

Periodontal Diseases in a Representative Urban Population in Southern Brazil

Cristiano Susin

Doctor Odontologiae
Thesis



October 2004
Bergen
Norway





Periodontal Diseases in a Representative Urban Population in South Brazil

October 2004
Bergen
Norway

Cristiano Susin

A thesis submitted in partial fulfillment of the requirements for the degree of Doctor Odontologiae at the University of Bergen, Norway

ISBN 82-8006-018-9
Bergen, Norway 2004

I would like to dedicate this thesis to my grandmother who left us some days before I began my doctorate. She never understood “why her once smart grandson was still studying”

You are always with me

“Some people believe in fate, others don’t. I do and I don’t. It may seem at times as if invisible fingers move us about like puppets on strings. But for sure, we are not born to be dragged along. We can grab the strings ourselves and adjust our course at every crossroad, or take off at any little trail into the unknown.”

Thor Heyerdahl, 1984.

PREFACE

The best way to understand a person's work is to learn something about his life. Life has made me a storyteller, and those that know me best know that I cannot miss an opportunity to tell another one. This is my own story ...

... when I was 6 years-old I got a chemistry lab, which was quite an accomplishment for a child living in a small city in southern Brazil in the middle of the 70's. After some days following the instructions and formulas given by the kit, I become bored and started trying my own formulas. After staining many clothes and blowing up my right hand fingers (my fate as a dentist could have been decided there) I decided to become a "scientist" (at that time scientists were those guys in white coats that work in a lab with glass tubes and computers). I started my career by disassembling my father's beloved radio, which I never managed to reassemble again. Nevertheless, it was still working, and much lighter.

My first "real" experiment was during a high school contest. Together with Lisiane (my high school sweet heart) and another friend, we tried to prove that global warming was caused by smoke (very easy subject for a first project). We used a small box filled with smoke and covered it with a plastic film. After exposing it to a strong light the temperature inside the box raised more when it was filled with smoke than when it was not. I have to admit that the temperature was not recorded in an unbiased fashion, and that the smoke was not applied randomly. Nevertheless, we managed to prove our point and won the prize.

In Dental School I was part of a Special Training Program and our first project was a split-mouth double-blind crossover randomized placebo-controlled trial (very easy design for a first research project). This was the time when my interest in methodology and biostatistics started, especially after I realized that very few people could make sense of the results of a study. After that we began a study in dogs where 4 different antimicrobial treatments were applied locally in the periodontal pockets. Unfortunately, the histological preparation went wrong and we lost all specimens. As "wannabe" researchers this was a huge blow to our confidence. As a backup plan we started to work with toxicity of mouthwash products using a rat model. Meanwhile some students in the laboratory next door were performing stress-related research, and I was amazed by the way they were inducing stress and depression. This experience would be very important for my Master studies when after several months into the program I was the only student without a project. With this idea in mind, we designed a long and very cumbersome 4 months study, which consisted of daily sessions (including Saturdays, Sundays and holidays) lasting for several hours trying to induce stress by very unusual means in more than 60 rats. Amazingly enough that was when I started to like epidemiology, in particular the study of risk factors (perhaps hoping that it would be less stressful). As a first idea for my Doctorate I wanted to use a convenience sample to study a few risk factors for periodontal disease. I had no idea what I would end up doing.

During my doctorate I have had a lot of *déjà vu*. I have done a lot of unusual things in my life, and my father has a great deal of participation in many of them. I got my first job working as an office boy when I was thirteen years old. That is when I learned how to chase (and I really mean it) people that did not want to pay their bills. It turned out to be an invaluable skill that helped me to overcome the nonresponse of this study. Later on, when I was sixteen my father gave me the chance to be a truck driver for several months (of course I didn't have a driver's license at that time). The resemblance between the truck and our mobile examination unit is obvious. Meanwhile I had been promoted to a less "exciting", but more serious job; and my duty was to enter endless codes and values into a computer. Any similarity with typing the study data is not mere coincidence. After parking the mobile examination unit for the very first time, and asking people to participate, provide light, water, and allow us to use their bathrooms (you only remember it is important when you don't have it) I felt I was back to my teens. I used to participate in charitable campaigns asking for donations in my hometown. The main difference was that at that time I didn't used to poke a periodontal probe in 169 different sites in the mouth of those willing to participate.

Some moments will always be remembered:

- The first time I had to park the mobile examination unit. It took me probably more than 20 minutes to fit it in a space where at least 3 or 4 units would fit.
- The first time we had to assemble the plastic cover extension of the trailer to provide shelter from a thunderstorm. We weren't fast enough and unfortunately we lost some participants.
- The trailer's first blowout tire. We had to transport the repairman in the open trunk of my car for many blocks because we couldn't fit him and his tools inside.

- The nun who thought we were very suspicious people, and started a campaign in the neighborhood asking people not to participate in the survey.
- The dentist that thought we were providing unauthorized cheap treatment and threatened to sue us.
- The arrest of a drug dealer a few meters from our trailer.
- The lady that I drove to her children's school in order to enable her to have time to participate in our survey.
- And, finally on the last day of field work; we had one of the biggest thunderstorms I have ever seen in my life and yet it felt like a nice and calm spring day.

The following lines will remain in our memories, even though I cannot quite understand some of them:

Patricia, a hundred times: "Why are these sectors close to each other?" (the explanation that the sectors were by study design clustered never satisfied her)

Patricia, also a hundred times: "Oh!!!! Look at my fingernails!!!!!" (no comments)

Alex to an elderly patient: "Open wide, like a crocodile!!!!!" (he always wanted to be a pediatric dentist)

Alex after the release of his band album debut: "Do you want to buy a CD of the Colored Universe band?"

Caroline presenting her Master degree proposal: "... SECOVI ... PT ... gordinhos (obese individuals) ..." (I can still remember Rui's face that day)

Caroline trying to have lunch during the fieldwork: "Do you still have chicken breasts?" Waiter's answer: "I'm sorry, but we don't have chicken anymore because the blond girl that works with you has eaten it all." (by the way, the blond girl was Patricia).

The very first days of my journey were unforgettable. I almost had a heart attack when I found out that Lisiane was flying with me to Sao Paulo in some sort of farewell trip. A secret that everybody, except me, knew about. After arriving in London I found out that my baggage was missing. Unfortunately, most of my money was in my suitcases (yes, I know: carry it with you at all times). When I tried to change airports the fire alarm went off and I was stuck inside the airport. When I finally managed to get out of the airport the bus ticket that I should have used to transfer between airports disappeared and my credit card didn't work when I tried to buy a new ticket (have you heard about Murphy's law). I obviously lost my connection flight. To make a long trip a short story, I arrived in Bergen on a rainy day (what's new). I had to fight for a room in Fantoft student house using my Portu-English, and I had to spend the first week using the same cloths and without bed covers or pillows. That was quite a start.

Someone could argue that these remarks do not belong in a PhD thesis and that these stories have nothing to do with serious research. But, I can assure you that this is what it is all about.

Well, 200 field working days, 20,000 km driving a mobile examination unit, 1,600 interviews and examinations, 5+ papers and 4 years later I am about to finish another chapter of my life. I'm not sure if I have accomplished my childhood dream, but hopefully I'm closer now than I was before.

What wonderful days.
Cristiano, 2004.

ACKNOWLEDGEMENTS

This work was carried out during the period of August 2000 to October 2004 partly in Porto Alegre, Brazil; Bergen, Norway; and Philadelphia, U.S.A.; in many different academic and non-academic settings, therefore it is impossible to express my thanks to all of those that have made this work possible. CAPES Foundation from the Brazilian Ministry of Education, for which I am most grateful, supported my studies. The Lutheran University of Brazil provided early support as well.

During the early process of getting my scholarship some people were very important. I would like to express my warm thanks to Maria Antonieta Lopes de Souza and Marisa Maltz.

The fieldwork would not have been possible if we did not have our mobile examination unit, for that I'm thankful to SESI. The training of the examiners of this study were performed at the Lutheran University of Brazil and at the Federal University of Rio Grande do Sul. Most of my credit-courses were taken at the Faculty of Dentistry and Center for International Health at the University of Bergen, and at Temple University School of Dentistry, thus I want to express my appreciation to the Faculty of these institutions.

I would like to express my deep emotion to those anonymous people that helped us during the fieldwork. People that did not know us, but that opened their homes and tried to make our life easier. Thank you very much for the glasses of water and lemonade, access to the bathroom and to parking places for our trailer, words of encouragement and nice chats.

My special thanks are given to the trailer adventurers who dared to participate in this journey. First of all to my colleagues Pop (Alex Haas), Nini (Caroline Dalla Vecchia) and Pati (Patricia Valle) for their extraordinary contribution and deep commitment to our work. To our assistants Anna and Jooice for their extraordinary work. Marcia for her help in the beginning of the fieldwork. Without your patience and hard work we would never have finished the endless data collection.

I want to express my appreciation to Drs Pantelis Rados and Manoel Santana from the Department of Oral Pathology at UFRGS for their support with our patients in need of special oral diagnosis.

I want to thank Gry Kibsgaard, Marit Norbert, Linda Forshaw, and Torgunn Haar for the ever-prompt help.

The boundaries of my learning process were gratefully increased thanks to the insightful discussions about periodontal biology with Dr. Wikesjö. I also learned that statistics is only necessary when "you hold a histologic section against the light and you cannot see the differences".

I want to express my deep respect and appreciation for Dr. Kingman's collaboration and help. Our insightful discussions about statistics and your "tricky quizzes" will always be remembered.

I am in great debt with Dr. Rams for all the support and opportunities at the Department of Periodontology at Temple University School of Dentistry.

I would like to express my deep gratitude and admiration to my advisors Jasim, Ola and Rui. Jasim's friendship and support during my stay in Philadelphia, endless patience with my Brazilian way of writing, and commitment to my work were essential. Ola's nice talks and wise advices were extremely important during the hard times, and our discussions about epidemiology were always inspiring. Rui's friendship, encouragement and never-ending support during my whole PhD were fundamental.

To my "financial manager" Maria Tereza Martins Fasolo. For the countless times I asked one more favor. Your support was always very important.

To my friends Cassiano Rösing, Marios Epaminondas and Vinicius Dutra, who shared many good and bad moments of my PhD life and always gave me confidence to continue. To all my friends, and in special to Andre, for the innumerable encouragement phone calls and emails.

To my parents Osvaldo and Sonia my deepest love and respect. My father's perseverance and sense of equality, and my mother's sensibility and kindness were the cornerstones of my long journey.

Finally, I want to express my love to Lisiane, who has embraced my difficult way of life for so long. We almost lost ourselves during my journey, but we did find ourselves in love.

Thank you all. Tusen takk. Muito obrigado.

