Modern management of periodontal diseases

The UK Adult Dental Health Survey (1998) has shown that approximately half the adult population of England and Wales have moderate periodontal disease, and approximately 8% have severe periodontal disease. Similar results have been found in population surveys in other countries. Periodontal diseases are bacterially induced destructive conditions in which random bursts of acute inflammation occur in susceptible sites around the mouth. This results in progressive destruction of the connective tissues and bony support for the teeth.

By Dr Peter Galgut

Numerous studies have demonstrated that a clear relationship exists between dental plaque and gingivitis, but other factors in addition to dental plaque are necessary to predispose individuals to periodontal diseases. Periodontitis and Peri-implantitis have a multi-factoral aetiology, and this is summarised in Figure 1.

Gingivitis is distinguished by the fact that it is superficial inflammationonly, presenting as swelling and bleeding . Periodontitis is inflammation and usually destruction of the deeper underlying tissues. It has also been shown that gingivitis does not necessarily progress to perio0dontitis as previously thought, but more importantly, periodontitis can and frequently does, occur without any clinical signs of gingivitis (ie redness, swelling and bleeding of the gingivae) as illustrated in figures 1 and 2

Environmental factors such as smoking, stress, and psychological states such as depression, anxiety, and detrimental life events are all well recognised co-factors in the aetiology of periodontitis.

However, some individuals are more susceptible to periodontal breakdown than others, and the question still remains as to why this should be so. In the absence of medical conditions such as diabetes, which compromise the immune response, other factors, such as genetic polymorphisms (i.e. variations in the sequence of amino acids on the DNA of chromosomes, which determine the variations in the genes) have been shown to be important.

Early studies into the role of genetic factors in periodontal disease susceptibility demonstrated that variations in periodontal tissue destruction existed between different strains of mice, indicating a possible genetic background.[1] Other small human studies on specific conditions like Early Onset Periodontitis, and monozygotic/ dizygotic twins, confirmed the possibility of a significant contribution to the genetic susceptibility to periodontal destruction.[2] Recent studies into genetic polymorphisms have concentrated on the genes for coding inflammatory mediating molecules such as interleucins (IL-1,6,8), prostaglandins (PG), Tumour Necrosing Factors (TNF a), and metalloproteins.[3,4,5,6] Specific genotypes have identified individuals with a high susceptibility to severe periodontal destruction. It has been shown that an exaggerated response to infection may be present in as many as one in four individuals attributable to genetic factors. Interleucin 1b has been associated with severe inflammatory responses, particularly in destructive periodontal diseases. Inhibition of Interleucin production has been shown to have resulted in significant reductions in bone loss of in animal studies.[6] This inflammatory mediator has therefore been the subject of considerable interest.[2]

The Current view of how all of these factors interact is summarised in Figures 3 and 4.

Therefore, the aetiology and management of periodontal diseases has to not only concentrate on managing the

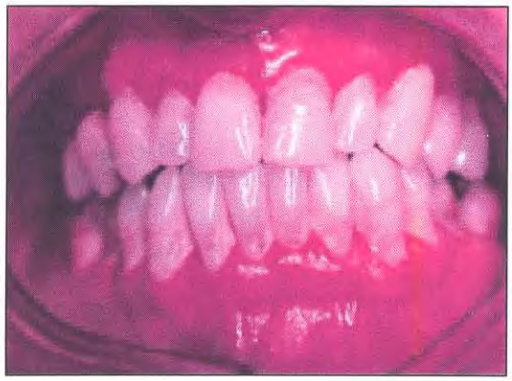

Fig. 1: Gingivitis is obvious, presenting with swollen, bleeding and red gingivae, usually with an abundance of dental plaque.

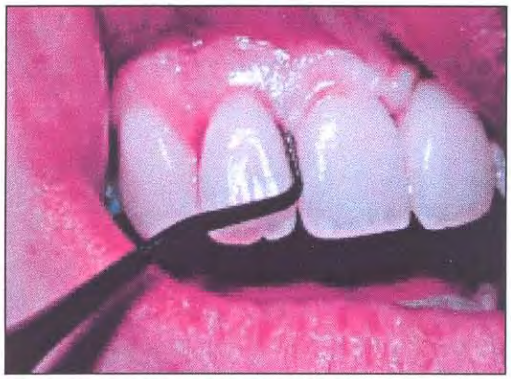

Fig. 2: Periodontitis is often missed because the gingivae look healthy, and are symptomless, but the problem is now subgingival where it can only be diagnosed with a periodontal probe.

infective condition as effectively as possible but also the genetic background of individuals, and the host responses to the infecting organisms which are directly related to the genetic factors described above.

