metabolism (N-acetyltransferase and myeloperoxidase).

IL-1 polymorphisms have been intensively studied using a cross-sectional design, except for one longitudinal study. Its relationship with respect to smoking is controversial. Several studies reported relationships between IL-1-positive genotypes and smoking; however, other studies demonstrated that the association of IL-1-positive genotypes with the severity of periodontal disease was independent of smoking, suggesting no relationship between smoking and IL-1 genotypes. Logistic regression analysis revealed that odds ratios of periodontal disease, in comparison with IL-1 genotype-negative non-smokers as a reference group, was 0.98 for genotype-positive non-smokers, 2.37 for genotype-negative smokers, and 4.50 for genotype-positive smokers, suggesting synergism between IL-1 polymorphism and smoking (Meisel et al., 2004).

An association between IL-6 and IL-10 genotype and periodontal status was more conspicuous in non-smokers. Fcγ receptors are important components in the binding and phagocytosis of IgG-sensitized cells. Genotypes for Fcγ receptor, FcγRIIa, and FcγRIIIb may be associated with periodontal disease in smokers (Yamamoto et al., 2004). Gene polymorphisms for enzymes that can metabolize smoking-derived substances may contribute to individual susceptibility to the risk of periodontitis among smokers. Subjects with the gene polymorphism for enzymes that can metabolize smoking-derived substances, e.g., cytochrome P450 1A1 M2 allele and the glutathione S-transferase M1 allele, exhibited an increased risk of periodontitis.

To date, gene-smoking relationships in periodontal disease are uncertain because of methodological limitations such as employment of subjects in a specific race, small sample size, and lack of detailed history of smoking and possible confounders. The gene-smoking relationships in periodontal disease may be bilateral; genetic susceptibility to periodontal disease is influenced by exposure to smoking, or the effect of smoking on periodontal disease is influenced by genetic susceptibility. Better understanding of gene–smoking relationship could contribute to the prevention of periodontal disease through personalized recommendation and targeted intervention in public and clinical dental programs.

4. Intervention of smoking cessation

4.1 Dental setting

Smoking cessation intervention is an important category in the dental practice. Smoking cessation intervention is performed in dental setting for a variety of purposes according to the oral condition of patients. Smoking cessation is effective in preventing not only oral diseases but also the progression of periodontal tissue breakdown. Smoking cessation intervention may be integrated in existing procedures of dental treatment because improvement of outcome of the treatment is expected by smoking cessation.

Periodontal practitioners should know the "5 A's" model for treating smoking and nicotine dependence (Fiore et al., 2008a). This model consists of five components for effective smoking cessation intervention: **Ask** about tobacco use; **Advise** about quitting; **Assess** willingness to make a quit attempt; **Assist** in the quit attempt; and **Arrange** follow-up. Although full implementation of the "5 A's" in clinical settings is superior to partial implementation, periodontal practitioners may be responsible for some parts of these components.

Several modalities of smoking cessation intervention have been proposed in the dental setting. The effectiveness of intervention modalities was examined with respect to the success rate of quitting. Since there are several pathways in both the clinical and social setting for smoking cessation, dental practitioners need to know about these pathways to assist patients routinely to choose an appropriate way to succeed in quitting in addition to improving the outcome of dental treatment specific to the patient.

Motivational interview strategies (Fiore et al., 2008a) such as "express empathy," "develop discrepancy," "roll with resistance," and "support self-efficacy" are specialized techniques. Dental hygienists may be able to accept these techniques because they routinely motivate dental patients about oral health behavior on the basis of these techniques. Another strategy that enhances future attempts to quit smoking is the "5 R's" (Table 1).

Relevance	Encourage the patient to indicate why quitting is **personally relevant**, being as **specific** as possible. Motivational information has the greatest impact if it is relevant to a patient's disease status or risk, family or social situation, health concerns, age, gender, and other important patient characteristics.
Risks	The clinician should ask the patient to identify potential negative consequences of tobacco use. The clinician may suggest and highlight those that seem **most relevant to the patient**.
Rewards	The clinician should ask the patient to **identify potential benefits** of stopping tobacco use. The clinician may suggest and highlight those that seem most relevant to the patient.
Roadblocks	The clinician should ask the patient to identify barriers or impediments to quitting and provide treatment that could address barriers.
Repetition	The motivational intervention should be **repeated every time** an unmotivated patient visits the clinic setting. Tobacco users who have failed in previous quit attempts should be told that most people make repeated quit attempts before they are successful.