Periodontal Diseases in a Representative Urban Population in South Brazil

Cristiano Susin
Faculty of Dentistry
University of Bergen
ISBN: ISBN 82-8006-018-9

Abstract.

Background and Aims: There is little information about the epidemiology and risk factors of periodontal diseases in Latin America in general, and Brazil in particular. The principal aims of this study were to: 1) describe the prevalence and severity of periodontal attachment loss and gingival recession, and to assess the contribution of demographic, behavioral, and environmental exposures to the occurrence of periodontal disease outcomes in a sample representative of the urban population in the state of Rio Grande do Sul in south Brazil; and 2) report the epidemiology and risk indicators of aggressive periodontitis in this population.

Methods: A representative sample consisting of 1,586 subjects 14-103 years of age (mean 38 y) and comprising 45.3% males and 54.7% females was selected using a multi-stage, probability, cluster sampling strategy. The subjects were interviewed using a structured questionnaire and underwent a full-mouth, six sites per tooth clinical examination in a mobile examination center.

Results: Moderate and severe clinical attachment loss and gingival recession were widespread among adults in this population. The prevalence and extent of attachment loss ≥ 5 and ≥ 7 mm were 79% and 52% subjects, and 36% and 16% teeth; and for gingival recession ≥ 3 mm and ≥ 5 mm were 52% and 22% subjects, and 17% and 6% teeth, respectively. Aggressive periodontitis was diagnosed in 5.5% of subjects, which is significantly higher than the reported prevalence in most other populations. Among the main risk indicators for chronic as well as aggressive destructive periodontal diseases were: older age, low socioeconomic status, dental calculus, and smoking. Cigarette smoking accounted for an important part of periodontal disease burden, particularly in adults, and should be considered an important target in any prevention strategy aimed at reducing the burden of periodontal diseases. Partial recording methods consistently underestimated the prevalence of attachment loss in the population, and the extent of underestimation was dependent on the type of system used and the threshold of attachment loss.

Conclusions: Destructive periodontal diseases are prevalent in this Brazilian population. Suitable disease prevention and health promotion programs should be established to improve the periodontal health in this population.

Keywords: periodontal diseases, periodontal attachment loss, epidemiology, risk factors, cigarette smoking, partial recording

LIST OF PAPERS

The present thesis is partially based on the following articles, which are referred to in the text by their Roman numbers:

Paper I: Susin C, Dalla Vecchia CF, Oppermann RV, Haugejorden O, Albandar JM. Periodontal attachment loss in an urban population of Brazilian adults, and the effect of demographic, behavioral and environmental risk indicators. *J Periodontol* 2004;75: 1041-1049.

Paper II: Susin C, Oppermann RV, Haugejorden O, Albandar JM. Periodontal attachment loss attributable to cigarette smoking in an urban Brazilian population. *J Clin Periodontol* 2004;32: doi: 10.1111/j.1600-051X.2004.00588.x.

Paper III: Susin C, Kingman A, Albandar JM. Effect of Partial Recording Protocols on Estimates of Prevalence of Periodontal Disease. *J Periodontol* 2005;76: (in press).

Paper IV: 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;75:1383-1392.

Paper V: Susin C, Albandar JM. Early-onset aggressive periodontitis in an urban population in South Brazil. *J Periodontol* 2005;76: (in press).

ABBREVIATIONS

AF	Attributable fraction
AgP	Aggressive periodontitis
CI	Confidence interval
CPITN	Community Periodontal Index of Treatment Needs
CPI	Community Periodontal Index
IBGE	Brazilian Institute of Geography and Statistics
METROPLAN	Rio Grande do Sul State Agency for Metropolitan Affairs
NHANES I	The First National Health and Nutrition Examination Survey (U.S., 1971-1974)
NHANES III	The Third National Health and Nutrition Examination Survey (U.S., 1988-1994)
OR	Odds ratio
p	Probability value
CAL	Clinical attachment level
PAF	Population attributable fraction
PPD	Probing pocket depth
PRP	Partial recording protocols
PSU	Primary sampling units
RRR	Relative risk ratio
WHO	World Health Organization

CONTENTS	page
1) Introduction	1
1.1) Epidemiology of Periodontal Diseases	1
1.1.1) Periodontal Diseases in Adults	1
1.1.2) Periodontal Diseases in Young Subjects	2
1.2) Partial Recording Protocols	2
1.3) Risk Factors and Risk Indicators	3
1.3.1) Eras and Paradigms	3
1.3.2) Definitions	3
1.3.3) Causality and Confounding in Risk Assessment	3
1.3.4) Risk Factors and Risk Indicators for Periodontal Diseases	4
Chronic Periodontal Disease in Adults	4
Aggressive Periodontitis	5
Gingival Recession	5
1.3.5) Attributable Fraction	6
2) Aims	7
3) Materials and Methods	9
3.1) Population	9
3.2) Sample Size	10
3.3) Study Design	10
3.4) Study Sample	11
3.5) Interview and Clinical Examination	11
3.6) Assessment of Measurement Error	12
3.7) Ethical Considerations	13
3.8) Data Management	13
3.8.1) Data entry	13
3.8.2) Dependent Variables	13
3.8.3) Independent Variables	14
3.8.4) Partial Recording	15
3.9) Statistical Methods	15
4) Results and Comparisons with Previous Surveys	17
4.1) Clinical Attachment Loss, Age Group 30 Years and Older	17
4.1.1) Overall Estimates	17
4.1.2) Effect of Partial Recording Protocols on Prevalence of Clinical Attachment Loss	18
4.1.3) Risk Assessment	18
4.2) Gingival Recession	21
4.2.1) Overall Estimates	21
4.2.2) Risk Assessment	21
4.3) Aggressive Periodontitis, Age Group 14-29 Years	22
4.3.1) Overall Disease Estimates	22
4.3.2) Risk Assessment	23
5) Discussion	25
6) Concluding Remarks	27
References	29
Appendix I: Quality Assurance	39
Appendix II: Non-response and Missing Information	41
Appendix III: Sampling Weight and Design Effect	45

Periodontal diseases are a group of inflammatory diseases that affect the supporting tissues of teeth and share common clinical manifestations. Periodontal diseases may be divided into non-destructive and destructive forms.³² Gingivitis is a non-destructive infectious disease and is characterized by inflammation of the soft tissues surrounding the teeth, which appears clinically as a change in tissue color and consistency, and can be associated with swelling and a tendency to bleed upon slight provocation.¹⁵⁹ Periodontitis is a destructive infectious disease in which there is inflammation of the periodontal soft and hard tissues resulting in apical migration of the epithelial attachment, periodontal attachment and alveolar bone loss.⁸⁴ Sites with periodontitis are characterized with bleeding on probing and increased probing depth, and may also show gingival recession.

Periodontitis may occur at any age after eruption of teeth, and may affect the permanent as well as the deciduous dentition. However, the disease is most common during adulthood and old age. Certain distinctive forms of periodontitis occur in children and young adults and are characterized by a high rate of periodontal tissue loss and other characteristic features.^{21,57,200,232}

Periodontal diseases are infectious in nature, and bacteria are believed to play a major role in their initiation and progression.^{82,143,148,156,15,185,211} The current understanding of the pathogenesis of periodontal diseases suggests that these diseases occur as a result of complex interactions between periodontopathic microorganisms and the host tissues.^{1,135} This process is modified by the status of the host immune system,^{1,135} genetic factors,^{103,107} and a complex array of environmental exposures.^{13,16} Recently, it has been hypothesized that also certain viruses may play a role in the pathogenesis of chronic and aggressive periodontitis.^{64,182,213}

In its later stages, periodontitis may compromise oral function and esthetics, and may also lead to tooth loss. Periodontitis is an important cause of tooth loss in developing^{59,176,237} and developed countries.^{166,233} Furthermore, there are several studies showing significant association of periodontitis with certain systemic diseases,

including cardiovascular diseases,^{47,93} low birth weight syndrome,^{123,151} and bacterial respiratory diseases.^{118,196} These findings have generated the hypothesis that preventing and treating periodontitis may improve systemic health.^{195,222}

Little is known about the epidemiology and risk factors of periodontal diseases in Latin America, and this is particularly true for Brazil.⁹⁰ Of the few surveys that have been conducted in Brazil, most have used the Community Periodontal Index of Treatment Needs (CPITN).^{61,74,75,85,168} This and the use of convenience samples^{18,67,91,229} and other methodological limitations significantly undermine the validity and usefulness of these studies.

1.1) Epidemiology of Periodontal Diseases

Gingivitis is the most prevalent form of periodontal disease, and its occurrence seems ubiquitous.¹⁵ On the other hand, periodontitis affects a small proportion of children and adolescents, but its prevalence and extent increase significantly with increasing age.^{17,21,147} There is evidence of a higher occurrence of destructive periodontal diseases in developing than in developed countries¹⁵ and in deprived groups within populations.^{3,147} Generally, young individuals have low prevalence rates (~0.1-0.5%) of severe destructive periodontal diseases in developed countries,^{3,17,21,124,138,193,194,235} whereas much higher rates have been reported in developing countries.^{17,23,101,124} A similar trend has been described among adults. Hence, less than 30% of populations in developed countries have severe periodontitis and/or severe periodontal tissue loss,^{12,66,205} whereas those in developing countries show higher levels of disease severity.^{36,66,90} However, the view that populations in developing nations have more severe periodontal disease than those in developed nations has been questioned.⁴⁰

1.1.1) Periodontal Diseases in Adults

The prevalence of destructive periodontal diseases seems to vary greatly in different regions of the world.^{13,15} A national survey in the United States used a partial recording system and estimated that clinical attachment loss (CAL) ≥ 5 and ≥ 7 mm affected 20% and 7% of subjects aged 30-90 years, respectively.^{12,22} A national survey in the United Kingdom used the Community Periodontal Index

(CPI) and found that 42% of 35-44 years olds, and 70% of 55-64 years olds had CAL >3.5 mm.¹⁶⁷ Prevalence rates of CAL \geq 4 mm in Southern China ranged between 56% and 61% among 35-44 years olds, and between 48% and 55% among 65-74 years olds;⁶⁵ and in Kenya the estimates were approximately 90% among subjects 35 years and older.³⁷ It is, however, unclear whether the differences in disease prevalence between different studies show true differences in levels of disease between these populations, or these may be attributed to differences in methodologies used, including differences in measurement methods and the use of convenience samples or samples of questionable representativity.