Another major problem is the recognition that there are a number of different "Periodontal Diseases"! In other words, not all periodontal disease is the same. The classifications and terminology for these conditions changes every few years, but currently the classification includes chronic adult periodontitis, aggressive periodontitis (which includes such conditions as Early Onset and Refractory periodontitis) and periodontitis complicated by medical and other factors.

The pathogenesis of periodontal diseases has also come under scrutiny, and this has added to the complexity of periodontal disease management. It was

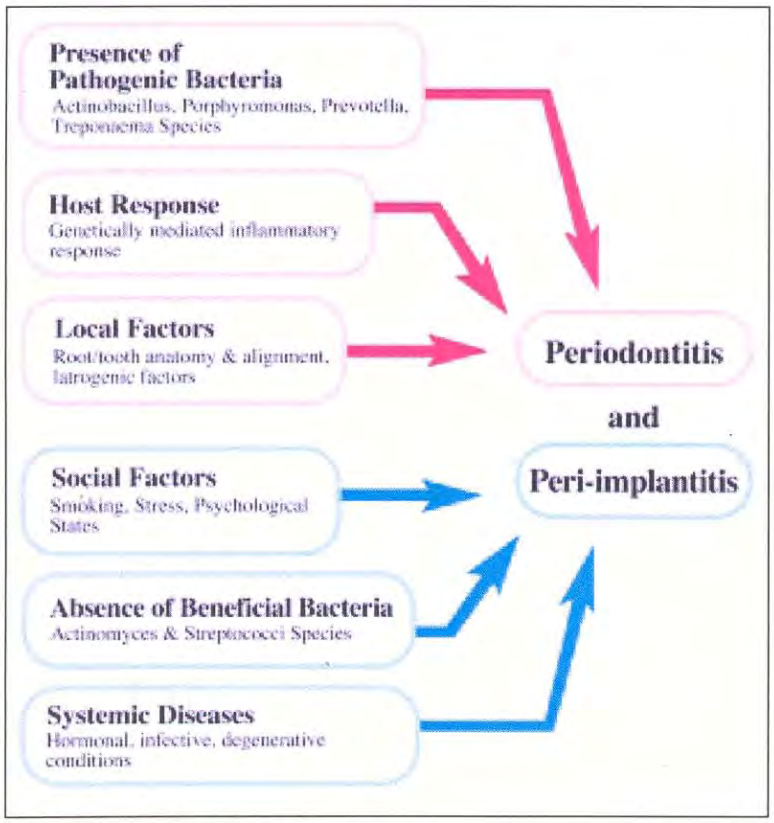

Fig. 3: Aetiology of periodontal diseases

assumed that once periodontal disease had started, it slowly progressed with the increasing pockets depths due to on going inflammatory breakdown with time. It is now believed that periodontal diseases are not only site-specific (in other words some sites seem to be more susceptible than others), but also different sites are randomly affected at different times. Also, there seem to be periods of activity and quiescence so condition seems to come and go and wander around the mouth haphazardly. This is known as the random burst hypothesis of periodontal disease progression.[7] Also, it is increasingly recognised that pockets can heal so that pocket depths appear to reduce with time in some areas, increase in others, and yet to in others, don't seem to change at all!

Management of periodontal diseases

The clinical management of a condition that randomly affects different sites in the mouth at different times is difficult. When the additional genetic factors are considered, it is even more difficult to manage.[2] Strategies for the management of periodontal diseases with these factors in mind are summarised in Figure 5.

Clearly the accepted regimen of repeated scalings, and root planing by quadrants, or even root planing sites that are active only, is inadequate to control this condition. Increasingly it is recognised that partial debridement of the mouth simply results in cross contamination or re-infection from sites that have not received debridement and from other areas of the mouth.

Thus, "Full Mouth Disinfection" as described by Quirinen and his group[7,8,9,10,11,12,13,14,15,16] is increasingly seen as a means of controlling this condition more effectively. In this treatment strategy, elimination of as much bacterial contamination of the oral cavity is attempted at a single appointment. This may include not only full mouth scaling and root planing.

