

An overview of the epidemiology of periodontal diseases in Latin America

Visão geral da epidemiologia das doenças periodontais na América Latina

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Abstract: The aim of the present review was to identify the presence of periodontal diseases and the relative importance of known risk factors in Latin American countries. The retrieved data are sparse and inconsistent, lacking information for the majority of the countries. Gingivitis is ubiquitous in the region, affecting all ages independently of the socioeconomic background. The extension of bleeding may vary greatly, ranging from 40% to 70% of sites. The prevalence of aggressive periodontitis is higher in Latin America than in industrialized countries. Prevalence ranges from 0.3% to 4.5%, and the localized form is the less prevalent. The prevalence of chronic periodontitis is high, with a large variation (40-80%) probably due to differences in methodology and diagnostic criteria. Regional differences may be relevant. Known risk factors are present in the populations studied. Non-modifiable factors such as age, gender and genetics have been associated in Brazilian and Chilean populations. Tobacco smoking and diabetes are relevant risk factors. The importance of socioeconomic status, although present as a risk factor, has been largely underestimated. Oral hygiene is extremely deficient in the area although it is a cultural habit in most populations. It can be concluded that periodontal diseases are highly prevalent in Latin American populations. Its prevalence and extent are associated with known risk factors. Oral hygiene habits are deficient. Well designed epidemiological studies with external validity are needed.

Descriptors: Epidemiology; Latin America; Gingivitis; Periodontitis; Risk factors.

Resumo: O objetivo do presente estudo foi identificar a presença das doenças periodontais e a importância relativa de reconhecidos fatores de risco nos países da América Latina. Foram encontradas informações esparsas, sendo que os dados se mostraram inconsistentes. Para a maioria dos países não existem informações. Gengivite é um achado universal na região, afetando todas as idades, independentemente do nível socioeconômico. A extensão da presença de sangramento pode variar entre 40% e 70% dos sítios. A prevalência de periodontite agressiva é maior na América Latina do que nos países industrializados. A prevalência varia de 0,3% a 4,5%, sendo que a forma localizada é a menos prevalente. A prevalência de periodontite crônica é alta e pode variar muito (40% a 80%) provavelmente em decorrência de diferenças na metodologia e nos critérios diagnósticos. Diferenças regionais podem ser relevantes. Os fatores de risco reconhecidos estão presentes nas populações estudadas. Fatores próprios como idade, gênero e genética foram relacionados em estudos brasileiros e chilenos. Tabagismo e diabetes demonstraram-se importantes fatores de risco. A importância do nível socioeconômico, ainda que presente como um fator de risco, é muito pouco relevada. Higiene oral é extremamente deficiente na região, embora seja um hábito cultural presente na maioria das populações. Pode-se concluir que as doenças periodontais apresentam alta prevalência nas populações da América Latina. Sua presença e extensão estão associadas a reconhecidos fatores de risco. Hábitos de higiene oral são deficientes. Estudos epidemiológicos com metodologia adequada e validade externa são necessários.

Descritores: Epidemiologia; América Latina; Gengivite; Periodontite; Fatores de risco.

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Introduction

Epidemiology is the study of health and disease in populations and how the different social strata are influenced by hereditary, biological, physical, environmental, and socioeconomic factors, as well as by individual behavior. Gingivitis is the most common form of periodontal disease and is found globally. Its prevalence and severity are less pronounced in industrialized, developed countries as compared to less developed regions.³ Gingivitis is less frequent in children, increases its occurrence in adolescents and young adults and tends to level in adulthood.⁴¹ The presence of supragingival biofilm is closely related with the presence of gingivitis. There are several modifiers to the expression of gingivitis: Medications associated with overgrowth such as cyclosporin, nifedipine or Dilantin; medical conditions such as diabetes and HIV-infection; behavioral factors such as smoking; and life conditions such as pregnancy or menopause. Until very recently the prevalent model for the pathogenesis of periodontal disease pointed out that not all sites with gingivitis developed into periodontitis.^{23,24} There are, however, indications that the long-term presence of gingivitis is associated with increased attachment loss.³⁹ In fact, gingivitis and periodontitis are considered a continuum of the same disease.²¹ These observations point out for a renewed interest in the preventive measures for gingivitis.¹³