Studies of destructive periodontal diseases in Latin America and Brazil often have used inadequate methodologies.⁹⁰ A survey was performed in 1986 and examined samples from the capital cities of 16 major Brazilian states and reported that 5.2% of 35-44 years olds, and 7.4% of 50-59 years olds had one or more sites with probing pocket depth (PPD) \geq 5.5 mm (CPITN code 4).¹⁶⁸ Another survey using subjects from Rio de Janeiro found that 20% of 35-44-year-olds, and 49% of 55-64-year-olds had PPD \geq 5 mm.⁸⁵

1.1.2) Periodontal Diseases in Young Subjects

The prevalence of early-onset aggressive periodontitis (AgP) has been reported to vary significantly between different populations and between subgroups within the same population.^{17,124} Among U.S. schoolchildren the prevalence of AgP ranged from 0.06% in whites, to 2.6% in African-Americans, and from 0.4% in 13-15 years old to 0.8% in 16-17 years olds.²¹ Low prevalence rates of AgP ranging between 0% and 0.3% have been reported in random samples of teenaged schoolchildren in Amsterdam, the Netherlands,²³⁵ Oslo, Norway,³ Switzerland,¹³⁸ and Santiago, Chile.¹⁵²

There are little data about the prevalence of periodontal diseases in young Latin American populations.⁹⁰ Tinoco et al.²²⁹ used a 2-stage screening strategy and examined 7,843 subjects 12-19 years old from selected schools and charity institutions from low socio-economic status populations in 3 Brazilian cities. Initially, the screening procedure used interdental wooden sticks to identify subjects having PPD \geq 5 mm at the proximal surfaces of first molars.²²⁹ Subjects with positive lesions then underwent a full-mouth clinical

and radiographic examination, and those having intrabony periodontal lesions involving molars and/or incisors and CAL >2mm were diagnosed as "localized juvenile periodontitis" cases. The overall prevalence of cases among 12 to 19 years olds in this population was 0.3%, and ranged between 0.1% and 1.1% in the 3 cities.²²⁹

Two other studies^{18,91} assessed the prevalence of AgP using bitewing radiographs as a screening method and examining convenience samples of schoolchildren from two Brazilian cities. Gjermo et al.⁹¹ reported that the prevalence of AgP was 2.6% in 15 years old students of a low socio-economic area of Belo Horizonte, Brazil. Albandar et al.¹⁸ found a prevalence of 1.3% of AgP in a sample of 13 years old schoolchildren of high socio-economic status in Sao Paulo, Brazil. Other studies in young Brazilian populations have used the CPITN methodology, and the results show that CPITN score 4 was either very infrequent or non-existent.^{61,74,75,85,168}

1.2) Partial Recording Protocols

Clinical assessments of periodontal attachment level made on 6 sites per tooth using a manual periodontal probe is considered a valid method for the assessment of the status of periodontal diseases.¹³¹ However, this method is laborious, and its use in large surveys would require a lot of resources and time that may not be readily available. In addition, the use of 6-sites and full-mouth protocol also may have other potential adverse effects, including higher measurement errors and high dropout rates due to patient and examiner fatigue.

Various partial recording protocols (PRP) have been used in epidemiological studies of periodontal diseases. Among the first reported systems is the Periodontal Disease Index¹⁸⁶ in which only 6 index teeth are examined. Another widely used PRP is the method used in the CPITN index⁶ where the full circumference of 10 index teeth is examined. A third method is often used in national and regional surveys in the U.S.^{12,14,22,89,116} in which clinical examinations are done on 2-3 surfaces per tooth in a random half mouth. An important limitation of PRPs is their potential to underestimate the prevalence, and either underestimate or overestimate the extent and severity of periodontal diseases in the studied population.^{34,71,78,131,132,177,180,223} It has been shown that the magnitude of underestimation may be

dependent on the type of parameters studied.^{34,115,131,132,177,223} However, the effects of other factors have not been adequately addressed.

1.3) Risk Factors and Risk Indicators

1.3.1) Eras and Paradigms

Epidemiology has gone through different paradigms over time and has evolved into a complex discipline.^{202,217,219} The second half of the 20th century has witnessed the flourishing of the discipline of risk factor epidemiology under the *chronic disease paradigm*. Risk factor epidemiology has been defined as the search for multiple factors associated with outcomes at the individual level.²⁰⁸ This model of research has been justified by a pragmatic, “whatever works”, approach,¹⁹² in contrast to a proposed development of an epidemiologic theory that would provide a conceptual framework for the field.^{136,137} Whereas the risk factors’ causal plausibility has been considered, the explanation of the causal processes was not a priority and was seen as a limiting factor in some circumstances.¹⁹¹ Hence, some critics call it “black box” epidemiology, implying the limited attention given to the study of the processes of causation.^{208,219}

Recent advances in molecular biology technology prompted the emergence of molecular epidemiology as a new discipline. The possibility to study the molecular or microlevel factors has provided novel opportunities of diagnosis and better understanding of the mechanisms of disease pathogenesis. On the other hand, the role that contextual variables (families, peer groups, communities, cultures) have over the occurrence of disease has been greatly neglected in epidemiology.²¹⁸ Recently a call has been made for a multilevel approach integrating all possible sources of explanation, from social groups to molecular interactions. This approach is named “eco-epidemiology”.²¹⁸

1.3.2) Definitions

The concept of risk predisposition has become widely used in the 1960’s, and the term **risk factor** has since been used to loosely imply an increased probability of occurrence of an outcome due to an exposure. The Dictionary of Epidemiology¹³⁹ defines a risk factor as “*an aspect of personal behavior or life-style, an environmental exposure, or an inborn or inherited characteristic, that, on the basis of epidemiologic evidence, is known to be*

associated with health-related condition(s) considered important to prevent”. To this definition, Beck⁴⁴ added the requirement of a temporal relationship, and the idea of preventability. Albandar,¹³ on the other hand, provided a more operational definition of risk factors applied to periodontal diseases, which were defined as “*distinctive characteristics, or exposures, that increase the probability of developing periodontitis, or lead to a measurable change (loss) in the status of the periodontal supporting tissues*”.

A terminology that distinguishes between different degrees of evidence has been proposed.⁴⁴ **Risk factors** would include factors for which strong evidence is available, usually derived from longitudinal studies. **Risk indicators** would be factors that are strongly associated with the outcome based on cross-sectional and case-control studies. Hence, an intervention should target risk factors, as it may decrease the likelihood of disease occurrence.

In this perspective it is important to note that a risk indicator that is not confirmed in longitudinal studies to have a causal relationship with a health status, should be called a **predictor**, suggesting that it has a strong correlation with the outcome without causing it. Basically, a predictor should be simple to measure, and be able to identify individuals with a high probability of having or developing a condition. The main use of a predictor is to establish groups of individuals with a higher probability of disease.⁴⁴

1.3.3) Causality and Confounding in Risk Assessment

Causality is a central issue in epidemiology, and it has evolved over time with the rise and fall of different paradigms.^{129,238} During the microbiological revolution the concept of mono-causal diseases emerged, and the search for causes that were necessary to the development of disease was the main concern. However, this concept could not explain the occurrence of chronic diseases, and the notion of the existence of a web of causation was introduced. The underlying theory was that different exposures and susceptibilities have to occur concurrently for disease to occur. A distinction has been made between **necessary** and **sufficient** causes. A cause is **necessary** when it always precedes an effect, and **sufficient** when it inevitably initiates or produces an effect.¹³⁹

Another key issue in risk assessment is

confounding and how to deal with it. Discerning true causal relationships from artifacts and delusions is an important part of risk assessment. This is especially true where the precise mechanisms of causation are poorly understood.^{129,161} Statistical adjustment and control by design are common ways to try to remove extraneous factors that are interfering with the association between an independent factor and an outcome. However, control of confounding can be performed only if a factor is known to exist, and can be measured accurately.^{54,55}

1.3.4) Risk Factors and Risk Indicators for Periodontal Diseases

The occurrence of gingivitis seems necessary for the development of periodontitis.^{25,143,172,197} However, not all sites with gingivitis may progress into periodontitis,^{24,149,172,197,198} and this demonstrates the significance of risk factors in the occurrence of destructive periodontal diseases. There are still unresolved issues regarding defining the full extent of these modifying factors and their exact role in disease development. Nevertheless, demographic variables, genetic traits, certain microorganisms, smoking behaviors, diabetes mellitus, and other diseases have been shown to significantly influence the course of periodontitis.^{2,13,87} Perhaps also other significant exposures exist that have not yet been elucidated.¹⁵ It also has been noted that destructive periodontal diseases in young individuals have some similarities with, and distinctions from, corresponding diseases of adults with respect to predisposition to disease development and occurrence of tissue loss.¹⁷

Scarce information exists regarding the factors that are associated with the onset and progression of periodontal diseases in Brazil and other Latin American populations. Information about associations between some risk factors and periodontal diseases established in other populations may also be pertinent to Latin American populations. However, risk assessment in a given population may not be invariably valid for other populations of different characteristics.¹⁸⁴ In addition, for a given factor, different populations may be exposed to different levels of exposure, which could be attributed to the population's unique historical, cultural and socio-economic background. Hence, it is likely that other risk factors more unique to the Brazilian population may have not yet been characterized.

Chronic Periodontal Disease in Adults

There is ample evidence showing that in adults the prevalence and severity of CAL increase with age.^{12,40,58,65,108,181,241} Whether there is an increased risk of destructive periodontitis in older individuals, or this relationship is mainly a consequence of the cumulative effect of time is still unknown.^{4,13,181,206}

Several studies have shown an association between gender and CAL in adults, with males having higher prevalence and extent of periodontal destruction than females.^{22,30,37,51,65,96,117,167} The level of CAL may also be influenced by race/ethnicity, although the exact role of this factor is not fully understood. Only a few well-designed studies have compared the occurrence of severe disease in different races. In the U.S. population, African-Americans and Mexican-Americans have higher levels of CAL than whites.²² However, this association was significantly reduced after adjusting for important covariates, such as cigarette smoking and income.¹¹⁷ On the other hand, some epidemiological studies have suggested that African and Asian populations do not have substantially higher prevalence of severe periodontal diseases than other populations.⁴⁰

Socio-economic status is an important risk indicator of periodontal disease in that individuals with low socio-economic status have a higher occurrence of CAL and PPD than those with high socio-economic status.^{51,76,79,82,86,168}

Oral hygiene has been consistently associated with higher occurrence of periodontal diseases in various populations.^{29,30,43,52,56,99,122,155,172,178,197,199,216}

A study of the Natural History of Periodontal Diseases showed that attachment loss in a Sri Lankan population was significantly associated with dental calculus.¹⁷² In addition, there is evidence that sites with gingival inflammation show a higher progression of attachment loss than sites without gingival inflammation.¹⁹⁷

Smoking behaviors have been consistently associated with CAL in most studies.^{13,87} Smokers have higher risk of developing chronic periodontal disease^{76,96,117,172,230} and show higher rate of periodontal destruction over time than non-smokers.^{49,82,122,156,157} However, findings from a few recent longitudinal studies suggest less detrimental effects of smoking on periodontal health than has been suggested by cross-sectional studies.^{62,172,225}

Certain systemic diseases have been associated with an increased risk of having CAL.