In those cases that respond poorly to mechanical debridement (i.e. deep scaling and root planing), and do not heal, other strategies for controlling the destructive inflammatory process need to be adopted. In the first instance an assessment of contributing social factors should be carried out. If the patient is a smoker, even if it is only a few cigarettes per day, smoking cessation advice should be given in view of the well-documented detrimental effects of smoking on the gingival and periodontal tissues.[17, 18] Other related factors such as recent job losses, marital breakdowns, bankruptcy etc which cause stress should be investigated as well as the possibility of an underlying anxiety state, or state of depression.[19, 20] If necessary the patient might need to be referred for counselling and help in management of these social factors. Bad dietary habits may also play a part in facilitating plaque buildup, but also inhibiting healing and these too should be investigated.

A thorough investigation of host modifying factors should be undertaken. Patients with diseases such as diabetes or other major illness have compromised immune systems which compromise healing. However, a large number of patients are not frankly ill, but take medications such as tranquillisers, anti-depressants, calcium channel blockers, immunosuppressants, and many others that affect the state of the gingivae, and their healing capacity. Appropriate action may be required if any of these host

modifying factors are pertinent to the poor healing response.

Increasingly the use of chemotherapetic adjunctive agents to enhance the effects of mechanical root surface debridement are being used to overcome the limitations of scaling and root planing techniques, as well as the difficulties presented in those patients with poor healing. Traditionally, the use of antiseptic mouthwashes before and after treatment, and flushing out periodontal pockets with antiseptics during treatment has been the only way to achieve this. As antiseptics are rapidly flushed away, they have been shown to have limited benefit only. A range of topical slow release antimicrobial products is now available to place into pockets to prevent re-infection, and also to eliminate residual bacteria that are not destroyed by mechanical debridement alone. Possible pathways to the use of these chemical adjunctive agents are illustrated in Figure 6

Exciting new products are also available that are specifically designed to manage host factors. Two products are currently available. Hyaluronic acid is one of the basic ingredients in tissue ground substance. It mediates the inflammatory response, and potentiates wound and tissue healing. In reducing the inflammatory response, and encouraging healing of damaged tissues, it has great potential to dampen the inflammatory response. In individuals who are susceptible to periodontal destruction, or who demonstrate poor healing after mechanical treatment, or have a high propensity for recurrent periodontal disease this product offers exciting possibilities for better management of periodontal problems. This product (Trade name Gengigel) is a topical application that is available for professional application into pockets, and a mouthwash for regular home use by patients. A systemic anti-inflammatory product has also been launched recently. This product relies on the well established anti inflammatory characteristics of the tetracycline group of antibiotics. Doxycycline is a member of this group of antibiotics, and it is administered at such low doses that it has no antimicrobial effects, but the dampening of the overactive host response to plaque bacteria remains. Low dosage Doxycycline is another product that offers great potential to improve our long term management of periodontal diseases more effectively in clinical practice.

This is all very well, but the genetically mediated host response to the bacterial infection is not managed by these strategies. Although tests are available to determine the inherent susceptibility to periodontal diseases, at this point in time Terry is no treatment available to neutralise these genetic predispositions. As