Destructive forms of periodontal disease are rather infrequent in childhood, tend to increase during adolescence and present a steady increase with age.⁴ In the USA, 80% of the adult population present at least one tooth with clinical attachment loss (CAL) of 2 mm. If CAL of 4 mm is taken as a threshold, then this proportion falls to 50% and it is further reduced to less than 20% with a CAL of 6 mm.⁹ Periodontitis is present in higher proportions in developing countries and, within the same country, it affects higher proportions of the lower socioeconomic classes.^{1,3,25} Baelum⁵ (1998) showed that the prevalence of a CAL of 4 mm was approximately 90% in Kenya. Besides the geographical and socioeconomic variables, there are a number of other factors involved in the prevalence and severity of periodontitis. Non-modifiable risk factors include age,

gender, race-ethnicity, and gene polymorphisms. Environmental acquired and behavioral factors include socioeconomic factors, specific microbiota, cigarette smoking, alcohol consumption, diabetes, obesity, osteoporosis, HIV-infection, and psychosocial factors. Many of these and other factors are considered putative as they have not reached the level of evidence that will identify them as true risk factors. In the latter category, poorly controlled diabetes and smoking are accepted as true risk factors.^{8,19,50}

Epidemiology has helped to understand the multiple aspects of the interplay that characterizes periodontal healthy and diseased populations and individuals. New and more complex approaches in the study of periodontal diseases have relocated traditional clinical instruments of diagnosis, highlighting the importance of some indicators, such as clinical attachment loss, and giving new perspectives to others, such as periodontal probing.⁵⁰ On the other hand, recognizing several limitations of these diagnostic tools, new approaches have been proposed. Genetic markers, immune-inflammatory mediators, and specific bacteria are among those with promising results.⁵⁰

The aim of this paper was to analyze the epidemiological evidence concerning periodontal diseases available for Latin American populations in an attempt to picture particular characteristics for the area and to suggest alternatives for the prevention and treatment of these diseases in the region.

Epidemiology of periodontal diseases in Latin America Gingivitis

Gingivitis is a common observation in the studies analyzed. In 1998, the prevalence in Brazil was reported as high as 90% to 100% among children 7-14 years of age.¹² The mean gingival index (GI) was 1.24 irrespective of the socioeconomic background. The prevalence and severity of intense gingivitis increased with age in a study from Argentina with a large sample (2,279) of 8-9- and 12-13-year-old children. Only 2.7% of the 7-year-olds had intense gingivitis while 27.2% of the 14-year-olds were in this condition.¹⁴ In a study with a representative sample of the population of the metropolitan area of Porto

Alegre, Brazil, it was shown that nearly 100% of the population 14-30 years of age had marginal bleeding in approximately 70% of the teeth present.⁵²

Aggressive periodontitis

Gjeramo *et al.*¹⁷ (1984) examined bone loss in radiographs of 304 adolescents from a low socioeconomic area. The results showed that 28% had one or more sites with bone loss, and aggressive periodontitis was diagnosed in 2.6%. Albandar *et al.*² (1991) also used bitewing radiographs and reported a prevalence of 1.3% among a population of 13-year-old schoolchildren of high socioeconomic status in São Paulo, Brazil. Localized forms of aggressive periodontitis were diagnosed in only 0.3% (0.1-1.1 range) of 7,483 adolescents from three different Brazilian cities.⁴⁸ Lopez *et al.*²⁸ (1991) examined 2,500 children 15-19 years old and reported a prevalence of 0.32% of individuals with localized juvenile periodontitis in Chile. More recently, Lopez *et al.*³⁰ (2001) examined 9,203 subjects 12-21 years old. Establishing a CAL of 3 mm as a cut-off, the authors observed a 4.5% prevalence. Susin, Albandar⁴³ (2005) have shown a prevalence of 2.5% among 14-19-year-olds in Porto Alegre, Brazil, using as diagnostic criterion 4 or more teeth with a CAL \geq 4 mm.

Cortelli *et al.*¹¹ (2005) examined 25 aggressive periodontitis and 178 chronic periodontitis patients. The prevalence of *Porphyromonas gingivalis* was similar to that of other South American populations. The prevalence of *A. actinomycetemcomitans* and its highly leucotoxic subgroup was higher in Brazilians. Highly leucotoxic *A. actinomycetemcomitans* was more prevalent in aggressive periodontitis.