The association between diabetes mellitus and periodontal diseases has been studied extensively, and the evidence suggests that diabetics have considerable higher risk of having CAL than non-diabetics.^{220,225} There is incomplete information about the relationship of periodontal tissue loss with other systemic diseases and conditions such as osteopenia and osteoporosis,^{183,187} arthritis,^{10,163} and stress.^{88,112} Other forms of periodontitis may be associated with certain systemic diseases such as the acquired immunodeficiency syndrome²⁸ and Down syndrome.⁵

Aggressive Periodontitis

Understanding the etiopathogenesis of AgP at the micro-level has received much attention. Specific microorganisms have been associated with the occurrence of AgP, and significant work has focused on the association of this disease with *Actinobacillus actinomycetemcomitans*,^{27,100,173,210,212,226,227} although associations with other microorganism have also been reported.²⁰ These microbiological associations also have been described in Brazilian samples.^{68,229} A familial aggregation of AgP has been noted, and recently certain genetic polymorphisms have been linked to its occurrence.^{72,73,107}

It has been shown that the prevalence of AgP is correlated with age.²¹ A strong association has been demonstrated in young Americans between age and localized AgP, whereas no significant relationship has been shown for the generalized form of AgP.¹⁴⁷ Studies show inconsistent associations of gender with the occurrence of AgP. Some studies have found higher prevalence rates of AgP among females than males,^{42,152,194,235} whereas others did not corroborate this association.^{21,23,104,138,154,193,201}

The prevalence of AgP seems to differ significantly by race/ethnicity. In the U.S. population, the prevalence of AgP was significantly higher among African-Americans and Hispanics than in whites,²¹ and the difference remained significant after controlling for other important covariates.¹⁴⁷ On the other hand, the prevalence of destructive periodontal disease in a young population from the Netherlands was not significantly different between subgroups of different race/ethnicity.²³⁵

Studies suggest that socio-economic status also may play an important modifying role for occurrence of AgP. Children and adolescents of low socio-economic status in developing countries often

have a higher occurrence of this disease.^{91,101,152,154,229} In addition, a relatively high prevalence of AgP has been reported in underprivileged groups in developed nations,^{3,147} and significantly higher prevalence of CAL has been found in schoolchildren from low- than high education level families.²³⁵

There is little information about the role of smoking behaviors in the development of periodontal disease in young subjects. The available data suggest an association of cigarette smoking with AgP^{169,201} and the presence of CAL in young individuals.^{106,127,142,158} More studies are needed to shed light on this important issue.

Baer⁴² suggested that "juvenile periodontitis" cases typically show little inflammation and no significant local etiological factors, such as dental plaque and calculus. However, this hypothesis was not supported by findings of Albandar and co-workers²¹ in a national survey of U.S. schoolchildren which showed that individuals with AgP have higher percentage of sites with gingival bleeding and subgingival calculus than individuals with no periodontitis. Moreover, sites with gingival inflammation were at higher risk of having further periodontal tissue breakdown over time.²⁴

Gingival Recession

Gingival recession is a condition associated with multiple factors.^{128,214} It may occur following loss of the periodontal tissue attachment to teeth due to destructive periodontal diseases,^{45,150} or as a result of physical trauma from brushing^{128,144,150,190,203,214} and other oral hygiene habits.⁸¹ Perhaps other factors also play a role in the occurrence of gingival recession, such as certain anatomical factors,^{126,128} malalignment of teeth,¹²⁶ smokeless tobacco¹⁸⁸ and certain behavioral factors. However, the effects of these factors have not been adequately studied.

Subject groups with high levels of gingival recession share certain characteristics. Studies in different populations consistently show that the prevalence and severity of gingival recession increase in the older age cohorts.^{14,26,108,150,203,241} However, there is no convincing evidence that old age, per se, is a significant risk factor in the development and/or progression of gingival recession. Studies also show that the prevalence and severity of gingival recession is higher in males than in females,^{14,125,160,171} and in non-whites than in whites.¹⁴

Poor oral hygiene results in gingival

inflammation and periodontal tissue loss,^{109,125,150,171,236} and may therefore be a risk factor for gingival recession. Tobacco smoking also is an important risk factor for attachment loss and for the development of severe periodontitis^{13,26,49,87} and also has been shown to increase the risk for gingival recession in adults.^{26,60,97,160} Hence, it is reasonable to conclude that smoking may contribute indirectly to the occurrence of gingival recession through its contribution to the development of periodontitis and CAL. On the other hand, it is also possible that smoking has a direct effect on periodontal tissues leading to tissue recession, though there is insufficient evidence about this effect.

1.3.5) Attributable Fraction

Historically, relative risk and odds ratio have been the most common measures of the association between a potential risk factor and disease. However, neither of these measures take into consideration the prevalence of exposures in the target population, and therefore do not provide a good appreciation of the impact of a risk factor at the population level.^{48,240} The concept of **population attributable fraction** (PAF) was introduced in the 1950s¹⁸⁹ in order to estimate how much of the disease burden could be attributed to a given risk factor, or how much disease might have been prevented by the elimination or reduction of the risk factor. Various terms have been used to refer to the frequency of cases attributable to a given exposure.^{133,189} However, the term **attributable fraction** may be preferred because it does not imply causality.¹⁸⁹

Very few studies have addressed the issue of periodontal disease burden. Among the exposures studied, cigarette smoking seems to have a considerable impact on periodontal health in the population. In an early study, Haber et al.⁹⁸ estimated that the proportion of cases of periodontitis attributable to smoking in non-diabetic and diabetic patients attending medical institutions ranged between 13% and 56%, depending on the age group of the subjects. Two recent studies used U.S. national survey data (NHANES III) to estimate the periodontal disease attributable fractions due to smoking.^{117,230} Tomar & Asma²³⁰ defined cases as subjects with one or more periodontal sites showing a PPD as well as CAL ≥ 4 mm. They estimated that current and former smoking habits could account for the disease in 42% and 11% of the individuals, respectively. Among current smokers, 75% of cases could be attributed to smoking, while the corresponding figure among former smokers was 41% of cases. Hyman & Reid¹¹⁷ defined disease cases as the 10% of population with the greatest mean attachment loss. The attributable fraction for U.S. current smokers was 82% and 84% cases in the 20-49 and 50+ years old groups, respectively. They also estimated that the attributable fractions for the whole U.S. population (smokers and non-smokers) were 60% and 47% of cases in the two respective age groups. A case-control study in a Taiwanese population defined cases as individuals with two or more inter-proximal sites with CAL ≥ 6 mm at different teeth, and one or more sites with PPD ≥ 5 mm. This study found that only 12% of chronic periodontitis cases could be attributed to cigarette smoking.²²¹

This study aimed to investigate the epidemiology of periodontal diseases, and to assess demographic and environmental risk indicators of these diseases in the urban population of Porto Alegre, Brazil. Specific aims were to:

1. Assess the prevalence, extent, and severity of clinical attachment loss in the age group 30 years and older.
2. Study the effect of use of partial recording protocols in epidemiological studies of the prevalence of clinical attachment loss.
3. Assess the prevalence, extent, and severity of gingival recession in this population.
4. Assess the prevalence of aggressive periodontitis in young subjects.
5. Study the associations between demographic, socio-economic, and behavioral risk indicators with periodontal tissue loss.
6. Assess the proportion of adults with severe clinical attachment loss that may be attributed to cigarette smoking.

MATERIAL AND METHODS

3.1) Population

This cross-sectional survey was performed between June and December of 2001. The target population was individuals aged 14 years and older in the metropolitan area of Porto Alegre, in the State of Rio Grande do Sul, Brazil (Fig. 1). This population comprised more than 3 million inhabitants living in 14 major municipalities, encompassing Alvorada,

Cachoeirinha, Campo Bom, Canoas, Estância Velha, Esteio, Gravataí, Guaíba, Nova Santa Rita, Novo Hamburgo, Porto Alegre, São Leopoldo, Sapucaia do Sul, Viamão. Porto Alegre is the capital city of the Brazilian state Rio Grande do Sul. Rio Grande do Sul is located in the southern part of Brazil, neighboring Argentina and Uruguay.