Table 1. Motivational strategies to enhance attempts to quit smoking; the "5 R's."

The "5R's" strategy is available to dental practitioners. Particularly, four components of "relevance," "risks," "rewards," and "repetition" in the motivational strategies include some issues specific to dental practice. Various oral symptoms and dental treatments relevant to

smoking may be used to motivate dental patients. For example, periodontal patients need to know the risk posed by continuing smoking for the development of periodontal tissue breakdown, because these patients are susceptible to smoking-associated periodontal disease. In other words, the issue of smoking and periodontal disease is personally relevant. Therefore, smoking cessation is recommended for the substantial benefit on the outcome of periodontal treatment. Periodontal practitioners can repeat motivational interventions when unmotivated patients visit for periodontal treatments. Periodontal practitioners need to acquire new knowledge about only one technique among the motivational strategies– roadblocks.

The level of willingness to quit smoking varies among dental patients. Dental practitioners need to know the stages of behavior change (Prochaska et al., 1992) and approaches that can be used to promote progress through the stages of behavior change. The theoretical model with behavioral approaches involves stage-based interventions. This model categorizes smokers into five different stages; precontemplation, contemplation, preparation, action, and maintenance.

The effectiveness of brief interventions by dental professionals using the feedback of oral symptoms and dental treatments personally relevant to smoking was examined with respect to quitting smoking and motivation for smoking cessation (Hanioka et al., 2007). Levels of changes in smoking behavior and cessation attempts were assessed using a standardized questionnaire. The questionnaires used at the first and final visits were analyzed for movement through the stages of behavior change. Experience with respect to quit attempts during the dental visits was surveyed in the questionnaire at the final visit. The intervention consisted of a brief explanation regarding dental events relevant to smoking, employing color charts. Patients in the non-intervention group received no intervention other than dental treatments.

The percentages of patients who attempted to quit among those who were not ready to quit were 9.1% and 3.3% in the intervention and non-intervention groups, respectively (Fig. 7). The percentages of patients who progressed through the stages were 22.6% and 17.7%, and the percentages of those who regressed through the stages were 7.7% and 15.8%, respectively. The differences between groups were all significant. The effects were not significant in patients who were ready to quit within 1 month (data not shown). However, the percentage of patients willing to quit was less than 10% (Fig. 8). Dental visits provide an important opportunity for health professionals to influence smokers with respect to motivation for smoking cessation.
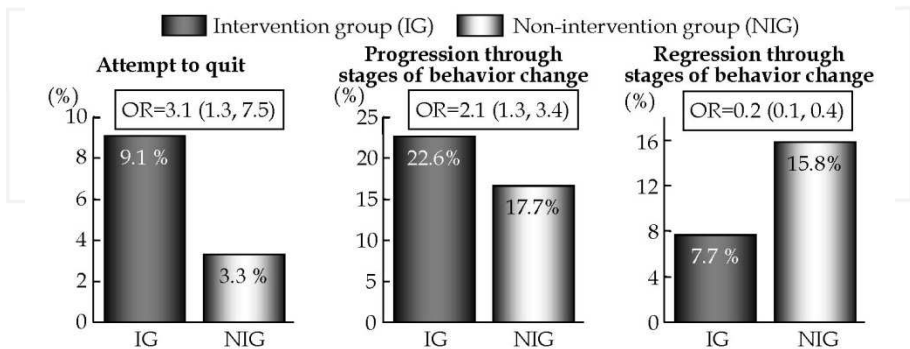