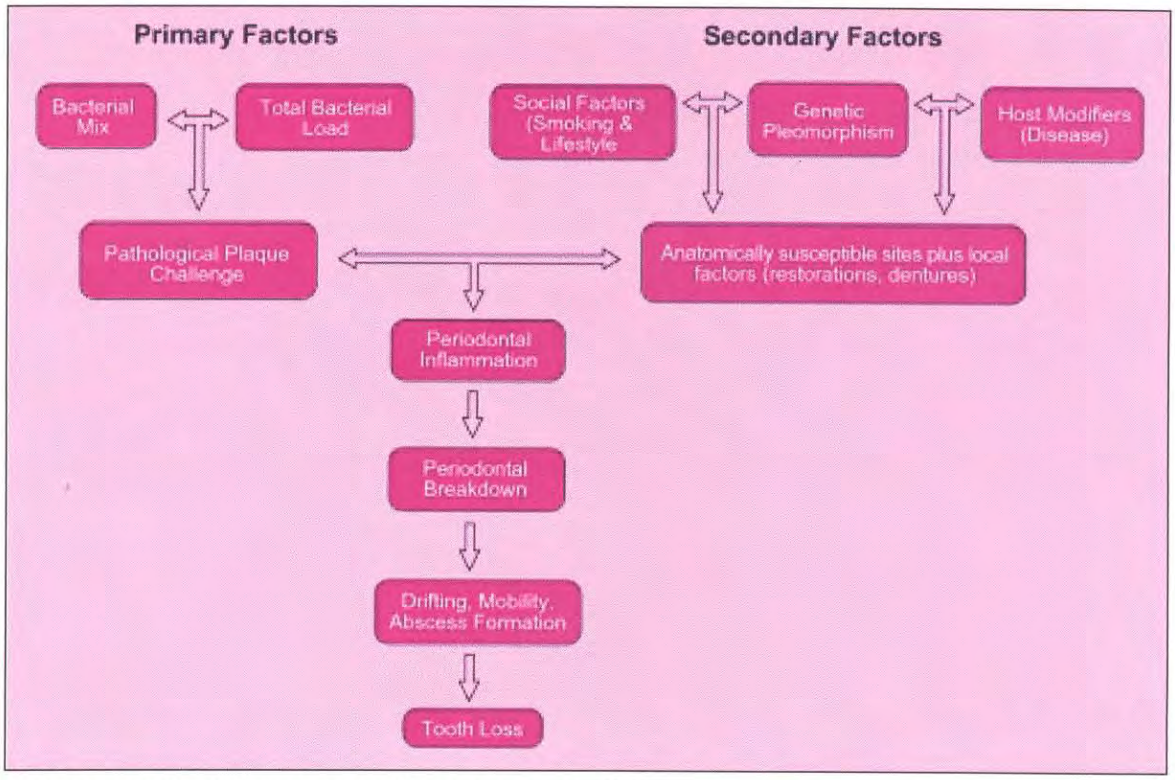

Fig. 4: The complex aetiology of periodontal disease and peri-implantitis

a result, we have to accept that in some of our patients we will never be able to achieve a cure. Increasingly, the aim of treatment is not so much to cure the condition, but to stabilise it to preserve of much of the dentition for as long as possible. In those individuals in which host factors cannot be managed, then indefinite retreatments for the ongoing condition may necessary and even then it may not be possible to arrest the destructive process completely.

Conclusion

Combating periodontal diseases seemed to be so simple in the past, as it was considered to be a simple plaque based infection. With better understanding of the aetiology and pathogenesis of the condition, more sophisticated treatment strategies are being developed to improve our clinical management of periodontal our patients. These new treatment strategies aimed at achieving long term stability of the gingival tissues and preventing further loss of periodontal supporting tissues are already enabling clinicians to manage periodontal diseases more effectively, and are likely to do so even more effectively in the future.

References

1. Baer, P.N. and Lieberman, J.E. Observation on some genetic characteristics of the Periodontium in three strains of inbred mice. OS,OM & OP. 1959:12: (7):820-829.
2. Galgut PN., Dowsett SA., Kowolik MJ., : Periodontics: Current Concepts and Treatment Strategies: Martin Dunitz Ltd., London. 2001: ISBN 1-85317-981-7
3. Kornman KS.and di Giovine FS.: Genetic Variations in Cytokine Expression: A Risk Factor for Severity of Adult Periodontitis. Ann. Periodontol. 1998: 3: 1: 327-338.
4. Diehl SR., Wang YF., Books CN, et al.: Linkage Disequilibrium of Interleukin-1 Genetic Polymorphisms with Early-Onset Periodontitis. J. Periodontol. 1999: 70: 418-430.
5. Kobayashi T., Sugita N., Van Der Pol W-Ludo., Nunokawa Y., Westerdall Nomdo AC., Yamamoto K., van de Winkel Jan GJ. And Yoshi H.: The FcY Receptor Genotype as a Risk Factor for Generalized Early-Onset Periodontitis in Japanese Patients. J. Periodontol. 2000: 71: 1425-1432.
6. Graves DT., Delima AJ., Assuma R.,