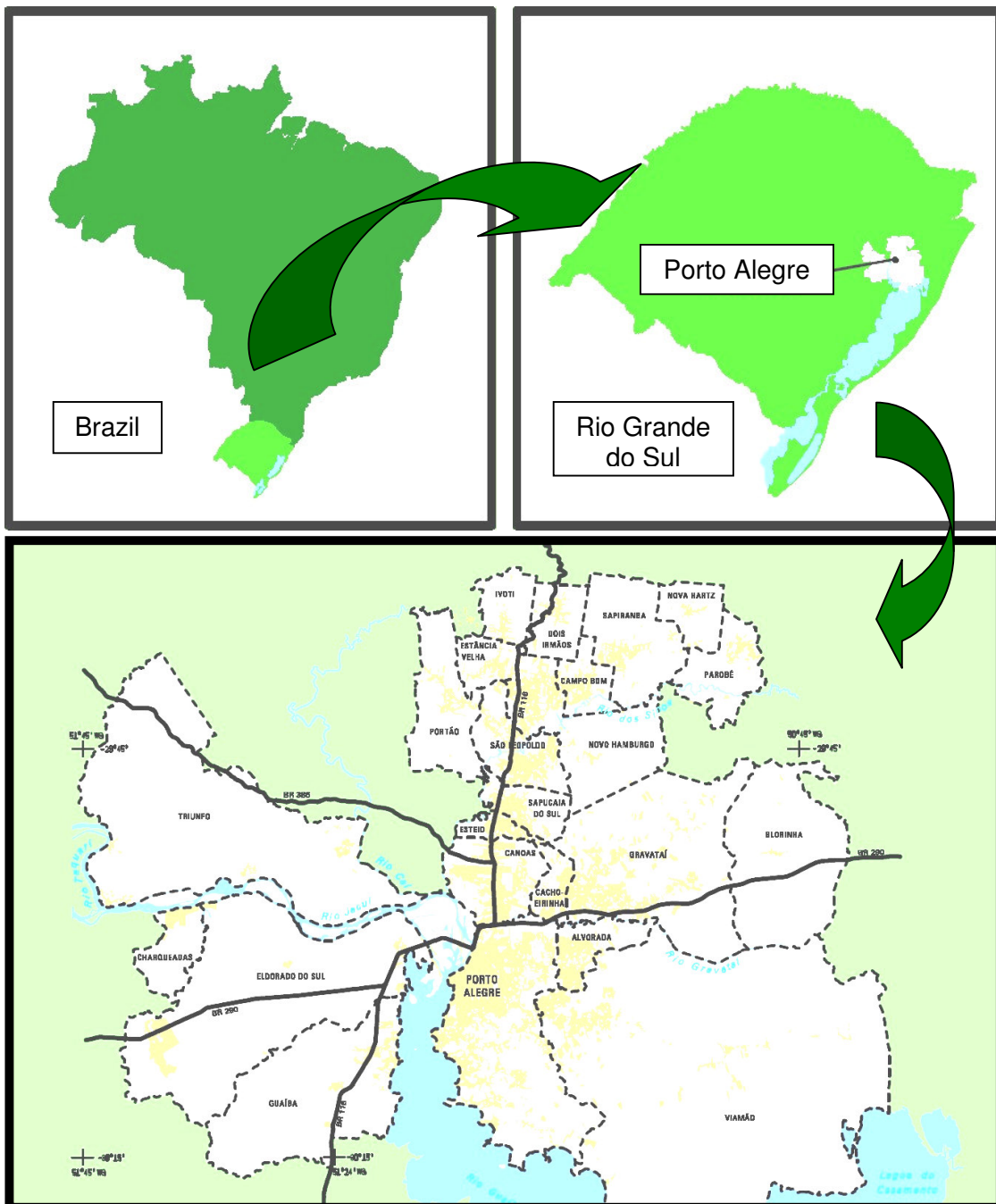


Fig. 1. Geographic location of the metropolitan area of Porto Alegre

3.2) Sample Size

Determining the accurate sample size for the study of a given disease requires a reasonable knowledge of the prevalence of the disease in the target population. However, the prevalence of periodontal disease in the study population was not known. For this reason, in this study a conservative approach to calculate the sample size using the “worst case scenario” method, whereby the prevalence of periodontal disease was assumed to be 50%. The precision level was set at $\pm 3\%$ for the 95% confidence interval for the reporting of results. Based on information reported by previous complex surveys²³⁴ an inefficiency of approximately 30% was expected due to the present sampling strategy (more details are provided in Appendix III). The sample size calculation used standard formulas for simple random sampling¹⁴¹ and it was adjusted for the design effect.¹³⁴ It was estimated that the minimum sample size was approximately 1,400 subjects.

3.3) Study Design

A multistage probability sampling method was used to derive a sample representative of the target population using information provided by Rio Grande do Sul state Government Agency for Metropolitan Affairs (METROPLAN) and the Brazilian Institute of Geography and Statistics (IBGE). The multistage sampling strategy is shown in Figure 2.

First stage: Using area maps, the survey area was divided into 90 geographic areas, 10 km² each. Using the 1991 census data 119 and other relevant municipal information 162 these geographic areas were stratified into 13 (14.4%) high-income and 77 (85.6%) low-income status areas. Low-income geographic areas were defined as areas in which more than 40% of head of the households had a monthly income ≤ 2 standard Brazilian salaries (about US\$ 180), whereas areas with a higher level of income than this threshold were defined as high-

income areas. Within each income stratum, primary sampling units (PSU) were selected randomly with a probability proportional to size and using a sampling frame of these PSUs. A total of 11 PSUs were selected, and included 2 (18.2%) geographic areas with high-, and 9 (81.8%) areas with low-income status.

Second stage: The area sectors have been defined by IBGE as map areas comprising approximately 300 households. Area sectors were selected randomly within each geographic area, and the number of sectors selected was proportional to the number of sectors in each geographic area. Thirty (3.5%) sectors were selected, out of a total of 846 eligible sectors. In each sector, approvals for conducting the study were sought from key community, religious, and/or administrative leaders. Permission and/or support were granted for 29 of these sectors, whereas permission was denied for 1 sector. Hence, only 29 sectors were sampled.

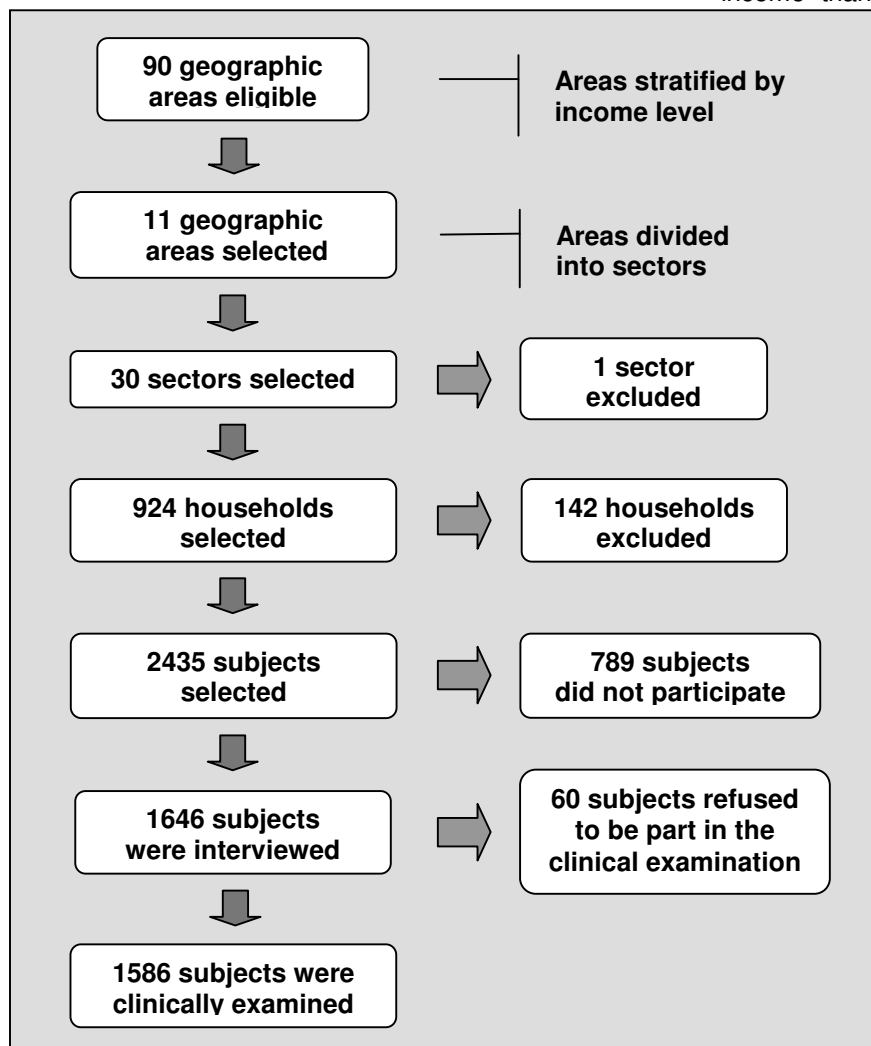


Fig. 2. Sampling strategy and study sample

Third stage: This stage included selecting households within each of the 29 area sectors. Using information about the population density it was estimated that a maximum of 25 households were needed per sector to provide a sufficient number of subjects in the sample. In each sector, a starting point for the selection of households was established on area maps that were provided by the IBGE. Households were sampled consecutively beginning with the next block after the starting point.

Eligible household members were invited to participate in the study. Inclusion criteria included subjects 14 years of age or older who agreed to participation and signed a consent form. Individuals requiring a prophylactic regimen of antibiotics were provided with the appropriate medicine before the clinical examination and were included in the study. Exclusion criteria were presence of diseases/conditions that may pose health risks to the participant or examiner, or that may interfere with the clinical examination. Generally, subjects were excluded if they were diagnosed with psychiatric problems or

communicable diseases, or were intoxicated with alcohol or drugs.

3.4) Study Sample

924 households and 2,435 persons 14 years and older were eligible for inclusion in the survey. At least three attempts on different days were made to make contact with these households while the examination team was in the same residential area. Despite these attempts, 142 (15.4%) households could not be reached (Fig.2). A total of 1,646 (67.6%) subjects were interviewed, and 1,586 (65.1%) were examined clinically.

The study subjects had an age range of 14 to 103 years (mean: 37.9, SD: 13.3 years), and comprised 719 (45.3%) males and 867 (54.7%) females, 1,309 (82.5%) whites and 277 (17.5%) non-whites. The study group comprised 1,465 dentate and 121 edentulous subjects. The distribution of the participants and the corresponding target population by gender and age groups is shown in Table 1.

Table 1. Number and percentage* of subjects in the study sample by gender and age group, and the corresponding number and estimated percentage of subjects they represented in the target population according to the 1996 census.

Age (years)	Dentate sample*				Whole sample*				Target population§	
	Male		Female		Male		Female		Male†	Female‡
	n	%	n	%	n	%	n	%	%	%
14-19	133	9.1	130	8.9	133	8.4	130	8.2	6,5	6,5
20-29	158	10.8	191	13.0	158	10.0	191	12.0	11,4	11,7
30-39	137	9.4	158	10.8	137	8.6	160	10.1	11,2	12,1
40-49	108	7.4	146	10.0	109	6.9	151	9.5	8,4	9,3
50-59	84	5.7	91	6.2	91	5.7	109	6.9	5,1	5,8
60-69	40	2.7	45	3.1	58	3.7	69	4.4	3,1	4,1
≥ 70	19	1.3	25	1.7	33	2.1	57	3.6	1,8	3,1
Total	679	46.3	786	53.7	719	45.3	867	54.7	47,4	52,6

* Percentages are not adjusted for sampling bias

§1996 Population census^{119, 121}

† N=1,241,926

‡ N=1,375,868

3.5) Interview and Clinical Examination

Interview

Eligible, consenting participants were interviewed at home or in the examination center using a structured written questionnaire form. A trained dental assistant performed the interviews. The interview gathered demographic data and information about oral hygiene practices, smoking habits, psychosocial and economic variables, the person's own history and that of their immediate

families regarding oral and general diseases, and knowledge and attitudes towards dental treatment. The interview was conducted in Portuguese, and medical terms were translated into locally understood terminology when necessary. Quality assurance procedures were employed (Appendix I).

Clinical Examinations

Four dentists and two dental assistants conducted the fieldwork. Clinical examinations were performed

in an examination center comprising a dental unit equipped with a dental chair, light, compressor, and other basic amenities. The unit was mounted in a trailer and was moved from one examination location to the next according to the survey schedule. Subjects were examined clinically to assess the status of the oral mucosa, oral prosthesis, dental caries, and periodontal health. The dental assistants recorded the data on prepared record sheets.