The prevalence of aggressive periodontitis in Brazil and Chile is somewhat higher than the estimates of the disease in most developing countries⁴ and is significantly higher than in developed countries.³⁵

Chronic periodontitis

The prevalence varied from 40% to 80% in the overall population. This variation can be associated with true regional differences, although great variations in methodology and sampling are present and may account for part of the differences observed. In a recent study, Susin *et al.*⁴⁴ (2004) examined the

prevalence and extent of CAL in adults from a representative sample of the metropolitan area of Porto Alegre, Brazil. Of the 853 people examined above the age of 30, 79% and 52% of the subjects and 36% and 16% of the teeth per subject had a CAL \geq 5 mm and \geq 7 mm, respectively. These results show a greater prevalence and severity of adult periodontitis in this population as compared to results reported for Brazil as a whole. It should be noted, however, that the surveys were based on different methodologies. The study by Susin *et al.*⁴⁴ (2004) performed a full mouth examination whereas the Brazilian census used the Community Periodontal Index (CPI). Regional and socioeconomic differences should also be kept in mind.

The prevalence of periodontitis in Porto Alegre was considerably higher than that reported in developed countries. In the United Kingdom, 42% of those with 35-44 years of age and 70% of those with 55-64 years of age are reported as showing a CAL \geq 4 mm.³³ In the Brazilian study, the corresponding figures were 92.2% and 99.2%. Similar differences can be detected when comparing these results and those reported in Chinese, Thai and Kenyan populations.^{6,7,10}

Lopez *et al.*²⁹ (2004) determined the characteristics of the subgingival microbiota of chronic periodontitis in Chileans residing in Santiago. Participated in the study 26 subjects (mean age 45 ± 7 years) with chronic periodontitis. Levels and proportions of 40 bacterial taxa using whole genomic DNA probes and checkerboard DNA-DNA hybridization were determined. Each of the individual test species was present in at least 25 of the 26 subjects, and 12 subjects (46.1%) harbored all 40 test species. Sixteen of the 40 species differed significantly between Chilean and U.S. subjects. The composition of the subgingival plaque differs among different subject populations. Thus, care should be taken when extrapolating the findings of one study to different ethnic groups.

The influence of risk factors

Age

Several studies in Latin America show that age is an important factor in the prevalence and severity

of periodontitis.¹⁸ In a recent epidemiological survey performed by the Brazilian Government, the prevalence of severe disease jumped from 1.34% in young adults to 9.98% in the adult population.³¹ Interestingly, it drops to 6.3% above 60 years of age. This, however, is explained by the high rate, 60.8%, of edentulous subjects in this age group. Similar trend is also seen in surveys from Argentina and Chile.¹⁶ It is still unclear whether age increases the risk of periodontitis or this relationship is mainly a consequence of the cumulative effect characteristic of the disease.⁸

Gender

Susin *et al.*⁴⁴ (2004) have shown a higher risk of developing periodontal diseases among males (relative risk ratio = 1.6). The relationship is often attributed to worse oral hygiene and smoking habits. Although gender-specific differences in the immune-inflammatory response to bacterial challenges in periodontitis have not been shown, it is plausible that such differences, in fact, exist.⁸

Race/ethnicity

Race is a common factor associated with periodontal diseases in the United States. In Latin America, this issue is not regarded with the same level of interest. Susin *et al.*⁴⁷ (2005) observed that self-reported non-whites were associated with a higher risk of pocket depth (PD) \geq 5 mm than whites. The same group, however, could not find a correlation between these variables and attachment loss.⁴² There are serious questionings on the definition of race by an individual's color of the skin. Besides that, it is a well established fact that race/ethnicity belongs to the social construct and is often mingled with socioeconomic settings. It is doubtful that the diagnostic instruments presently used in periodontal epidemiology can measure the impact of this association.⁸

Genetics

The field of genetics and specially that of genetic polymorphisms in relation to susceptibility to periodontal disease has evolved tremendously in the past few years. The influence of familiar characteristics in the history especially of aggressive forms of peri-

odontitis has been known for a long time. It is calculated that around 50% of the periodontal disease present in a population may be explained through genetic determinants. Few studies have addressed polymorphisms in relation to susceptibility to periodontal disease in Latin America and they present inconsistent results.

