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POSSIBILITIES TO PREVENT DENTAL CARIES, PERIODONTITIS AND PERIIMPLANTITIS

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Abstract: The prevention of oral diseases (dental caries, periodontal disease, peri-implant diseases) must become the main way to act both individually and in community in order to reduce the risk of local and loco-regional complications and to reduce the financial burden of secondary and tertiary prevention of these diseases.

Despite decades of significant decline, dental caries remains a global public health burden. It is estimated that approximately half of all people worldwide suffer from caries in primary and permanent teeth (1). It is therefore clear that there is a need to intensify the focus and efforts on effective caries prevention and non-restorative caries management.

Dental caries is not a classical infectious disease that can be controlled by removing the pathogens(2). All species of microorganisms associated with dental caries exhibit an acidogenic and acid-tolerant phenotype (3). Species commonly referred to as caries pathogens, are frequently present even in caries-free individuals and constitute 1% of the total bacterial community isolated from caries lesions (4). Recent advances in the role of the oral microbiome in health and disease have provided insights into the various ecological events that act as drivers for the shift of the oral microbiota from symbiotic equilibrium to fatal dysbiosis. The composition of the oral microbiome is individually unique and diverse and the resident oral microbiota is relatively stable and resistant to exogenous influences (2). However, this stability can be lost when environmental stressors override resilience. In caries, the causes of dysbiosis are not only related to bacterial acid production, but also to other biological and non-biological factors such as age, genetics, lifestyle, habits, nutrition and socioeconomics (5).

Established caries prevention interventions therefore need to be understood in a new and broader context to improve their effectiveness, adherence and cost-effectiveness. Some of these general risk factors, such as socioeconomic circumstances and health literacy, are clearly beyond the control of dentists, while others can be influenced but may be underutilised. There are a number of strategies that can be used by patients and clinicians themselves to restore or maintain a healthy oral microbiome. The most important lifestyle-related measures are the reduction of sugar consumption and regular oral hygiene. The most important

biological measures are the use of metabolic inhibitors, pH-enhancing supplements, improvement of salivary flow, and biofilm engineering with pre- and probiotics.

A high-sugar diet is thought to promote the accumulation of acid-forming and acid-tolerant bacterial species and enhance their protection in the extracellular matrix . The amount of sugar ingested was found to have only a marginal effect on microbial profiles in dental plaque and saliva, although some species associated with dental caries were less abundant in the dental plaque of the low-sugar group. Apart from studies with various sugar substitutes, there are no randomized controlled trials that actually demonstrate the clinical efficacy of sugar reduction on caries activity in children and adults.(7)

Host and behavioural factors such as age, smoking, and neglected oral hygiene can lead to systemic changes associated with a disrupted oral ecosystem. Regular and gentle mechanical disruption is considered important for maintaining a healthy biofilm and preventing the development of a mature community, especially at plaque retention sites (1). The composition of the oral biofilm changes over time after oral hygiene (1), and proper and regular twice-daily tooth brushing is likely to maintain the oral microbiome in a favourable state (9). However, it should be emphasised that earlier concepts of plaque eradication or careful plaque control are no longer paramount in caries control. Adequate oral hygiene is undoubtedly central to biofilm control. However, systematic reviews also indicate that mechanical plaque measures without fluoride are not effective for the prevention and treatment of dental caries (10).

Acid production by the biofilm is responsible for the low pH that upsets the ecological imbalance of the oral microbiota. There is strong evidence that fluoride-containing deposits or reservoirs in the oral biofilm control the caries process. Fluoride is known to be able to influence the balance between de- and remineralization, but it can also hinder acid production by saccharolytic bacteria through the formation of calcium fluoride reservoirs in the biofilm matrix (11). The main mechanism of action of intracellular fluoride is inhibition of the enzyme enolase, which is required for catalysis of the breakdown of disaccharides to lactic acid ($_{12}$). Fluoride can also interfere with sugar transport across the cell wall by blocking the phosphotransferase system (13.). The deposition of fluoride reservoirs in oral biofilm is dose-dependent; the higher the fluoride concentration, the greater the number of calcium fluoride-like reservoirs formed. The use of toothpastes, rinses and varnishes with a high fluoride content therefore leads to slightly increased and sustained fluoride concentrations in saliva and in the liquid and solid phases of the dental biofilm (14,15).

Salivary secretion plays an important role in preventing dysbiosis and maintaining health in the oral cavity. In addition to mechanical cleansing and the ability to buffer pH, human saliva contains a variety of mucins, glycoproteins, enzymes, salts, immunoglobulins, and antimicrobial peptides that contribute to biofilm stability and control. Impaired salivary functions as a result of aging, systemic diseases, polypharmacy, and/or chemotherapy and radiotherapy are often

associated with biofilm dysbiosis and excessive growth of Candida species. Although the factors that stimulate salivary secretion are obvious, it is difficult to stimulate salivary flow in practice. Current interventions such as increased water consumption, regular use of chewing gum and saliva substitutes may increase saliva production in patients with residual secretory capacity, although the quality of evidence is considered low (16).