All permanent fully erupted teeth, excluding third molars, were examined with a Michigan-0 periodontal probe with color-coded Williams markings* at 1,2,3,5,7,8,9,10 mm. Six sites per tooth were assessed at the mesiobuccal, midbuccal, distobuccal, distolingual, midlingual, and mesiolingual sites. The following variables were measured:

- *Visible dental plaque*: teeth of one quadrant were dried with a blast of air, and sites with dental plaque which was visible to the naked eye were registered. Sites diagnosed with or without visible plaque were scored as 0 or 1, respectively.
- *Plaque retention factors*: surfaces of teeth were scored for plaque retention factors. Sites were scored as: without retention factor (0); supra-gingival calculus (1); overhanging restorations (2); cavities localized near the gingival margin (3); other retention factors (4). *Supra-gingival calculus* was defined as calcified deposits located on exposed crown and root surfaces that extend up to 1 mm below the free gingival margin.
- *Gingival bleeding*: the periodontal probe was inserted 1-2 mm into the gingival sulcus starting at one inter-proximal area and moving to the other interproximal area. Bleeding sites were scored (1) after the sites of a single quadrant were probed. Non-bleeding sites were scored (0).
- *Probing pocket depth*: the distance from the free gingival margin to the bottom of the pocket/sulcus was measured in mm and rounded to the lowest whole mm.

- *Gingival recession*: the distance from the cemento-enamel junction (CEJ) to the free gingival margin was measured in mm. If the CEJ was located apical to the gingival margin this assessment was given a negative sign. The measurements were made in mm and were rounded to the lowest whole mm.
- *Clinical attachment loss*: this was defined as the distance from the cemento-enamel junction to the bottom of the pocket/sulcus. This measurement was calculated as the sum of the probing depth and gingival recession measurements.

3.6) Assessment of Measurement Error Interview Data Reproducibility

Assessment of the interview data reproducibility was made by re-interviewing 79 study participants 1-4 days after the first interview. Between 12 and 17 core questions were used, and the consistency of answers evaluated. The mean kappa coefficient for categorical data was 0.93. The kappa coefficient for smoking status (categorized into non-smokers, light, moderate and heavy smokers) was 0.92 suggesting a high reproducibility of the interview data.

Clinical Data Reproducibility

Assessment of clinical measurements reproducibility was made by performing replicate periodontal measurements during the fieldwork. A total of 57 subjects, divided into four groups ranging from 8 to 20 subjects, were used for the reproducibility assessment. In one of the groups, the replicate measurements consisted of repeated measurements by the gold standard examiner. In each of the remaining 3 groups, one examiner and the standard examiner conducted replicate measurements. Measurement error was assessed as described by Kingman and Albandar.¹³¹ Measurement reproducibility of the standard examiner was assessed at the subject level by the intra-class correlation coefficient²⁰⁷ and weighted kappa,¹¹¹ and at the site level by the weighted kappa. A quality control methodology was used before and during the clinical data collection (Appendix I).

Intra-Examiner Reproducibility

The standard examiner showed a high agreement of attachment loss measurements, with 91.1% of

* PCP10-SE, Hu-Friedy Mfg. Co. Inc., Chicago, USA

measurements were within ± 1 mm. The weighted (± 1 mm) kappa coefficient was 0.87 for site measurements and 1.0 for subject level prevalence measurements (maximum clinical attachment loss). The intra-class correlation coefficient was 0.99 for mean attachment loss, and 0.97 and 0.98 for percentage teeth with attachment loss ≥ 5 mm and ≥ 7 mm, respectively. The estimate of the standard examiner's attachment loss measurement error was 0.24 mm.

The standard examiner also showed a high agreement of gingival recession measurements. The weighted kappa (± 1 mm) was 0.63 for the subject-level (maximum recession score), and 0.90 for the site level measurements. The intra-class correlation coefficient was 0.99 for mean gingival recession, and 0.99 and 0.81 for percentage teeth with gingival recession ≥ 3 mm and ≥ 5 mm, respectively.

The unweighted kappa coefficient for presence of supra-gingival calculus at the site level ranged between 0.59 and 0.81. The intra-class correlation coefficient for supra-gingival calculus ranged between 0.73 and 0.98 at the site level, and between 0.66 and 0.99 at the tooth level.

Inter-Examiner Reproducibility

The measurements made by the 3 other examiners were compared to that of the standard examiner to assess the inter-examiner reproducibility. For CAL measurements, the percentage agreement (within ± 1 mm) of site measurements ranged between 77.9% and 80.7%, and the weighted kappas were between 0.65 and 0.71 for site-level measurements, and 0.69 and 0.92 for subject-level prevalence (maximum CAL). The intra-class correlation coefficient ranged between 0.95 and 0.98 for mean CAL, and 0.80 and 0.94 for percentage teeth with CAL ≥ 5 mm and ≥ 7 mm, respectively.

The weighted kappas were between 0.82 and 1.00 for the prevalence of gingival recession (maximum subject level), and between 0.71 and 0.78 for the site level. The intra-class correlation coefficients ranged between 0.96 and 0.98 for mean gingival recession, between 0.66 and 0.96 for percentage of teeth with recession of ≥ 3 mm, and between 0.54 and 0.96 for extent of recession ≥ 5 mm.

3.7) Ethical Considerations

The study protocol was reviewed and approved by the following committees: Research Ethics

Committee, Federal University of Rio Grande do Sul, Porto Alegre, Brazil; the National Commission on Ethics in Research, Ministry of Health, Brasilia, Brazil; Ethics in Medical Research Committee, University of Bergen, Bergen, Norway. Subjects who agreed to participate signed an informed consent form. At the conclusion of the clinical examination, the participants were provided with a written report detailing their oral status and any diagnosed mucosal lesions. Patients diagnosed with oral conditions or diseases were advised to seek consultation and treatment with a professional. Denture-wearers who were diagnosed with candidiasis were informed about this condition and provided with information about methods of treatment, including prescriptions of anti-fungal medicines (and some times providing these medicines to the participant without charge). Treatment to relieve acute dental pain was provided free of charge.

3.8) Data Management

3.8.1) Data Entry

Interview and clinical examination data were transformed into electronic data files. The computer data files were compared with the original data sheets to check for and correct any typographical errors. Thereafter, basic frequency tables were generated to identify out-of-range values and outliers. These were identified and corrected.

3.8.2) Dependent Variables

Attachment Loss

CAL was used as a measure of chronic periodontitis. The following variables were assessed:

- The extent and severity of CAL were used to classify the subjects into the following groups (Paper I):
 - a) Severe: $>50\%$ of teeth with CAL ≥ 5 mm.
 - b) Moderate: $15\%-50\%$ of teeth with CAL ≥ 5 mm.
 - c) Slight or no attachment loss: $<15\%$ of teeth with CAL ≥ 5 mm, or only CAL <5 mm.
- Cases were defined as individuals with CAL ≥ 5 mm in $\geq 30\%$ of teeth (Paper II).
- Percentage of individuals (prevalence) with one or more sites with a given threshold of CAL. The thresholds ranged between ≥ 1 mm and ≥ 8 mm (Paper III).

Gingival Recession

Gingival recession cases were defined as follows (Paper IV):

Age group 14-29 years:

- a) Generalized recession: gingival recession ≥ 1 mm in $\geq 16\%$ of teeth.
- b) Localized recession: gingival recession ≥ 1 mm in 1% - 15% of teeth.
- c) No recession: no teeth showing gingival recession ≥ 1 mm.

Age group ≥ 30 years:

- a) Generalized recession: gingival recession ≥ 3 mm in $\geq 16\%$ of teeth.
- b) Localized recession: gingival recession ≥ 3 mm in 1% - 15% of teeth.
- c) No recession: no teeth showing gingival recession ≥ 3 mm.

Aggressive Periodontitis

Two definitions of AgP were used (Paper V):

- a) Age group 14-19 years: subjects with CAL ≥ 4 mm in ≥ 4 teeth.
- b) Age group 20-29 years: subjects with CAL ≥ 5 mm in ≥ 4 teeth.

Individuals in both age groups who had teeth with only attachment loss ≤ 2 mm, or had only one site with CAL = 3 mm were classified in the no-periodontitis group. Attachment loss at the mid-buccal site was not considered in the classification criteria.

3.8.3) Independent Variables

- *Race*: "white" or "non-white". Blacks and mulattos were combined into the "non-whites" group since there were no reliable criteria to distinguish between these two groups.

- *Socio-economic status*: information about family economy using a standard Brazilian economy classification (CCEB)³¹ and the level of education of the individual were used to group the subjects as follows:

- a) High status: had ≥ 9 years of education and ranked in the upper two tertiles of the CCEB economy classification; or had 5-8 years of education and ranked in the highest tertile of the CCEB classification.
- b) Medium status: had lower economy/education than the high socio-economic status

group, and higher economy/education than the low status group.

- c) Low status: had 1-4 years of education and ranked in the lowest two tertiles of the CCEB economy classification; or had 5-8 years of education and ranked in the lowest tertile of the CCEB classification.

- *Dental visits*: The subjects were classified according to their self-reported frequency and reasons for dental visits during the last 5 years:

- a) Regular dental care: had visited a dentist on a regular basis for maintenance care.
- b) Irregular dental care: had visited a dentist only for emergency dental treatment, or had not visited a dentist during the last 5 years.

- *Cigarette smoking*: the total exposure was calculated irrespective of their status as current and former smokers. The total number of packs of cigarettes consumed in a life time was calculated as the number of cigarettes consumed per day, multiplied by number of days of habit, divided by 20 (1 pack). Two classifications of cases were used:

Subjects 14-29 years old:

- a) Moderate/heavy smokers: consumed >2.5 pack years (or ≥ 1 pack/day for ≥ 2.5 years) or >912 packs in a lifetime.
- b) Light smokers: consumed 0.1 - 2.5 pack years (or ~ 1 pack/day for 0.1-2.5 years) or 1 - 912 packs in a lifetime.
- c) Non-smokers

Subjects 30 years and older:

- a) Heavy smokers: consumed >20 pack years (or ≥ 1 pack/day for >20 years) or >7300 packs in a lifetime.
- b) Moderate smokers: consumed 7.5 - 20 pack years (or ~ 1 pack/day for 7.5-20 years) or 2735 - 7300 packs in a lifetime.
- c) Light smokers: consumed 0.1 - 7.4 pack years (or ~ 1 pack/day for 0.1-7.4 years) or 1 - 2734 packs in a lifetime.
- d) Non-smokers

- *Supra-gingival dental calculus*: was classified by the percentage of sites with calculus into the following groups:

Subjects 14-29 years old:

- a) High: $>15\%$ of sites
- b) Moderate: 5% - 15% of sites

- c) Low: <5% of sites

Subjects 30 years and older:

- a) High: >50% of sites
- b) Moderate: 25-50% of sites
- c) Low: <25% of sites

In the study of the association between supra-gingival dental calculus and aggressive periodontitis (Paper V), dental calculus was classified into two groups, <10% and \geq 10% of sites with calculus.

- *Oral hygiene*: most participants claimed using a toothbrush regularly at least once a day, and this information was therefore not used in the present analysis.

