

Periodontal and Systemic Health - Shaping the Future of our Knowledge

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Abstract

Numerous studies have shown the existence of a close relationship between oral and systemic health, and a two-way interaction is likely to exist in some instances; however, as of today, the mechanisms involved in such an interaction are not completely understood. The scientific evidence demonstrating that people suffering from periodontal infections are more susceptible to metabolic endotoxemia, inflammation, obesity, type 2 diabetes, and other related systemic complications allows to conclude that periodontal diseases represent a risk factor for a wide array of clinically important systemic diseases. Research on the effects of endocrine-disrupting chemicals (EDCs) on obesity, type 2 diabetes, and associated metabolic disorders is a relatively new discipline; nevertheless, a growing number of epidemiological studies reveals associations between EDCs body burdens and a variety of diseases. Future research goals might be aimed at exploring the diverse mechanisms that hint to a certain connection between periodontal infections and EDCs.

Keywords: Periodontal infection, obesity, diabetes, endocrine-disrupting chemicals.

Introduction

One of the most prevalent chronic infections and inflammatory disorders in the world is still represented by periodontal diseases.¹ Periodontal diseases involve two major pathogenic mechanisms: microbial component and host response. Over the past two decades, there has been an extraordinary surge in understanding both aspects. Indeed, the advancement in knowledge and the available evidence have led the European Federation of Periodontology and the American Academy of Periodontology to incorporate power and build up a new classification system for periodontal diseases in 2017.² Periodontology is one of the most magnetic fields of dentistry with tons of new papers, which opens up completely modern pathways of research. It appears that the more we understand the scenario, the complex it becomes; however, this complexity inspires even more scientists to work in this field and triggers building new connections between various dentistry disciplines and medicine to make the story clear. Over the years numerous publications have shown the existence of a close relationship between oral and systemic health, and a two-way interaction is likely to exist in some instances, particularly in diabetes.³ Obesity is another epidemic health problem, and there is substantial evidence from in vivo and in vitro studies demonstrating a strong association between periodontitis and obesity.⁴ As of today, the mechanisms involved in such an association are not completely understood.

Endocrine-disrupting chemicals (EDCs) pertaining to periodontal infection, obesity, and diabetes -

An endocrine-disrupting chemical (EDC) is defined by the Endocrine Society and the World Health Organization (WHO) as “an exogenous substance or mixture that alters function(s) of the endocrine system and consequently causes adverse health effects in an intact organism, or its progeny, or (sub)populations.”^{5,6}

Research on the effects of EDCs on obesity, diabetes mellitus, and associated metabolic disorders is a relatively new discipline. Type 2 diabetes (T2D) and obesity have interconnecting and overlapping pathologies, but there is enough evidence to culminate that some EDCs act as obesogens, and others act as diabetogens. The mechanisms by which EDCs act in the body are quite complicated because EDCs, unlike endogenous hormones, are not natural ligands and do not interact with hormone receptors with the same specificity and affinity. Nevertheless, EDCs interfere with those endogenous systems. In recent years, a growing number of EDCs effects have been observed on these receptor classes, along with another nuclear receptor superfamily such as glucocorticoid and the peroxisome proliferative-activated receptor (PPAR) family. In addition to the nuclear receptor superfamily, more is now known about the effects of EDCs on nuclear and membrane receptors, including receptors associated with steroid membranes, peptide-protein receptors and neurotransmitter receptors.

Consistent evidence strongly supports the assertion that EDCs contribute to diminished quality of life, increased susceptibility to cancer and endocrine diseases in humans. However, as is the case for any system in which the timing of exposures (e.g., fetus) may occur years or even decades before the development of a disease, or when there are lifelong low-level exposures, the issue of causality is difficult to prove, especially in long-lived species such as humans. However, there is increasing evidence that exposures to EDCs play an important role in the causality or progression of the disease or may change the susceptibility to the disease over the course of life.

Experimental animal research, mostly on rodents but increasingly in primates, shows that low-level exposures to EDCs, especially in the early stages of development, cause transient and permanent changes in endocrine systems. This results in impaired reproduction, thyroid function, and metabolism, and increased incidence and progression of hormone-sensitive cancers.

Since the endocrine systems of all mammals, including humans, are highly preserved, this biomedical research literature is highly transposable. Finally, an increasing number of epidemiological studies show associations between body loads of EDCs and a variety of diseases.

Cell and animal models demonstrate the role of EDCs in the etiology of obesity and T2D. With respect to obesity, animal studies show that EDC-induced weight gain is dependent on the period of exposure and the age of the animals. Exposures during the perinatal period lead to obesity later in life. New results covering a range of EDCs doses have demonstrated the importance of nonmonotonous dose-response relationships; some doses have led to an increase in weight, while others have not. In addition, EDCs cause obesity by acting directly on white adipose tissue, although the brain, liver and even the endocrine pancreas can be direct targets.

In the case of T2D, animal studies indicate that some EDCs directly target β and α cells in the pancreas, adipocytes and liver cells and cause insulin resistance and hyperinsulinemia. These changes can also be linked to changes in adiponectin and leptin levels, often in the absence of weight gain. This diabetogenic effect is also a risk factor for cardiovascular disease, and hyperinsulinemia can lead to diet-induced obesity.

The human epidemiological studies also indicate an association between EDC exposure, obesity and TD2; however, given that multiple epidemiological studies are cross-sectional and diet is a major confounding factor in humans, its causality cannot yet be inferred.⁷

The oral cavity, with its various bacterial populations, could also function as an origin site for the propagation of pathogenic microorganisms at different sites in the body, especially in immunocompromised hosts, the elderly, or the underprivileged. Several scientific publications have advocated that patients with periodontal diseases are more susceptible to metabolic endotoxemia, inflammation, obesity, T2D, and other related systemic complications, concluding that periodontal diseases could be a potential contributing risk factor for a wide array of clinically important systemic diseases. However, despite a significant increase in the prevalence of periodontal infections and systemic diseases in the past few decades, the fundamental biological mechanisms of connection between these ailments are still not fully interpreted. As a result, the mechanisms by which these bidirectional damages occur are explored with a concentric vision in order to develop strategies that could prevent or control the complications of these ailments.⁸

Conclusion

Future research goals might be aimed at exploring the diverse mechanisms that hint to a certain connection between periodontal infections and EDCs. The results of the research may prove beneficial to researchers, dentists, physicians and other health care providers by translating the science of endocrine disruption into improved public health.

Conflict of Interest

The author declare no conflict of interest.

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