Introduction

Smoking is a habit that is extremely damaging to health and that continues to be widespread throughout society. It is currently the leading preventable cause of premature death in the world, and it is estimated that in 2020 it will be directly related to some 10 million annual deaths, mainly in developing countries (1). According to the most recent INE (Spanish National Institute of Statistics) 2011-12 survey data, approximately 1 in every 3 adults in Spain is a regular smoker.

The mechanism of action of tobacco smoke and its components can be seen both at the local and at the systemic level in the human body. At the local level, the mouth is the gateway for tobacco smoke to enter our bodies, and therefore is a direct irritant on oral mucosa. Nicotine also has direct effects on the gums. At the systemic level, tobacco alters the innate and adaptive defense mechanisms and interferes with multiple cellular processes.

Smoking is associated with a long list of systemic diseases and disorders, several of which have an impact on oral health, including, most importantly, periodontal disease and oral cancer. And, tobacco in all of its different forms and uses is, along with alcohol consumption and certain nutritional deficiencies, the leading cause of oral cancer (2). Other disorders that can be caused by smoking include delayed oral wound healing, whether lesions are caused by accidents or periodontal surgery and tooth extraction (3), bad breath, from of the odour of tobacco itself and slight changes in the bacterial microbiota in the oral cavity (4).

Nevertheless, the most commonly occurring smoking-related oral is periodontal disease. Now we will discuss the relationship between smoking and periodontal disease.
Etiopathogenic mechanisms of tobacco

Tobacco has an impact on defense mechanisms mainly affecting neutrophils and inflammatory cell activation, by increasing levels of C-reactive protein, fibrinogen, interleukin-6 and haptoglobin.

Neutrophils are white blood cells that belong to the body's first line of immune defense and are essential for managing the commencement of any infection invading the body. Therefore, neutrophils are essential in the periodontal pocket for holding off the continuous aggression of biofilm when it settles and matures. In this regard, smoking greatly affects the proper function of the neutrophils by reducing chemotaxis, and altering their migration and phagocytosis. In fact, in aggressive periodontal disease a similar problem is observed with an altered neutrophil function, whether acquired or genetic. An altered phagocytic activity may lead to a deficiency in the elimination of pathogens from the oral cavity (5).

Also, B- and T-lymphocytes, when exposed to tobacco smoke, show reduced capacity for proliferation and production of antibodies or immunoglobulin G (IgG), particularly IgG2 against some periodontal pathogens. One explanation could be the increase in systemic oxidative stress that is produced by smoking.

Regarding the effect on cytokines, there is no real conclusive evidence, although overall there seems to be a decrease in cytokines in the gingival crevicular fluid of smokers compared to non-smokers. However, the opposite occurs for serum levels, and the effect on TH2 lymphocytes would increase in relation to periodontal disease progression.

With respect to collagen synthesis and the insertion and proliferation of fibroblasts, in vitro studies, where nicotine levels tend to be very elevated, indicate that these functions are impaired. However, on a clinical level, and with vastly lower nicotine plasma concentrations, it is difficult to extrapolate these results. Furthermore,
smoking greatly increases the release of collagenase-type tissue-degrading enzymes, and at the same time reduces their inhibition, both in serum and in gingival crevicular fluid.

Another key aspect is the increase in reactive oxygen species or free radicals that are observed in smokers. Thus, an imbalance would occur between the levels of oxidants and antioxidants, in favour of the former, and an increase would be observed in the proinflammatory processes such as periodontal destruction.

The relationship between smoking and certain genetic susceptibility traits, such as interleukin 1A and 1B polymorphism, also seems to be important. The probability of having periodontal disease would increase by between 3 and 5 times in smokers who test positive for these polymorphisms.

**Smoking and periodontal disease**

Smoking is the leading environmental risk factor and the second most important modifiable factor, following plaque control, for periodontal disease development (6).

A typical characteristic of smoking-related periodontal disease is tooth-support tissue destruction, with signs showing bone loss, periodontal pocket formation and ultimately, tooth loss.

**Smoking increases the risk for periodontal disease**, depending on the definition of disease, by 5 to 20 times, compared to non-smokers (7). This increase depends on the time of tobacco smoke exposure, and the cause may be related to changes in the oral microbiota or vascular and inflammatory phenomena, as previously explained. Furthermore, nicotine and carbon monoxide from tobacco smoke have a negative impact on wound healing.

Most studies also show that smokers with periodontitis respond less favourably to periodontal treatment, whether surgical or non-surgical, and regenerative and mucogingival surgery is not recommended in these patients.
Long-term studies have proven that smokers have a higher probability of experiencing periodontal disease recurrence during the periodontal maintenance phase, while people who smoke more than 10 cigarettes per day show the most severe disease progression (g). Some studies have even shown that second-hand smoke can have greater periodontal effects, although this is more difficult to determine. Smokers also are at higher risk for experiencing dental implant-related complications, both on the short and the long term. Although, the benefits experienced when people quit smoking are huge for periodontal health.

Disease progression has been shown to be inferior in former smokers and can even be stopped completely. Also, both surgical and non-surgical periodontal treatment outcomes are more satisfactory. Similarly, as gingival crevicular fluid increases from improved microcirculation, both innate and adaptive defense processes improve and periodontopathogenic species in the oral microbiota are shown to decrease.

**Conclusion**

In short, we must keep in mind that the oral cavity is particularly susceptible to the effects of smoking and that the risk of suffering from highly disabling diseases such as periodontitis or even deadly such as oral cancer, greatly multiplies among smokers. Therefore, the role of healthcare professionals is crucial for encouraging smokers to reduce their consumption or to quit smoking altogether. The entire staff in the dental office should be involved, and collaboration with other health professionals such as general practitioners and psychologists is very important. Helping dental patients kick their smoking habit is one of the biggest achievements that can be made in the dental office and is a great step toward helping patients attain optimal oral health, and therefore overall health as well.
References