Fig. 7. Effects of a brief intervention using the dental strategy "5 R's" in patients who were not ready to quit.
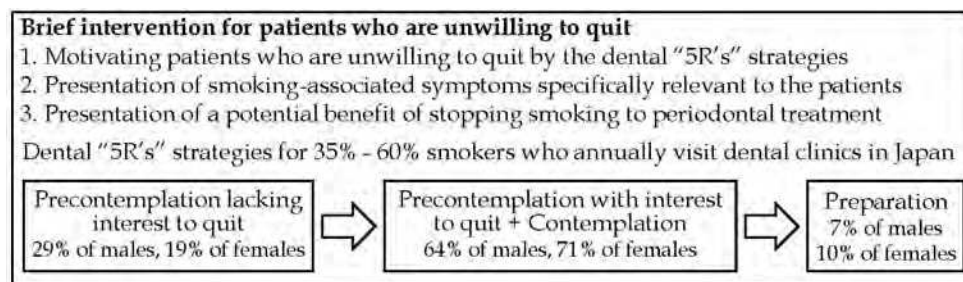
Fig. 8. Intervention for patients unwilling to quit, and distribution of patients by stage of behavior change

Dental practitioners have the opportunity, by routine assessment, to find out whether the patient plans to attempt to quit during the motivational intervention or for another reason. Several strategies are available for patients willing to quit (Fig. 9). The dentist can assist the patient by offering medication and providing or referring for counseling or additional treatment, and arrange for follow-up contacts to prevent relapse (Fiore et al., 2008a). The success rate of smoking cessation differs among the different strategies. The cost and availability of each strategy and approved medication may be suited to the personality of the patient. Therefore, the provision of information about effective smoking cessation aids is an essential component of intervention for patients who are willing to quit.
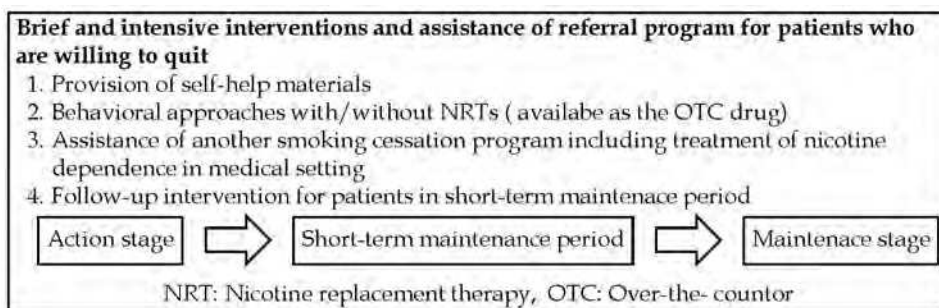

Fig. 9. Intervention and assistance strategies for patients willing to quit.

A feasibility study was conducted to evaluate the potential effectiveness of an intensive smoking cessation intervention delivered by dental professionals with the outcome measure of abstinence rate (Hanioka et al., 2010). Patients who were willing to quit smoking were randomly assigned to either an intervention or a non-intervention group. Intensive intervention was provided, consisting of five counseling sessions, including an additional nicotine replacement regimen. Reported abstinence was verified by measuring the salivary cotinine level. On an intent-to-treat basis, 3-, 6-, and 12-month continuous abstinence rates in the intervention group were 51.5%, 39.4%, and 36.4%, respectively, while the rates in the non-intervention group were consistent at 13.0% (Fig. 10). Adjusted odds ratios (95% confidence interval) by logistic stepwise regression analyses were 7.1 (1.8, 28.5), 8.9 (1.7, 47.2), and 6.4 (1.3, 30.7), respectively.

Intensive smoking cessation intervention is effective in the dental setting. An intensive smoking cessation intervention conducted by dental hygienists was also successful (Binnie et al., 2007). Nicotine replacement therapy is, however, not allowed because of the limitation of the dental medication list (Action on Smoking and Health, 2008). The pharmaceutical approach may reduce withdrawal symptoms, which may include oral symptoms such as mouth ulcers, the prevalence of which is 40% (McEwen et al., 2006). Dentists are ideally placed to promote cessation because they are able explain the impact of tobacco on oral health to their patients, many of whom consider themselves to be perfectly healthy. The WHO oral health program has strengthened its support of countries that incorporate oral health into tobacco control (Petersen, 2003), and the FDI World Dental Federation has urged dental professionals to advise patients to quit smoking (FDI World Dental Federation, 2004). Referral programs for intensive intervention for smoking cessation are also available.
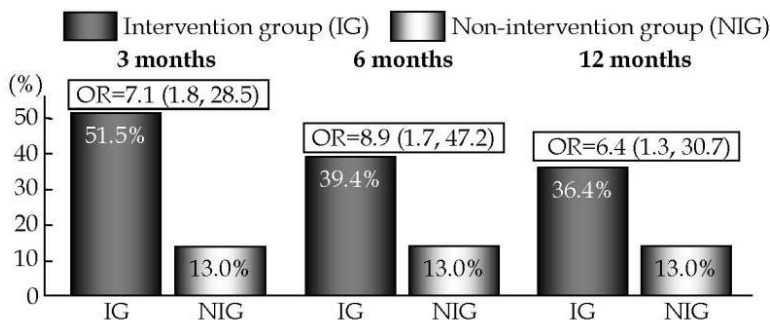