Trevilatto *et al.*⁵¹ (2002), in a case report, examined polymorphisms and periodontal status within a family where some of the 14 members presented aggressive periodontitis. No correlation was found between polymorphism and periodontal disease status among the members of the family. Pontes *et al.*³⁶ (2004) evaluated if interleukin 4 (IL-4) polymorphisms were associated with periodontal disease in a Brazilian population of African heritage. Sixty patients were divided into two groups: A periodontitis group (n = 30) and a control group (n = 30). No significant differences were found in the genotype frequency of the polymorphisms between the control and periodontitis groups.

Scarel-Caminaga *et al.*³⁸ (2003) evaluated the relationship between the polymorphism in the IL4 gene and the different levels of chronic periodontal disease. DNA was extracted from buccal epithelial cells of 113 unrelated adult individuals with different levels of periodontitis. No significant differences in the allele and genotype frequencies of the polymorphism were found between control and groups with periodontal disease.

Quappe *et al.*³⁷ (2004) investigated the association of the interleukin-1 gene polymorphisms with aggressive periodontitis (AgP). Thirty-six patients with AgP, 75 healthy controls, and 75 subjects of unknown periodontal status (reference population) were genotyped for the IL-1A-889 and IL-1B+3954 loci. The results of this study support a positive association only between AgP and the presence of the IL-1B +3954 allele 2 polymorphism.

The same Chilean group recently published a study to determine the prevalence of the IL-1A-889 and IL-1B+3954 polymorphisms in Chileans and their association with periodontitis. Subjects aged 20 to 48 were selected comprising 330 cases and 101 healthy controls. Cases were categorized as having initial, moderate, or severe periodontitis accord-

ing to the percentage of sites with clinical attachment loss ≥ 3 mm. Genomic DNA was analyzed for polymorphism in the IL-1A gene at site-889, and IL-1B gene at site +3954 by polymerase chain reaction (PCR). The results showed that individuals carrying the positive genotype have significantly greater risk for developing periodontitis.²⁶

Most of these cross-sectional studies show a positive correlation between the genetic polymorphism and the periodontal state of the populations. The results should be interpreted with caution as the strength of the association is often weak, several confounding factors are not considered, the number of participants is often small, levels of disease are not well defined and differences in the effect of different polymorphisms can be expected although unknown up to now.

Socioeconomic status (SES)

A great number of the studies performed in Latin America have addressed the issue of socioeconomic status as a risk factor for periodontal disease.¹⁸ It is not surprising that many of them have shown positive correlations. Periodontal diseases, as most diseases, carry a social determinant that is often underestimated through statistical corrections as if they could erase it as a significant factor in the establishment and progression and also as a determinant of access to adequate prevention and treatment. Typically, a recent systematic review found approximately as many studies showing socioeconomic levels as risk factors as studies not showing it.²² The authors concluded that socioeconomic variables associated with periodontal disease appear to be of less importance than smoking. However, Sheiham, Nicolau⁴⁰ (2005) observed that there is a strong gradient in periodontal disease and it is socially patterned with an additive effect of low socioeconomic conditions across the life course of periodontal diseases. The authors observed that as SES increases, the amount of plaque and gingival bleeding, and the proportion of individuals with periodontal diseases decreases.

In the Brazilian national survey of 1986³², the results showed a tendency of higher prevalence of periodontitis among lower class, middle-aged subjects (5.6%) as compared to higher social class indi-

viduals (5.5%). In a more recent survey published in 2004, there was a clear difference in the prevalence of periodontitis between country-side small villages and larger cities.³¹

Rather clearer differences were observed in the population of Santiago, Chile, where 98% of middle-aged subjects of the low social strata presented CPITN scores 3 or 4 whereas only 56% of subjects in the same age group but belonging to the higher social classes were in this condition.¹⁵ Similar results were observed in a study dealing with younger Chileans.²⁷ Besides that, the prevalence of aggressive periodontitis was shown in an earlier study to be heavily concentrated in lower classes.²⁸

Tobacco smoking

Few studies have addressed in a representative sample of the population the issue of smoking and periodontal disease. This is particularly true for Latin America. Smoking was significantly associated with clinical attachment loss in the Brazilian population, across different age groups. Compared to non-smokers, moderate smokers were 2 times more likely to have moderate CAL, and 3 times more likely to have severe CAL. Heavy smokers were 3 times more likely to have moderate CAL, and 8 times more likely to have severe CAL than non-smokers.⁴²