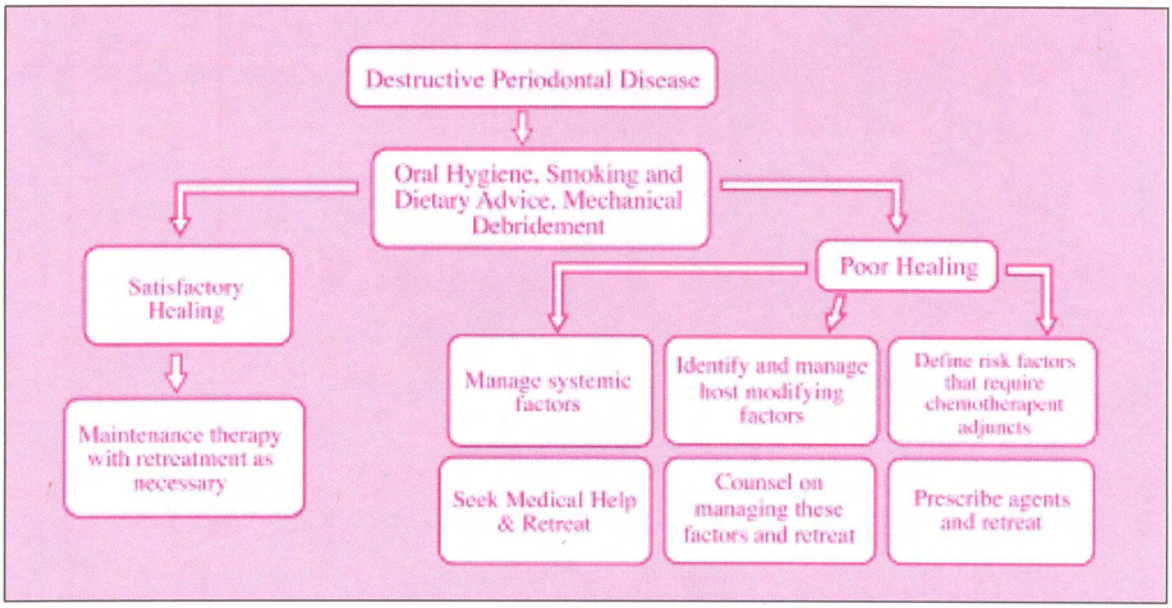

Fig. 5: A guide to decision making in periodontal therapy

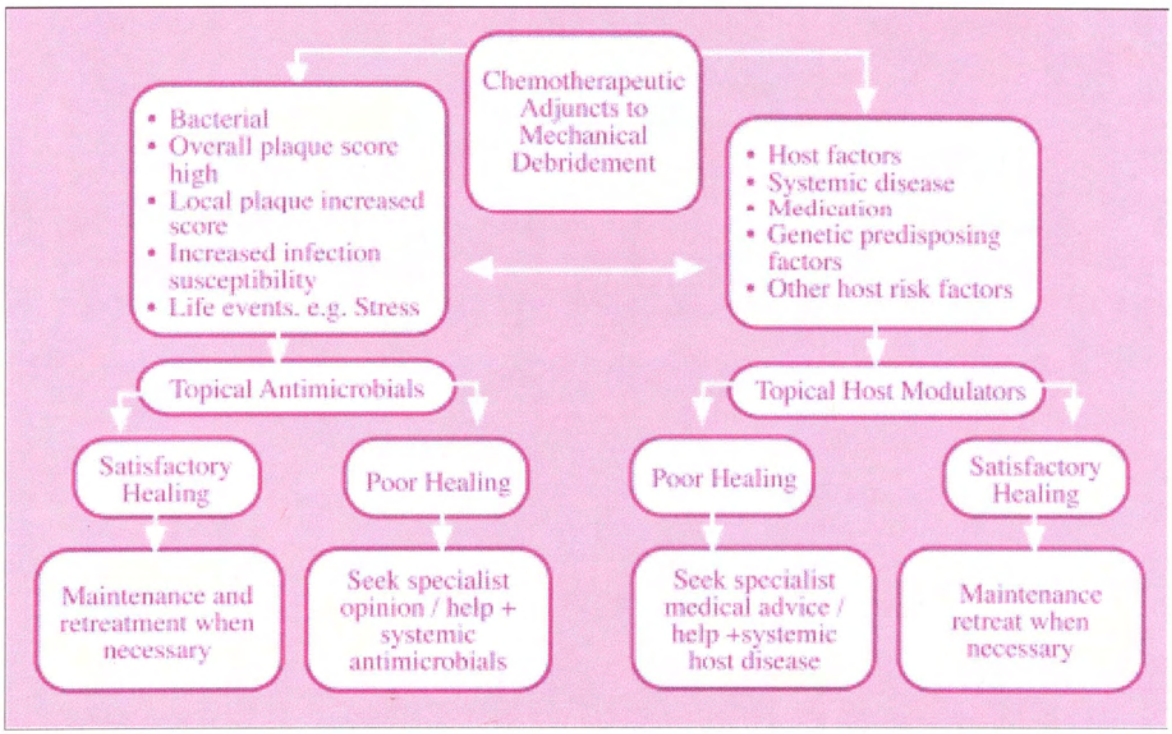

Fig. 6: A guide to using chemical adjunctive agents in clinical management of periodontal diseases