Fig. 10. Effects of intensive smoking cessation intervention in terms of abstinence rate in a feasibility study in a dental setting.

4.2 Referral services for intensive intervention

Intensive smoking cessation treatment is more effective than brief intervention. There is a strong dose–response relationship between counseling intensity and quitting success (Fiore et al, 2008b). Treatments may be made more intense by increasing the duration and number of individual treatment sessions. Many different types of providers (e.g., physicians, nurses, dentists, psychologists, and pharmacists) play substantial roles in increasing quit rates, and involving multiple providers can increase abstinence rates. Individual, group, and telephone counseling are effective formats for treatment of chronic tobacco use. Particular types of counseling strategies, like practical counseling (problem solving/skills training approaches) and the provision of intratreatment social support (emotional support during treatment) by providers, are especially effective and associated with significant increases in abstinence rates (Fiore et al, 2008b). In addition, pharmacological treatment with nicotine replacement therapy (NRT), bupropion, and varenicline consistently increase abstinence rates (Fiore et al, 2008a). A combination of counseling and pharmacotherapy also increases abstinence rates to a great extent.

Two major programs have proven to be effective referral services for smokers willing to quit, and are thus highly recommended(WHO report on the global tobacco epidemic, 2011):

1.   free telephone help lines (known as quitlines)
2.   treatment services with pharmacotherapy

4.2.1 Quitlines

Quitlines are telephone-based cessation support services and have been established in many countries since the late 1980s. In proactive quitlines, the call is initiated by the counselor, while reactive quitlines only respond to incoming calls. Services range from a single brief reactive counseling session, provided at the time a caller reaches the quitline, to intensive counseling via multiple proactive follow-up calls initiated by the counselor in addition to with self-help materials, web-based services, or pharmacotherapy for smoking cessation provided to the caller (McAfee, 2007; Centers for Disease Control and Prevention, 2004). The advent of quitlines indicates that intensive, specialist-delivered interventions are now available to smokers on an unprecedented basis (Fiore et al., 2008b).

Evidence regarding the effectiveness of proactive quitlines is well established, with a recent meta-analysis of randomized control trials (RCTs) demonstrating a higher likelihood of abstinence after 6 months or more of follow-up (risk ratio = 1.37; 95% confidence interval [CI], 1.26–1.50), but the evidence for reactive quitlines is not convincing (Stead, 2009). Adding telephone support to brief intervention or pharmacotherapy increases long-term abstinence rates when compared with brief intervention alone or pharmacotherapy alone (Stead, 2009). There is some evidence of a dose response; 1 or 2 brief calls are less likely to provide a measurable benefit when compared with a longer intervention. Three or more calls increase the chances of quitting when compared with a minimal intervention such as providing standard self-help materials or brief advice, or when compared with pharmacotherapy alone (Stead, 2009).

In addition to their effectiveness, quitlines have other advantages (Centers for Disease Control and Prevention, 2004): 1) easy access (quitlines reduce barriers in accessing traditional cessation services, including time, transportation difficulties, childcare responsibilities, financial costs, and the psychological barrier); 2) the benefits of centralization (quitlines can serve a large geographic area from a single, centralized base of operations, which leads to economy of scale, ease of promotion, better quality control, and ease of evaluation); and 3) acting as the hub of a network of cessation resources (quitlines serve not only as direct service providers but also as the hub of a comprehensive network of cessation resources in a community, and the coordinating function of the hub can include referral to appropriate resources for pharmacotherapy and intensive cessation programs in medical settings). These advantages increase the population impact of quitlines not only by providing effective counseling services, but also by enhancing the use of other available cessation resources. The U.S. Public Health Service-sponsored Clinical Practice Guideline and the Guide to Community Preventive Services recommend proactive quitlines as a way to help smokers quit (Fiore et al., 2008b; Task Force on Community Preventive Services, 2005).