In the age group 14-29 years, moderate/heavy smokers were 2 times more likely to have localized recession ≥ 1 mm, and 3.8 times more likely to have generalized recession ≥ 1 mm than non-smokers. In the age group 30+ years, smokers were to some extent more likely to have localized recession ≥ 3 mm, and were significantly (Relative Risk Ratio = 3.0) more likely to have generalized recession ≥ 3 mm than non-smokers.⁴⁵

In a study where the attributable risk of smoking was calculated, it was demonstrated that if moderate and heavy smokers had not smoked, these groups would have respective reductions of 28% and 48% in the prevalence of subjects with 30% or more teeth with CAL ≥ 5 mm. Furthermore, the estimated reduction in the total population would be approximately 12%, or nearly 90,000 potential cases.⁴⁶

In conclusion, it can be stated that cigarette smoking represents a risk factor for the progression

of periodontitis, the effect of which may be dose-related. Heavy-smokers should be considered as high-risk individuals. The clinical implications are that smokers should be identified during patient examination and efforts should be made to cease the habit.^{19,20}

Diabetes

Little is known of the impact of diabetes *mellitus* on periodontal health in Latin American individuals. In a case-control study, it was shown that insulin-dependant diabetic cases had greater alveolar bone loss than matched controls.³⁴ Tomita *et al.*⁴⁹ (2002) observed that Japanese-Brazilians with insulin-independent diabetes presented a higher prevalence of CAL > 6 mm than individuals with normal glucose levels. The differences, however, were not significant. Susin *et al.*⁴⁴ (2004) showed that individuals who self-reported having diabetes were 3.3 times more likely to have severe CAL than non-diabetics. However, this association was reduced in the multivariable model, and the effect was no longer statistically significant. The prevalence of diabetics

may have been underestimated in this study due to the method of identification and possibly because low socioeconomic subjects may not be aware of their condition.

Conclusion

There are many potential areas of development in descriptive, analytical and clinical epidemiology research in Latin America. Unfortunately, epidemiological research is greatly underestimated. Too many studies and too much effort are placed in inadequate designs, with unsatisfactory methods resulting in invalid evidence. Epidemiological research is badly needed for public health planning, for the understanding of both the onset and progression of periodontal disease and for testing preventive and therapeutic measures directed towards the population and individual patients. The increasing relevance of periodontal problems in tooth loss in a rapidly ageing population, not to mention the role of these diseases in the individual's well being, warrants all efforts in giving priority to this type of research in Latin America.

References

1. Albandar JM, Brunelle JA, Kingman A. Destructive periodontal disease in adults 30 years of age and older in the United States, 1988-1994. [published *erratum* in J Periodontol. 1999;70(3):351]. J Periodontol. 1999;70(1):13-29.
2. Albandar JM, Buischi YA, Barbosa MF. Destructive forms of periodontal disease in adolescents. A 3-year longitudinal study. J Periodontol. 1991;62(6):370-6.
3. Albandar JM, Rams TE. Global epidemiology of periodontal diseases: an overview. Periodontol 2000. 2002;29:7-10.
4. Albandar JM, Tinoco EM. Global epidemiology of periodontal diseases in children and young persons. Periodontol 2000. 2002;29:153-76.
5. Baelum V. The epidemiology of destructive periodontal disease: Causes, paradigms, problems, methods and empirical evidence [Doctorate Thesis]. Aarhus: Royal Dental College, Faculty of Dentistry of University of Aarhus; 1998.
6. Baelum V, Fejerskov O, Manji F. Periodontal diseases in adult Kenyans. J Clin Periodontol. 1988;15(7):445-52.
7. Baelum V, Pisuthanakan S, Teanpaisan R, Pithpornchaiyakul W, Pongpaisal S, Papapanou PN *et al.* Periodontal conditions among adults in Southern Thailand. J Periodontol Res. 2003;38(2):156-63.
8. Borrell RN, Papapanou PN. Analytical epidemiology of periodontitis. J Clin Periodontol. 2005;32 Suppl 6:132-58.
9. Brown LJ, Oliver RC, L  e H. Periodontal diseases in the U.S. in 1981: prevalence, severity, extent, and role in tooth mortality. J Periodontol. 1989;60(7):363-70.
10. Corbet EF, Wong MC, Lin HC. Periodontal conditions in adult Southern Chinese. J Dent Res. 2001;80(5):1480-5.
11. Cortelli JR, Cortelli SC, Jordan S, Haraszthy VI, Zambon JJ. Prevalence of periodontal pathogens in Brazilians with aggressive or chronic periodontitis. J Clin Periodontol. 2005;32(8):860-6.
12. Cunha ACR, Chambrone LA. Preval  ncia de gengivite em crian  as de um n  vel social baixo. Rev Periodontia. 1998;7(1):6-10.
13. Davies I, Karring T, Norderyd O; European Workshop in Periodontology group E. Advances in the behavioral and public health aspects of periodontitis. Group E consensus report of the fifth European workshop in Periodontology. J Clin Periodontol. 2005;32(Suppl 6):326-7.
14. De Muniz BR. Epidemiologic oral health survey of Argentine children. Community Dent Oral Epidemiol. 1985;13(6):328-33.

