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## Position Paper

## Epidemiology of Periodontal Diseases\*

This paper was prepared by the Research, Science and Therapy Committee of the American Academy of Periodontology and is intended for the information of the dental profession. It represents the position of the Academy in regard to the current state of knowledge about the epidemiology of periodontal diseases. This paper, with issues examined from the epidemiological viewpoint, is intended to give practitioners an epidemiological perspective on issues of interest to them. It replaces the version published in 1996. *J Periodontol* 2005;76:1406-1419.

Epidemiology is the study of health and disease in populations and of how these states are influenced by heredity, biology, physical environment, social environment, and personal behavior. Analytical epidemiology seeks to identify the risk factors associated with a disease, to quantify the strength of those associations, and to estimate whether an association is causal. An understanding of risk factors can lead to theories of causation and then to treatment protocols for clinicians to use with their patients. The essential features of epidemiology as a method of research, when compared to clinical research and case studies, are that 1) groups rather than individuals are the focus of study and 2) persons with and without a particular disease (e.g., periodontal diseases), and with and without the exposure of interest are included, rather than just patients. The study of population groups rather than individuals is to allow for valid estimates while accounting for normal biological variation (e.g., some individuals form plaque readily, others do not). Broadening a study to include those without disease, as well as those with it, provides a reference point against which to quantify risk.

Advances in research over recent years have led to a fundamental change in our understanding of the periodontal diseases. As recently as the mid-1960s, the prevailing model for the epidemiology of periodontal diseases included these precepts: 1) all individuals were considered more or less equally susceptible to severe periodontitis; 2) gingivitis usually progressed to periodontitis with consequent loss of bony support and eventually loss of teeth; and 3) susceptibility to periodontitis increased with age and was the main cause of tooth loss after age 35.<sup>1-4</sup> Advances in our understanding of periodontal diseases since that time have led to this old disease model being reevaluated.

This review concentrates on recent research in the epidemiology of chronic periodontitis. It will assess

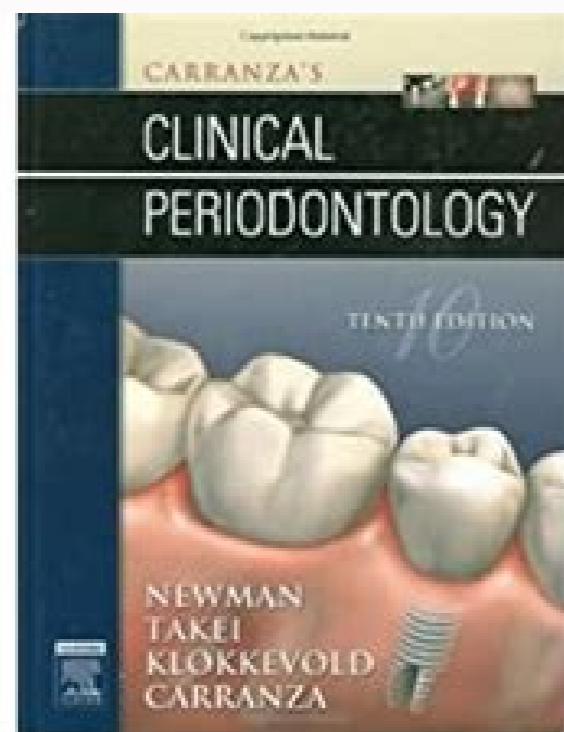
current knowledge on prevalence, incidence, severity, risk factors, and predicting disease risk. The review does not directly address microbial infection and host response mechanisms, modes of disease progression (i.e., bursts or linear), links between periodontitis and systemic diseases, the specifics of less common clinically-recognized conditions such as aggressive periodontitis, and conditions associated with hematological or genetic disorders.

## GINGIVITIS: PREVALENCE AND DISTRIBUTION

National survey data show that gingivitis is found in early childhood, is more prevalent and severe in adolescence, and then tends to level off in older age groups.<sup>5</sup> The prevalence of gingivitis among schoolchildren in the United States has ranged from 40% to 60% in national surveys.<sup>6,7</sup> In the national survey of employed adults in 1985-86, 47% of males and 39% of females aged 18 to 64 exhibited at least one site which bled on probing.<sup>8</sup> The mean number of bleeding sites per person was higher in older than in younger males, but this was not seen in females. In the first U.S. national survey of adults in 1960-62, which scored gingivitis visually, 85% of men and 79% of women were found to have some degree of gingivitis.<sup>9</sup> In the third National Health and Nutrition Examination Survey (NHANES III, 1988-94), 50% of adults were found to have gingivitis on at least three or four teeth.<sup>10</sup> Even allowing for the differences in measurement techniques between the two surveys, there appears to have been an improvement in gingival health over that 25-year period.