Many clinicians find it hard to deliver all of the recommended 5As (ask, advise, assess, assist, and arrange) in a busy practice setting. One way of improving the efficiency of smoking cessation treatment delivered in primary care settings may be to give the physician a defined, but limited, role in delivery of the 5As (Bentz, 2006). This could be accomplished by incorporating quitlines.

QuitWorks is a free smoking cessation service developed by the Massachusetts Department of Public Health in collaboration with all major health plans in Massachusetts (Centers for Disease Control and Prevention, 2004; Massachusetts Department of Public Health). Nearly 22,000 patients have been referred to the QuitWorks program since its launch in April 2002. QuitWorks links healthcare providers and their patients who smoke to proactive telephone

counseling and other smoking cessation services. Any physician, nurse, dentist, or other clinician can easily and quickly refer any smoking patient irrespective of health insurance status. The referral forms are faxed or electronically transmitted to the state-funded quitline service provider. Upon referral, the quitline counselor provides free multisession proactive counseling, internet counseling, and referral to community-based treatment programs. Every referring provider will receive reports about their patients. Within 1 month, a patient contact report is sent to confirm contact with the patient and the services accepted. About 6 months after the initial assessment, QuitWorks calls the patient to assess his or her smoking status and sends the provider a patient outcome report.

New Zealand's health system uses an ABC approach instead of the 5As model. ABC is a simple and easy tool for the guidance of all healthcare workers: Ask about smoking status, give Brief advice, and offer Cessation support, which implies face-to-face support with pharmacotherapy or referral to smoking cessation services, including quitlines (McRobbie, 2008). The national quitline assists more than 50,000 New Zealanders attempting to quit smoking each year. To promote smoking advice and assistance in healthcare settings, the Government introduced a health target of "better help for smokers to quit" as 1 of only 6 governmental priority health targets, with the ultimate goal being that 95% smokers who are admitted to a hospital should receive advice and assistance for quitting by July 2012 (WHO Framework Convention on Tobacco Control, 2011). Because of the success of this approach, it will be extended to primary healthcare services as well.

4.2.2 Treatment services with pharmacotherapy

In November 2010, WHO issued detailed guidelines for implementation of Article 14 of the Framework Convention on Tobacco Control (demand reduction measures with regard to tobacco dependence and cessation). These guidelines intend to encourage each government to strengthen or create a sustainable infrastructure to motivate quit attempts and to ensure wide access to smoking cessation treatment (WHO Framework Convention on Tobacco Control, 2011). The costs of smoking cessation treatment can be covered or reimbursed by public health services to reduce out-of-pocket expenses for people trying to quit. Eighty per cent high-income and 40% middle-income countries provide at least some cost coverage for smoking cessation treatment, including pharmacotherapy, while only 1 in 8 currently covers any costs of cessation services in low-income countries (WHO report on the global tobacco epidemic, 2011). Pharmacotherapy is generally more expensive and less cost-effective when compared with cessation advice in healthcare settings and quitlines; however, it has been shown to double or triple quit rates (Fiore et al., 2008a). NRT is usually available over the counter, whereas bupropion and varenicline require a doctor's prescription (WHO report on the global tobacco epidemic, 2009). NRT reduces withdrawal symptoms by replacing some of the nicotine absorbed from tobacco. Bupropion, an antidepressant, can reduce craving and other negative sensations when tobacco users stop their nicotine intake. Varenicline, a selective α4β2 nicotinic acetylcholine-receptor partial agonist developed specifically for smoking cessation, relieves nicotine cravings and withdrawal effects while reducing the reinforcing effects of nicotine through its partial agonistic mechanism of action.