15. Gamonal JA, Lopez NJ, Aranda W. Periodontal conditions and treatment needs, by CPITN, in the 35-44 and 65-74 year-old population in Santiago, Chile. *Int Dent J*. 1998;48(2):96-103.
16. Gasparini DO, Buri MH. Determinacion de las necesidades terapeuticas periodontales en la comunidad de Corrientes, Republica Argentina. *Rev La Asociación Odontol Argentina*. 1998;86(4):349-54.
17. Gjermo P, Bellini HT, Pereira Santos V, Martins JG, Ferracyoli JR. Prevalence of bone loss in a group of Brazilian teenagers assessed on bite-wing radiographs. *J Clin Periodontol*. 1984;11(2):104-13.
18. Gjermo P, Rosing CK, Susin C, Oppermann RV. Periodontal diseases in Central and South America. *Periodontol 2000*. 2002;29:70-8.
19. Heitz-Mayfield LJ. Disease progression: identification of high-risk groups and individuals for periodontitis. *J Clin Periodontol*. 2005;32(Suppl 6):196-209.
20. Hujuel PP, Del Aguila MA, DeRouen TA, Bergstrom J. A hidden periodontitis epidemic during the 20th century? *Community Dent Oral Epidemiol*. 2003;31(1):1-6.
21. Kinane DF, Attstrom R. Advances in the pathogenesis of periodontitis. Group B consensus report of the fifth European workshop in Periodontology. *J Clin Periodontol*. 2005;32(Suppl 6):130-1.
22. Klinge B, Norlund A. A socio-economic perspective on periodontal diseases: a systematic review. *J Clin Periodontol*. 2005;32(Suppl 6):314-25.
23. Lindhe J, Okamoto H, Yoneyama T, Haffajee A, Socransky SS. Longitudinal changes in periodontal disease in untreated subjects. *J Clin Periodontol*. 1989;16(10):662-70.
24. Loe H, Anerud A, Boysen H, Morrison E. Natural history of periodontal disease in man. Rapid, moderate and no loss of attachment in Sri Lankan laborers 14 to 46 years of age. *J Clin Periodontol*. 1986;13(5):431-45.
25. Loe H, Brown LJ. Early onset periodontitis in the United States of America. *J Periodontol*. 1991;62(10):608-16.
26. Lopez NJ, Jara L, Valenzuela CY. Association of interleukin-1 polymorphisms with periodontal disease. *J Periodontol*. 2005;76(2):234-43.
27. Lopez NJ, Rios V, Fernandez O. Periodontal conditions in 15-19 year-old Chileans. *Int Dent J*. 1996;46(3):161-4.
28. Lopez NJ, Rios V, Pareja MA, Fernandez O. Prevalence of juvenile periodontitis in Chile. *J Clin Periodontol*. 1991;18(7):529-33.
29. Lopez NJ, Socransky SS, Da Silva I, Japlit MR, Haffajee AD. Subgingival microbiota of Chilean patients with chronic periodontitis. *J Periodontol*. 2004;75(5):717-25.
30. Lopez R, Fernandez O, Jara G, Baelum V. Epidemiology of clinical attachment loss in adolescents. *J Periodontol*. 2001;72(12):1666-74.
31. Ministério da Saúde do Governo Brasileiro. Coordenação de Saúde Bucal. Projeto SB Brasil 2003. Condições de saúde bucal da população brasileira 2002-2003 – Resultados principais. Brasília; 2004.
32. Ministério da Saúde do Governo Brasileiro. Levantamento epidemiológico em saúde bucal: Brasil, zona urbana, 1986. Brasília: Centro de Documentação do Ministério da Saúde; 1988.
33. Morris AJ, Steele J, White DA. The oral cleanliness and periodontal health of UK adults in 1998. *Br Dent J*. 2001;191(4):186-92.
34. Novaes Junior AB, Pereira AL, de Moraes N, Novaes AB. Manifestations of insulin-dependent diabetes *mellitus* in the periodontium of young Brazilian patients. *J Periodontol*. 1991;62(2):116-22.
35. Papapanou PN. Periodontal diseases: epidemiology. *Ann Periodontol*. 1996;1:1-36.
36. Pontes CC, Gonzales JR, Novaes AB Jr, Junior MT, Grisi MF, Michel J. Interleukin-4 gene polymorphism and its relation to periodontal disease in a Brazilian population of African heritage. *J Dent*. 2004;32(3):241-6.
37. Quappe L, Jara L, Lopez NJ. Association of interleukin-1 polymorphisms with aggressive periodontitis. *J Periodontol*. 2004;75(11):1509-15.
38. Scarel-Caminaga RM, Trevilatto PC, Souza AP, Brito RB Jr, Line SR. Investigation of IL4 gene polymorphism in individuals with different levels of chronic periodontitis in a Brazilian population. *J Clin Periodontol*. 2003;30(4):341-5.
39. Schatzle M, Loe H, Burgin W, Anerud A, Boysen H, Lang NP. Clinical course of chronic periodontitis. I. Role of gingivitis. [published *erratum* in *J Clin Periodontol*. 2004;31(9):813]. *J Clin Periodontol*. 2003;30(10):887-901.
40. Sheiham A, Nicolau B. Evaluation of social and psychological factors in periodontal disease. *Periodontol 2000*. 2005;39:118-31.
41. Stamm JW. Epidemiology of gingivitis. *J Clin Periodontol*. 1986;13(5):360-70.
42. Susin C. Periodontal diseases in a representative urban population in South Brazil [Doctorate Thesis]. Norway: Faculty of Dentistry, University of Bergen; 2004.
43. Susin C, Albandar JM. Aggressive periodontitis in an urban population in southern Brazil. *J Periodontol*. 2005;76(3):468-75.
44. Susin C, Dalla Vecchia CF, Oppermann RV, Haugejorden O, Albandar JM. Periodontal attachment loss in an urban population of Brazilian adults: effect of demographic, behavioral, and environmental risk indicators. *J Periodontol*. 2004;75(7):1033-41.
45. Susin C, Haas AN, Oppermann RV, Haugejorden O, Albandar JM. Gingival recession: epidemiology and risk indicators in a representative urban Brazilian population. *J Periodontol*. 2004;75(10):1377-86.
46. Susin C, Oppermann RV, Haugejorden O, Albandar JM. Periodontal attachment loss attributable to cigarette smoking in an urban Brazilian population. *J Clin Periodontol*. 2004;31(11):951-8.