3.8.3) Partial Recording

Seven PRPs were assessed:

- a) Mesiobuccal, and midbuccal measurements on all teeth (MB-B, full-mouth).
- b) Mesiobuccal, midbuccal, and distobuccal measurements on all teeth (MB-B-DB, full-mouth).
- c) Mesiobuccal, midbuccal, and distolingual measurements on all teeth (MB-B-DL, full-mouth).
- d) Mesiobuccal, and midbuccal measurements on all teeth in one maxillary and one mandibular, randomly selected quadrants (MB-B, half-mouth).
- e) Mesiobuccal, midbuccal, and distobuccal measurements on all teeth in one maxillary and one mandibular, randomly selected quadrants (MB-B-DB, half-mouth).
- f) Mesiobuccal, midbuccal, and distolingual measurements on all teeth in one maxillary and one mandibular, randomly selected quadrants (MB-B-DL, half-mouth).
- g) Mesiobuccal, midbuccal, distobuccal, distolingual, midlingual, and mesiolingual sites on all teeth in one maxillary and one mandibular, randomly selected quadrants (6 sites, half-mouth).

True prevalence was defined as the percentage of individuals with a given threshold of attachment loss obtained through 6 measurements and full-mouth examinations. For a given partial system, absolute bias was calculated as the difference between prevalence estimates using that system and the true prevalence (Bias = PRP estimate – FM

score). An inflation factor was derived to adjust for the underestimation of prevalence measurements. This factor was calculated as the inverse function of sensitivity. Sensitivity was defined as the proportion of diseased persons who have a positive test, and was calculated as the ratio of the prevalence of attachment loss using a given partial system, relative to the true prevalence.

3.9) Statistical Methods

Pairwise comparisons of crude estimates were carried out using the Wald test.¹³⁴ The chosen level of statistical significance was 5%, and the 95% confidence intervals (CI) were calculated.

Multinomial logistic regression analyses for complex survey data were used to model the relationship between clinical attachment loss (Paper I), gingival recession (Paper IV), and potential explanatory variables. Binary logistic regression analysis was performed to study the associations between aggressive periodontitis and explanatory variables in young individuals (Paper V) and to estimate probabilities of occurrence of periodontal disease according to smoking status (Paper II).

The probability of occurrence of an event was expressed as a relative risk ratio (RRR) when the multinomial logistic model was used, and as odds ratio (OR) in the binary logistic model. Generally, the RRR may be interpreted similarly as for the OR. The two parameters are equivalent when the outcome variable is binary, and are somewhat different when the outcome variable is multinomial. This is because in ordinary logit models, exponential coefficients are the ratio of the odds for a one-unit increase in a given independent variable to the odds when this variable is unchanged; whereas in multinomial logistic regression, exponential coefficients are the ratio of the relative risk for a one-unit increase in a given independent variable to the relative risk when this variable is unchanged. In other words, in the multinomial logistic regression there is always a base category for the outcome.⁹²

Statistical analysis was initially performed using a univariable model. Exposures showing associations with $p \leq 0.25$ in the univariable analyses were included in the multivariable model.¹¹⁰ The contribution of each variable to the model was assessed by means of the Wald statistic. Confounding and interactions were also evaluated.

The relationship between mean gingival recession and age was further studied using a linear regression analysis for complex surveys (Paper IV). The analysis suggested a non-linear relationship, and therefore piecewise linear regression was used.¹³⁴ Knots were used at the age points 25 years

and 50 years, yielding three linear splines. Linear regression was also used to compare differences between young individuals with aggressive periodontitis and those without CAL in the percentage of sites with dental plaque, gingival bleeding and supra-gingival calculus after adjusting for age, smoking, and socio-economic status (Paper V).

A logistic model for complex survey was used to predict the probability of periodontal disease, expected prevalence, and number of cases according to cigarette smoking status (Paper II). After the initial model was calculated, the exposure effect, i.e. smoking, was removed from the dataset

by resetting the covariate to zero, and the probability of the outcome in the logistic model was predicted again. The resulting estimates are the predicted probability of the outcome if the exposure had been removed. Summing these probabilities gives the expected prevalence and number of cases of disease if the exposure was absent or removed from the population.^{48,94}

Data management was performed using procedures in SAS[†]. Data analysis was performed using survey commands available in STATA[‡] and considering stratification, clustering, weighting and robust variance estimation (Appendix III).

[†] SAS 9.0, SAS Inst., Inc., Cary, NC

[‡] Stata 7.0, Stata Corp., College Station, TX, USA

RESULTS AND COMPARISONS WITH PREVIOUS SURVEYS

4.1) Clinical Attachment Loss, Age Group 30 Years and Older

4.1.1) Overall Estimates (Paper I)

Seventy-nine percent of subjects and 36% of teeth per subject had CAL ≥ 5 mm, and 52% of subjects and 16% of teeth per subject had CAL ≥ 7 mm. When classified by extent of teeth with CAL ≥ 5 mm, 42% of subjects had slight/no disease experience (<15% of teeth affected), 28% of subjects had moderate disease (15% - 50% of teeth), and 30% of subjects had severe disease (>50% of teeth). Generally, mandibular incisors and first molars were the teeth most frequently affected with CAL, irrespective of age cohort.

Most studies of the Brazilian population have used the CPITN methodology.^{61,74,75,85,168} One of the limitations of the CPITN is its use of a PRP, and this leads to an underestimation of disease estimates of prevalence.^{6,35,38,71,131,132} In addition, the CPITN cannot be used to assess CAL.^{39,131} Hence, direct comparison with these studies is not feasible.

Previous studies have suggested that pronounced PPD and other signs of periodontal diseases are highly prevalent in the Brazilian population. The present survey corroborates these findings and show a high prevalence of destructive

periodontal disease in this Brazilian adult population. In comparison, 20% and 7% of U.S. adults aged 30-90 years had CAL ≥ 5 mm and ≥ 7 mm, respectively^{12,22} (Table 2). This would suggest that this Brazilian population had 4 and 7 folds higher prevalence of ≥ 5 mm and ≥ 7 mm CAL than the U.S. population, respectively. Similarly, the mean percentage of teeth per subject with attachment loss was considerably higher in this population than has been reported for the U.S. adult population (CAL ≥ 5 mm: 36% vs. 6%; CAL ≥ 7 mm: 16% vs. 2%).

It should be noted, however, that the NHANES III survey^{12,22} assessed periodontal disease using a partial recording protocol that may have significantly underreported disease prevalence in the U.S. population,^{71,78,115,131,132,177,223} and therefore adjusted estimates should be used when comparing with other surveys. Accordingly, Table 2 provides estimates of disease prevalence for this Brazilian population calculated using a similar PRP protocol as that used in the NHANES III (Paper III). The findings suggest that the prevalence of ≥ 5 mm and ≥ 7 mm CAL in this Brazilian population was about twice as high as for the U.S. population (37% vs. 20%, and 17% vs. 7%, respectively) in spite of the wider age range in this population (Table 2).

Table 2. Studies reporting prevalence of attachment loss in different populations, and the prevalence rates at the corresponding thresholds of attachment loss in the population of Porto Alegre, Brazil.

Population	Other Populations				Porto Alegre Population			
	Age (years)	CAL (mm)	Method	%	Age (years)	CAL (mm)	Method	%
					30+	≥ 5	FMP*	79.2
						≥ 7		51.9
United States ^{12, 22}	30-90	≥ 5	PRP**	19.9	14+	≥ 5	PRP**	37.3
		≥ 7		7.3		≥ 7		17.0
United Kingdom ¹⁶⁷	35-44	>3.5	CPI	42	35-44	≥ 4	FMP*	92.2
	55-64	>3.5		70	55-64	≥ 4		99.2
Southern China ⁶⁵	35-44	≥ 4	CPI	56 - 61	35-44	≥ 4	FMP*	92.2
	65-74	≥ 6		48 - 55	65-74	≥ 6		88.3
Hong Kong ¹⁰⁸	35-44	≥ 4	CPI	74	35-44	≥ 4	FMP*	92.2
	65-74	≥ 6		33	65-74	≥ 6		88.3
Kenya ³⁷	55-65	≥ 4	4 sites,	90	55-65	≥ 4	FMP*	99.3
	35-44	≥ 7	full	35	35-44	≥ 7		44.4
	45-54	≥ 7	mouth	55	45-54	≥ 7		66.1
	55-65	≥ 7		80	55-65	≥ 7		70.0

*FMP: 6 sites per tooth, full-mouth (Paper I)

**PRP: 2 sites per tooth, random half-mouth (Paper III)

A recent survey in the U.K. used the CPI methodology and reported that 42% of 35-44 years olds, and 70% of 55-64 years olds had CAL >3.5 mm.¹⁶⁷ The corresponding prevalence rates in this population were much higher than those reported for the U. K. population (92% and 99% of subjects, respectively). However, it should also be noted that the CPI index assesses attachment loss using the same PRP design as the CPITN index, and it therefore has the same limitations.

A study in urban and rural areas in Southern China also have used the CPI, and estimated that 56% - 61% of 35-44 years olds had ≥ 4 mm, and 48% - 55% of 65-74 years olds had CAL ≥ 6 mm.⁶⁵ In Hong Kong, the prevalence of CAL ≥ 4 mm and CAL ≥ 6 mm was 74% and 33% in 35-44 years old subjects, and 96% and 69% in 65-74 years olds, respectively.¹⁰⁸ The corresponding rates in our population were 92% with CAL ≥ 4 mm among 35-44 years olds, and 88% with CAL ≥ 6 mm among 65-74 years olds (Table 2). This suggests that the prevalence of destructive periodontal diseases is higher in this study than that reported for the populations of Southern China and Hong Kong.

A survey in a group of Kenyans used full-mouth clinical examinations of 4 sites per tooth and found that 90% of subjects aged 55-65 years had CAL ≥ 4 mm, and 35%, 55%, and 80% in the age groups 35-44, 45-54, and 55-65 years, respectively had CAL ≥ 7 .³⁷ Hence, except for severe disease in the oldest age group, the prevalence rates were somewhat higher in this Brazilian population than in the studied Kenyan population (Table 2). The difference in findings between the 2 studies may be partly due to the use of 6 sites per tooth in this survey.

A survey of the Japanese population showed that more than 50% of 40+ years olds had CAL ≥ 4 mm.²⁴¹ This is considerably less than the prevalence in this population. On the other hand, comparable prevalence and extent of attachment loss was reported in a recent study in a rural population in Southern Thailand.⁴¹ Other studies of elderly age cohorts in developed countries showed lower occurrence of CAL than the present population.^{46,77,164,209}

4.1.2) Effect of Partial Recording Protocols on Prevalence Estimates of Clinical Attachment Loss (Paper III)

Estimates of attachment loss prevalence showed considerable variability among the 7 recording systems evaluated. The results showed that PRPs that use full-mouth measurements produced smaller

underestimation of this parameter than corresponding PRPs that use half-mouth measurements. The type and number of tooth sites used in the PRP also influenced the amount of underestimation. A system that used 3 sites per tooth, mesiobuccal, midbuccal, and distolingual, yielded the least bias for both the half-mouth and full-mouth design. The underreporting was also a function of the underlying prevalence and severity of attachment loss. Hence, the bias due to a given system may increase with the increase in severity of disease in the population.