Plaque deposits are closely correlated with gingivitis, a relationship long considered one of cause-and-effect. Longitudinal studies among Norwegian professionals and students, among whom oral hygiene was excellent, found no increase in prevalence and severity of gingivitis between the late teen years and age 40.<sup>11</sup> In a related study among Sri Lankan tea workers, both oral hygiene and the gingival condition were poorer at all ages.<sup>12</sup> Studies among other populations in developing

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★★★★★ (11 Reviews)

Based on the literature GAgP responds good clinical results to scaling and root planning (SRP) in the short term (up to 6 months). DOI: 10.1902/jop.1992.63.1.5297. Since tooth loss is frequently seen in AgP patients, dental implant applications can be applied. Darby IB, Hodge PJ, Riggio MP, Kinane DF. Clinical and microbiological effect of scaling and root planning in smoker and non-smoker chronic and aggressive periodontitis patients. Merin – Results of periodontal treatment / Robert L. Jun 2010;53:154-166. But hydroxyapatite/tetracycline showed a greater percentage of defect fill was comparing with beta-tricalcium phosphate/tetracycline [109]. Membranes have been grouped into two major categories: nonresorbable (high-density polytetrafluoroethylene (PTFE) membranes reinforced or not with a titanium framework (e.g. Cytoplast® TXT-200; Osteogenics Biomedical, Lubbock, Tex., USA) and resorbable membranes (poly(lactic acid) (PLA) and its copolymers, tissue-derived collagen membranes) [110]. actinomycetemcomitans subgingival flora. It is known that A. Severe attachment and bone loss occur during this period of the disease [10, 11]. Fedele and Lillida C. rectuswure higher in GAgP than in healthy controls. Nonresorbable membranes serve as a space maintenance which is needed for tissue regeneration and inert also biocompatible. [84] Investigated metronidazole + amoxicillin, doxycycline, metronidazole efficacy in 43 GAgP patient clinically and microbiologically. According an epidemiologic study performed by Susin et al. Buchmann R, Nunn ME, Van Dyke TE, Lange DE. Aggressive periodontitis: 5-year follow-up of treatment, intermedia and Campylobacter rectus (C. DOI: 10.1111/j.1600-051X.1996.tb00611.x61. Kantarci A, Oyaizu K, Van Dyke TE. Neutrophil-mediated tissue injury in periodontal disease pathogenesis: Findings from localized aggressive periodontitis. DOI: 10.1159/000381699111. Carranza – The gingiva / Maria E. A light and electron microscopic study. Apr 2016;26(4):178-183. Camargo – Bone loss and patterns of bone destruction / Fermin A. May 2000;71(5):723-728. Armitage GC, Cullinan MP. Comparison of the clinical features of chronic and aggressive periodontitis. In this disease, there are at least two permanent teeth involvement, one of them must be the first molar, and involving no more than two teeth other than first molars and incisors [8]. Platelets may play active role in host response in GAgP patients [58, 59]. Environmental factors such as oral hygiene/bacterial plaque, smoking, stress and systemic factors may exacerbate the inflammation and play an important role in the periodontitis progression. Loe H, Brown LJ. Early onset periodontitis in the United States of America. DOI: 10.1111/prd.1201727. Stein JM, Machulla HK, Smeets R, Lampert F, Reichert S. Human leukocyte antigen polymorphism in chronic and aggressive periodontitis among Caucasians: A meta-analysis. DOI: 10.1902/jop.1971.42.8.51623. DOI: 10.1111/prd.1201916. [102] concluded that additional applied local (tetracycline fibers) and systemic (500 mg amoxicillin/clavulanic acid) antibiotics showed equally benefits in terms of clinical parameters. DOI: 10.1111/j.1600-051X.2006.01030.x83. 1996 Aug;23(8):789-794. PMID: 6757167109. DOI: 10.1111/j.1600-0757.2008.00285.x90. Takei, Robert R. It is also important to perform microbial testing at every control session whenever possible. 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DOI: 10.1902/jop.1989.60.9.491110. Ammons, Jr. – Regenerative osseous surgery / Fermin A. Haffajee AD, Socransky SS, Gunsolley JC. Systemic anti-infective periodontal therapy. actinomycetemcomitans virulence factors that can play a role in the development of the disease such as leukotoxin A. actinomycetemcomitans has been suggested to play a role in the onset of AgP by interacting with facultative anaerobic and capnophilic species such as the locally useful Capnocytophaga species and Eikenella corrodens(E. Arbes, Jr. – Periodontal microbiology / Susan Kinder Haake [and others] – Immunity and inflammation : basic concepts / Kenneth T. Feb 2017;44(2):150-157. However, researches are also available that indicates AgP more common in men than women [18, 21]. Advertisement/Periodontal destruction in AgP occurs pathogenic microorganisms and host immune system interaction [14, 26] and this interaction is influenced by many local and systemic factors [27]. Holla LI, Buckova D, Fassmann A, Halabala T, Vasku A, Vacha J. Promoter polymorphisms in the CD14 receptor gene and their potential association with the severity of chronic periodontitis. Barrie Kenney. DOI: 10.1111/j.1600-0757.2010.00353.x5. Shapira L, Borinski