47. Susin C, Valle P, Oppermann RV, Haugejorden O, Albandar JM. Occurrence and risk indicators of increased probing depth in an adult Brazilian population. *J Clin Periodontol*. 2005;32(2):123-9.
48. Tinoco EM, Beldi MI, Loureiro CA, Lana M, Campedelli F, Tinoco NM *et al*. Localized juvenile periodontitis and *Actinobacillus actinomycetemcomitans* in a Brazilian population. *Eur J Oral Sci*. 1997;105(1):9-14.
49. Tomita NE, Chinellato LE, Pernambuco RA, Lauris JR, Franco LJ; Grupo de Estudo Diabetes em Nipo-Brasileiros. [Periodontal conditions and diabetes *mellitus* in the Japanese-Brazilian population] [Article in Portuguese]. *Rev Saude Publica*. 2002;36:607-13.
50. Tonetti MS, Claffey N; European Workshop in Periodontology group C. Advances in the progression of periodontitis and proposals of definitions of a periodontitis case and disease progression for use in risk factor research. Group C consensus report of the 5th European Workshop in Periodontology. *J Clin Periodontol*. 2005;32(Suppl 6):210-3.
51. Trevilatto PC, Tramontina VA, Machado MA, Gonçalves RB, Sallum AW, Line SR. Clinical, genetic and microbiological findings in a Brazilian family with aggressive periodontitis. *J Clin Periodontol*. 2002;29(3):233-9.
52. Valle P. O fumo como indicador de risco à periodontite em adolescentes e adultos jovens [Dissertação de Mestrado]. Canoas: Lutheran University of Brasil; 2002.