Amar S., Oates T., Cochran D.,: Interleukin-1 and Tumor Necrosis Factor Antagonists Inhabit the Progression of Inflammatory Cell Infiltration Toward Alveolar Bone in Experimental Periodontitis. J. Periodontol. 1998: 69: 1419-1425.
7. Goodson JM, Tanner ACR, Haffajee AD et al: J Clin Periodontol. 1982;9: 472-481
8. Vandekerckhove BN, Bollen CM, Dekeyser C, Darius P, Quirynen M.: Full- versus partial-mouth disinfection in the treatment of periodontal infections. Long-term clinical observations of a pilot study. J Periodontol. 1996 Dec;67(12):1251-9.
9. Bollen CML and Quirynen M:Microbiological Response to Mechanical Treatment in Combiation with Adjunctive Therapy. A Review of the Literature. J. Periodontol. 1996: 67: 1143-1158
10. Quirynen M, Mongardini C, van Steenbergh: The effect of a 1-stage full-mouth disinfection on oral malodor and microbial colonization of the tongue in periodontitis. A pilot study. J Periodontol. 1998 Mar;69(3):374-82.
11. Quirynen M, Mongardini C, Pauwels M, Bollen CM, Van Eldere J, van Steenberghe D.: One stage full- versus partial-mouth disinfection in the treatment of chronic adult or generalized early onset periodontitis. II. Long-term impact on microbial load.J Periodontol. 1999 Jun;70(6):646-56.
12. Mongardini C, van Steenberghe D, Dekeyser C, Quirynen M.: One stage full- versus partial-mouth disinfection in the treatment of chronic adult or generalized early onset periodontitis. I. Long-term clinical observations. J Periodontol. 1999 Jun;70(6):632-45.
13. Quirynen M, Mongardini C, de Soete M, Pauwels M, Coucke W, van Eldere J, van Steenberghe D.: The role of chlorhexidine in the one stage full mouth disinfection treatment of patients with advanced adult periodontitis. Long-term clinical and microbiological observations. J Clin Periodontol. 2000 Aug;27(8):578-89.
13. De Soete M, Mongardini C, Peuwels M, Haffajee A, Socransky S, van Steenberghe D, Quirynen M.: One stage full-mouth disinfection. Long term microbiological results analyzed by checkerboard DNA DNA hybridization. J Periodontol. 2001 Mar;72(3):374-82.
14. Quirynen M, Peeters W, Naert I, Coucke W, van Steenberghe D.: Peri implant health around screw-shaped c.p. titanium machined implants in partially edentulous patients with or without ongoing periodontitis Clin Oral Implants Res. 2001 Dec;12(6):589-94.
15. Quirynen M, Teughels W, De Soete M, van Steenberghe D.: Topical antiseptics and antibiotics in the initial therapy of chronic adult periodontitis: microbiological aspects. Periodontol 2000. 2002;28:72-90.
16. Quirynen M, Zhao H, van Steenberghe D.: Review of the treatment strategies for oral malodour. Clin Oral Investig. 2002 Mar;6(1):1-10.
17. Page: The Pathobiology of Periodontal Diseases may Affect Periodontal diseases: Inversion of a Paradigm Ann Periodontol 1998, 3,108-20
18. Kamma JJ, Giannopoulou C, Vasdekis VG: Cytokine profile in gingival crevicular fluid of aggressive periodontitis: Influence of smoking and stress. Mombelli A. J Clin Periodontol. 2004 Oct;31(10):894-902.
19. Krahwinkel T, Willershausen B, Boekstegen C. Relationship between stress factors and periodontal disease. Pistorius A, Eur J Med Res. 2002 Sep 30;7(9):393-8.
20. Merchant AT, Pitiphat W, Ahmed B, Kawachi I, Joshipura K. A prospective study of social support, anger expression and risk of periodontitis in men. Am Dent Assoc. 2004 Apr;135(4):406, 408, 410

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