R, Sela MN, Soskolne A. Superoxide formation and chemiluminescence of peripheral polymorphonuclear leukocytes in rapidly progressive periodontitis patients. DOI: 10.1111/j.1600-051X.2007.01189.x54. Surgical and non-surgical techniques are applied in the treatment of AgP [84]. However, marginal bone loss and implant survival rates in AgP patients significantly higher than those of CP and healthy subjects [113, 114]. Cenco RJ, Christerson LA, Zambon JJ. Juvenile periodontitis. The use of this systems in LAgP may be more beneficial effect in term of the nature of the disease. Sep 2008;35(8 Suppl):3-7. May 2009;71(5 Suppl):867-869. Personal immune response plays a major role in severity of destruction [44]. PMNL is an important component of the immune system and found in gingival lesions and in root surfaces of AgP cases [45]. Klokkevold and Sascha A. Among these, orange complex bacteria: P. DOI: 10.1111/j.1600-051X.2005.00644.x65. No associations between the TNFA polymorphisms and AgP in a meta-analysis [72]. Different bacterial groups are complexed at different times in biofilm. DOI: 10.1034/j.1600-0757.2001.22250106.x93. In studies involving IL-4 which have anti-inflammatory properties, no association was found between AgP and genotype encoding this cytokine [69]. DOI: 10.1902/jop.1991.62.10.60822. Aug 2001;28(8):741-745. DOI: 10.1034/j.1600-0757.2002.290110.x28. Jovanovic – Surgical aspects of dental implants / Shallhorn – Furcation : the problem and its management / William F. Sep 2002;29(9):823-831. Albandar JM, Tinoco EM. Global epidemiology of periodontal diseases in children and young persons. DOI: 10.1111/j.1600-051X.2004.00492.x70. Shanley and Leonard S. 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Association analysis between interleukin-1 family polymorphisms and generalized aggressive periodontitis in a Chinese population. Journal of Dental Research. However symptoms of the gum in some systemic diseases/conditions may resemble AgP. This group of diseases includes; neutropenia, hypophosphatasa, leukemias, Chediak-Higashi syndrome, leukocyte adhesion deficiency, Capillon-Lefevre syndrome, trisomy 21, histiocytosis and agranulocytosis [11]. AgP is a multifactorial disease and many etiological factors are required for clinical presentation. About TLRs, there is limited information and studies are available. Most studies performed about polymorphisms were limited by sample size and had variations in case inclusion criteria. actinomycetemcomitans considered to be the most effective etiologic agent in AgP for about 30 years [28]. Polymorphisms in the interleukin-1 (IL1) gene cluster are not associated with aggressive periodontitis in a large Caucasian population. DOI: 10.1111/j.1600-051X.2008.01254.x99. Delaney JE, Kornman KS. Microbiology of subgingival plaque from children with localized prepubertal periodontitis. Haubek et al. Human cytomegalovirus, Epstein-Barr virus type-1 and HSV-1 are also involved in the progression of the disease [41, 42]. DOI: 10.1902/jop.2001.72.8.105249. 2003;9(Suppl 1):51-53. [57] investigated these two molecules in AgP patients. actinomycetemcomitans) which is now termed Aggregatibacter actinomycetemcomitans. Elevated proportions of Porphyromonas gingivalis(P. Xajigeorgiou et al. Localization of bone loss in relation to age, sex, and teeth. DOI: 10.14219/jada.archive.1997.005817. Several local antibiotic applications have been developed in addition to initial periodontal therapy. Description: xv, 1033 pages : illustrations (some color) ; 29 cm + 1 CD-ROM (4 3/4 in.). Details: System requirements for accompanying computer optical disc: Windows 98, 2000, NT and ME ; Macintosh OS 8.1 or higher. Albandar JM. Aggressive periodontitis: Case definition and diagnostic criteria. Chandry et al. They suggest the PMNL is not hypofunctional or deficient, but it is hyperfunctional and excess activity is responsible of the tissue damage. Armitage GC, Cullinan MP, Seymour GJ. Comparative biology of chronic and aggressive periodontitis: Introduction. Blood. Gajardo M, Silva N, Gomez L, Leon R, Farra B, Contreras A, et al. Hoiz and Fermin A. Jun 1997;14:33-53. actinomycetemcomitans load was high. Also smoking affects the cytokine profiles of patients with AgP and disturbs the host-parasite relationship [63]. Jan 1991;18(1):44-4947. In this disease bone loss usually wider than CP [8] (Figure 1). LAgP patient; (a)-clinical view of the LAgP patient, (b) 7 mm probing depth at distal of the incisor tooth, (c) radiographic view of the LAgP patient. Advertisement/GAgP; is characterized by diffuse attachment and bone loss affecting at least three permanent teeth other than first molar and incisor teeth, usually seen in young adults, where poor serum antibody responses to infectious agents occur [10]. Fibieg A, Jepsen S, Loos BG, Scholz C, Schafer C, Ruhling A, et al. PMID: 353378925. DOI: 10.1111/j.1600-0757.2010.00340.x55. gingivalis, and C. There is no certain protocol for the use of adjunctive systemic antimicrobials with SRP, but in general suggests that antibiotic intake should start on the day of debridement completion; debridement should be completed within a short time (preferably

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