The UK offers the world's most comprehensive support for smokers wishing to quit (WHO report on the global tobacco epidemic, 2009). A national smoking cessation treatment service is universally available to all smokers, mainly free of charge, through the National Health Service (NHS). This service offers weekly support for at least the first 4 weeks of a quit

attempt, with face-to-face intensive counseling and pharmacotherapy (McNeil, 2005). Typically, smokers are seen by smoking cessation counselors 1 week (maximum 2 weeks) before quitting and at weekly intervals for 4 weeks after quitting. Pharmacotherapy typically continues at weekly intervals for 8 weeks (Judge, 2005). Over 700,000 smokers per year (approximately 6%–7% of all smokers) set a quit date through the service and 49% smokers who set a quit date had successfully quit by self-report at the 4-week follow-up (NHS Health and Social Care Information Centre, 2010). Sixty-nine per cent smokers who successfully quit at the 4-week follow-up had their results validated with a carbon monoxide test. Most smokers who set a quit date received NRT (65%), whereas 23% received varenicline. The 1-year carbon monoxide-validated abstinence rate was 14.6%, rising to 17.7% when self-reported quitters were included (Ferguson, 2005).

In Japan, a smoking cessation treatment service for outpatients at registered medical institutions was started under public health insurance coverage in 2006. The reimbursed treatment program comprises 5 treatment sessions over a period of 12 weeks. Nicotine patches or varenicline can be prescribed under health insurance coverage during the treatment period. The number of registered medical institutions is increasing year by year. More than 12,800 institutions have now been registered. Access to treatment is improving but is still not satisfactory because the percentage of registered institutions is only 10 % among total medical institutions, limited to hospitals 20%.

According to the 2007 and 2009 surveys conducted by the Review Committee of the Central Social Insurance Medical Council of Japan (The Central Social Insurance Medical Council, 2008 and 2010), the self-reported continuous abstinence rate at randomly selected registered institutions was 32.6% (2007) and 29.7% (2009) after 9 months of completing smoking cessation treatment. If only those patients who received all 5 treatment sessions were considered, the rate was 45.7% (2007) and 49.1% (2009), respectively. These results indicate that smoking cessation treatment is functioning well.

The intervention factors related to the effectiveness of treatment services in the UK and Japan were examined using a large data sample of smokers using these services after adjusting for smoker characteristics (Brose, 2011; The Central Social Insurance Medical Council, 2010). In the UK, NRT alone was associated with higher success rates than no pharmacotherapy (Odds ratio [OR], 1.75; 95% CI, 1.39–2.22), whereas a combination of NRT and varenicline were more effective than NRT alone (OR, 1.42; 95% CI, 1.06–1.91 and OR, 1.78; 95% CI, 1.57–2.02). In addition, higher success rates were associated with group support than with one-to-one support (OR, 1.43; 95% CI, 1.16–1.76), and primary care settings were less successful than specialist clinics (OR, 0.80; 95% CI, 0.66–0.99). In Japan, a higher number of treatment sessions was associated with a higher success rate (OR, 1.78; $p < 0.05$). In addition, varenicline was more effective than NRT (OR, 1.24; $p < 0.05$), re-treatment was less successful than the initial treatment (OR, 0.68; $p < 0.05$), and physicians with greater experience in smoking cessation treatments were associated with higher success rates (OR, 1.03; $p < 0.05$). These findings using routine clinic data support those from RCTs.

To improve the quality of treatment services, England has established the NHS Centre for Smoking Cessation and Training for practitioners involved in smoking cessation activities. It offers certification to practitioners through its online training, which develops evidence-based competences (knowledge and skills) for treatment services. In Japan, the Japan Medical and Dental Association for Tobacco Control has developed e-learning programs to train healthcare providers, who can then administer reimbursed smoking cessation treatments as well as proactive brief intervention in routine healthcare settings.

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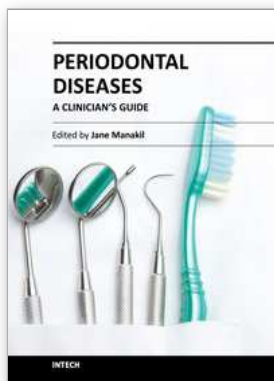
Periodontal Diseases - A Clinician's Guide
Edited by Dr. Jane Manakil

"Periodontal diseases" is a web-based resource intended to reach the contemporary practitioners as well as educators and students in the field of periodontology. It is fully searchable and designed to enhance the learning experience. Within the book a description is presented of the current concepts presenting the complex interactions of microbial fingerprint, multiple genotypes, and host modulations. In addition, an overview is given of the clinical outcome of the disease's progression, as influenced by the epigenetic factors. Emerging concepts on periodontitis as a risk factor for various systemic diseases and as a bilateral modulating factor have been elucidated in detail as well.

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