Periodontal diseasea (gingivitis and periodontitis) is an infection of the tissues that hold teeth in place. It is usually caused by poor oral hygiene lead to the formation and persistence of microbial biofilms on the hard, non-shedding surfaces of teeth. Gingivitis is the first manifestation of the inflammatory response to the biofilm and is reversible (i.e., if the biofilm is disrupted, the gingivitis disappears), but if the biofilms persist, gingivitis becomes chronic. In some people, chronic gingivitis develops into periodontitis. In addition to the presence of disease-associated biofilm, these individuals are exposed to additional risk factors, including smoking and systemic comorbidities. Periodontitis is characterized by irreversible tissue destruction that leads to progressive loss of the periodontium and eventual tooth loss. Severe periodontitis is the sixth most common disease in humanity, is associated with decreased quality of life and jaw dysfunction, and is a major contributor to the rise in oral health care costs. It is a public health problem as it is widespread and leads to disability and social inequality.

Periodontitis is initially a local disease of the oral cavity, the pathologies of which include tooth loss, inflammation, difficulty in chewing and bad breath (halitosis). However, it is important to recognize that the disease has a much broader spectrum of effects and possible pathologies that need to focus our attention on its treatment and prevention. Treatment includes good oral hygiene and professional dental cleaning. In certain cases, it also includes the use of antibiotics and periodontal surgery. The most important treatment is undoubtedly primary prevention, which is not only the most effective but also the least expensive method of managing the disease and its complications (17).

Primary prevention with toothbrushes or other mechanical aids has been performed for hundreds of years. In addition to the various types of toothbrushes, other mechanical devices have been on the market for years, including dental floss, water rinses, toothpicks, small interproximal brushes, gum rubber stimulators, and antiseptic solution that include chlorhexidine gluconate-based mouthwashes and other mouthwashes such as Listerine, hydrogen peroxide, slow-release adjuvants (periochip), antibiotics when needed, and toothpaste ingredients such as triclosan, which has recently been replaced by stannous fluoride. (18-21).

Secondary prevention of periodontitis refers to the prevention of recurrence of gingivitis, which can lead to additional attachment loss if periodontitis is successfully treated. In addition, control/treatment of risk factors for periodontitis such as smoking and diabetes is an important component of periodontitis prevention.

Dental implants supporting dentures are part of the oral environment

of a significant proportion of the population and therefore prevention of peri-implant disease should be part of general oral health care. There is wide variation in the age of patients treated with dental implants and the type and extent of implant-supported restorative procedures.

Implant placement requires minor surgical procedures that can lead to immediate surgical complications such as excessive bleeding, poor healing, and infection. After implantation, the implant must be stable and immobile, which is primary stability. This primary stabilization then leads to successful osseointegration, which is the secondary stabilization of the implant. After successful osseointegration of the implant, late complications may occur during the prosthetic and maintenance phases. If these complications are not prevented, peri-implant diseases (peri-mucositis and peri-implantitis) will occur. Peri-mucositis is defined as a reversible inflammatory reaction in the soft tissues surrounding a functioning implant, whereas peri-implantitis is more severe and is defined as a more profound inflammatory lesion characterized by a deepened peri-implant.(22,23). Therefore, prevention of peri-implant disease includes prevention of peri-implant mucositis and prevention of conversion of peri-implant mucositis to peri-implantitis by treating existing peri-implant mucositis (24).

A number of risk factors (genetic predisposition, smoking, periodontitis, poor oral hygiene, systemic diseases, radiation, work techniques) have been found to be associated with the development of peri-implantitis. Understanding the impact of these factors on a person's predisposition to peri-implantitis is extremely important to improve prevention strategies and therapeutic results.

Currently, there are no studies on primary prevention of peri-implant mucositis. This is in contrast to the primary prevention of gingivitis, where there is evidence of the frequency of complete plaque removal required to maintain gingival health (25). Three main treatment groups have been identified, namely mechanical plaque removal with manual or electric toothbrushes, chemical plaque control by the additional administration of antimicrobials, and toothpastes containing triclosan. Manual brushing with or without chlorhexidine gel was studied for 3 months and complete resolution of peri-implant mucositis was observed in 38% of patients/implants. (26).

Professional intervention mainly included oral hygiene instructions, mechanical debridement with various hand- or power-operated instruments and/or polishing tools, and resulted in a reduction of clinical signs of inflammation, while resolution of BoP at the subject level was not achieved. Mechanical plaque control performed by the patient is an effective preventive measure. Chemical plaque control by mouth rinses or a toothpaste tested so far had only a limited complementary effect. Mechanical plaque control by the patient alone (with a manual toothbrush or an electric toothbrush) should be considered the current standard of care. Professionally performed plaque control procedures should include regular oral hygiene instruction (tailored to individual needs) and mechanical debridement using a variety of hand or electric instruments with or without polishing tools.

Conclusions

The increased prevalence of dental caries, periodontitis and other oral conditions among the adult population is an alarm signal for both dental professionals and policy makers because they are due to both the relatively low level of oral health education among the population. as well as low accessibility to dental services. In order to reduce the risk of oral diseases and local and locoregional complications that increase the pressure on the health insurance system, it is necessary to carry out educational programs among the adult population to raise awareness about the importance of primary prevention actions.

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