These results are in agreement with the findings in young individuals examined in a national survey by Kingman and Albandar,¹³¹ who concluded that PRPs may significantly underestimate the prevalence of early-onset periodontitis. The findings also corroborate other studies that have addressed this issue in adult populations.^{71,78,114,115,132,177,180,223} However, the majority of these studies have used convenience samples and/or subjects with certain characteristics, and because sample selection may influence the disease prevalence and severity, these findings may only apply to similar study groups.

4.1.3) Risk Assessment Demographic Variables (Paper I)

The prevalence and severity of CAL increased steadily with increasing age, and was significantly higher in males than in females. Non-whites had a significantly higher prevalence of CAL ≥ 4 mm than whites, and the percentages of teeth with CAL were only marginally different between the 2 groups. Results of the multivariable analysis showed that individuals in the 40-49 and 50+ years old groups, respectively, were more likely to show moderate attachment loss (RRR=3.0 and 5.9), and severe attachment loss (RRR=7.4 and 25.4) than individuals in the 30-39 years old group, and males were 60% more likely to show severe CAL (RRR=1.6) than females. However, race did not show a significant effect in the multivariable model.

Significant associations of the prevalence and severity of CAL with age have been shown consistently in other populations.^{12,40,51,52,58,65,108,241} Papapanou et al.¹⁷⁹ retrospectively examined radiographic records of dental school patients followed-up during a 10 years period, and concluded that individuals 70 years and older had a significantly higher progression rate of alveolar bone loss than younger individuals. Albandar⁹ showed

that the progression of alveolar bone loss during 6 years was correlated with age in the age group 33 to 55 years, but found a significantly weaker correlation in the 56 years and older age group. A positive correlation between age and the rate of attachment loss also was reported in a Chinese group with limited access to dental care.⁶² Notwithstanding these results, there is no convincing evidence that age, as such, is a major risk factor for the development of severe CAL.^{4,13,58,206} Hence, the precise role of aging in the development and progression of periodontal disease and for the predisposition to tissue loss is yet to be determined.

National U.S. surveys have suggested a significantly higher prevalence and extent of CAL in males than in females after adjusting for age and race/ethnicity,²² and a higher likelihood of severe CAL in males than in females using multivariable models.^{51,117} Similar findings also have been reported in other studies.^{30,37,65,96,167} In contrast, a study in Southern Thailand did not find a significant difference in the likelihood of CAL between males and females.⁴¹

Only a few studies have assessed the association between CAL and race/ethnicity in other populations. Albandar et al.²² reported a significantly higher prevalence and extent of attachment loss in African-Americans and Mexican-Americans than in whites, after standardizing by age groups and gender. On the other hand, Hyman & Reid¹¹⁷ re-analyzed the same data using a multivariable model and did not find a significant association between race and CAL. A study of U.S. cohorts examined in the NHANES I and NHANES III showed that African-Americans were more likely to have periodontitis than whites in both surveys, and the disparity between the 2 race groups had increased over the 15 years interval that separates the two surveys.⁵⁰ On the other hand, the risk for having periodontitis was similar for Mexican-Americans and whites.⁵¹ Corroborating this relationship, a recent study in Detroit, U.S.A. found that African-Americans were more likely to have periodontitis than whites after adjusting for various demographic, socio-economic and health effects.⁵²

Socio-Economic Status (Paper I)

In the age group 30+ years, subjects in the low and medium socio-economic status groups, respectively, were 80% and 60% more likely to have severe CAL than individuals in the high socio-economic

status group. This is consistent with previous findings of higher frequencies of deep pockets (PPD >5.5 mm) in low- compared to high income groups in Brazil,¹⁶⁸ and among individuals of low than high socio-economic status in Chile.⁸⁶

A recent U.S. national survey showed a clear relationship between prevalence of CAL and socio-economic status.⁷⁹ A further analysis of the NHANES III data showed that lower income and fewer years of education were significantly associated with periodontitis.⁵¹ A study of 45+ years old subjects from Florida found a significant association of income and education with severe CAL.⁷⁶ In addition, a follow-up study of older individuals in North Carolina found that subjects in the low socio-economic status group were more likely to have new incidence of attachment loss during a 7-year period than those in the high socio-economic status group.⁸² Other studies also found significant association of education with CAL and alveolar bone loss.^{56,146} In contrast, a study among U.K. adults unveiled only small differences in the prevalences at various thresholds of CAL between groups of different social class.¹⁶⁷

Cigarette Smoking (Papers I and II)

Smoking was significantly associated with CAL in this 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.

This pattern of a strong association between cigarette smoking and higher prevalence and severity of periodontal disease is in agreement with the findings of several other epidemiological studies.^{13,51,52,76,87,96,117,230} Moreover, longitudinal studies show that periodontal disease progression is higher in smokers than in non-smokers.^{49,82,122,156,157} However, some longitudinal studies did not confirm that smokers have a higher risk for CAL than non-smokers. For instance, smoking was not significantly associated with periodontal disease progression in Sri Lankan tea laborers followed up for 20 years.¹⁷² In a group of Chinese subjects followed-up for 10 years, a relationship between cigarette smoking and progression of CAL was found in young subjects, but not in individuals 50+ years old.⁶² In addition, smoking was not significantly associated with incidence of CAL during a

follow-up period of 5 years in an elderly cohort in South Australia.²²⁵ Possible reasons for the lack of association in these studies may be a high frequency of tooth loss in the study groups, and inaccuracies in measuring smoking habits.

From a public health perspective, cigarette smoking was an important exposure in this population (Paper II). The present findings show 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 $\geq 30\%$ of teeth with CAL ≥ 5 mm. Furthermore, the estimated reduction in the total population would be approximately 12%, or nearly 90,000 potential cases.

Two recent studies have assessed the impact of cigarette smoking on periodontal health in the U.S. population.^{117,230} In comparison with our results the latter 2 studies may suggest that the population attributable risk due to cigarette smoking is much higher in the U.S. population than in this Brazilian population. However, a recent study in a group of Taiwanese adults reported estimates of attributable risk due to cigarette smoking similar to findings in this study population.²²¹ Furthermore, higher proportions of cases could be attributed to smoking among diabetic and non-diabetic patients compared to the present findings.⁹⁸ Different studies have used inconsistent definitions and diverse thresholds of attachment loss, and this may have contributed to the differences in the estimates of attributable risk due to smoking in these studies.

Dental Calculus (Paper II)

The risk for attachment loss associated with dental calculus was positively correlated with the percentages of sites exposed to calculus. The likelihood of having CAL ≥ 5 mm was significantly higher in individuals who had 25-50% (OR=2.7) and $>50\%$ of sites (OR=8.6) with calculus, compared to individuals with $<25\%$ of sites with calculus.[§]

Only a few surveys have studied the impact of oral hygiene and calculus on periodontal health in populations. The NHANES I survey in the U.S. population showed a positive correlation between the level of oral hygiene and the severity of periodontal disease as measured by Russell's periodontal index.^{4,13} Using data from the NHANES III, Hyman & Reid¹¹⁷ showed that gingival bleeding was significantly associated with increased CAL

after adjusting for demographic, socio-economic and behavioral factors. Other cross-sectional and follow-up studies corroborate these findings and suggest a similar relationship.^{29,30,43,52,56,99,122,155,172,178,197,199,216}

Diabetes Mellitus (Paper I)

Individuals with self-reported 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 association between diabetes mellitus and periodontal health has been shown in surveys in different populations and in longitudinal studies.^{13,51,76,83,220,225} However, little is known about the impact of diabetes mellitus on periodontal health in the Brazilian population. A study in a young Brazilian group of insulin-dependent diabetics found that cases had greater alveolar bone loss than age and gender matched controls.¹⁷⁴ Another study in Japanese-Brazilians found a somewhat higher prevalence of CAL >6 mm in insulin-independent diabetics than in individuals with a normal glucose level, though the difference was not statistically significant.²³¹ Interestingly, analysis using the NHANES III data did not find a significant association between history of diabetes mellitus and increased CAL.¹¹⁷

In this study, only approximately 4.9% of subjects in the 30+ years age group self-reported as being diabetics. In contrast, 7.1% and 9% of subjects in a random sample of Brazilian adults 30-69 years old in Rio de Janeiro, had diabetes mellitus and impaired glucose tolerance, respectively.¹⁷⁵ Comparatively, U.S. national surveys show that 7.8% of 20 years and older Americans have diabetes.¹⁰² Hence, the prevalence of self-reported diabetes in this survey population is lower than in other Brazilian studies as well as in the American population. Notably, a large segment of this study population was of a low socio-economic class and probably without medical coverage, thus it is likely that a high percentage of them were not aware of their diabetic status. This could have influenced the findings in this study.

Dental Visits Pattern (Paper I)

Individuals who have had irregular dental visits during the last 5 years were 2.1 times more likely to have severe CAL than individuals who reported regular dental visits. Analysis using NHANES III

[§] Additional results not shown in Paper II

data showed that individuals with infrequent dental visits had higher risk of having increased CAL than frequent users of dental care.¹¹⁷ A survey of adult Floridians found that subjects without regular dental care were more likely to have CAL ≥ 7 mm than those with regular care.⁷⁶ In another U.S. sample, an interaction between race/ethnicity and frequency of dental visits was observed, and frequent dental visits reduced the chances of having periodontitis for whites, but not for African-Americans.⁵² In a sample of adults 50+ years old from Canada, regular preventive visits and the time since last dental visit were significantly associated with increased CAL.¹⁴⁶ A follow-up study of a 65+ years old cohort showed that subjects who did not have a dental checkup during a period of 7 years were more likely to experience progression of attachment loss during this period.⁸²

Conversely, other recent studies found no significant association between dental visits and the prevalence of various thresholds of CAL in U.K. adults,¹⁶⁷ and no significant difference in the incidence of CAL between groups having a regular or episodic pattern of dental attendance in older Australians.²²⁵ Dental services utilization also was not significantly associated with alveolar bone loss over a period of 10 years.⁵⁶

4.2) Gingival Recession (Paper IV)

4.2.1) Overall Estimates

Age Group 14-29 Years

In this age group, 60% and 18% of subjects, and 13% and 2% of teeth per subject had gingival recession ≥ 1 mm and ≥ 3 mm, respectively. Mandibular incisors showed the highest prevalence of gingival recession ≥ 1 mm.

Only a few studies have assessed the occurrence of gingival recession in young populations. In a cohort of 26 years old in New Zealand, 71% and 5.5% of subjects had gingival recession ≥ 1 mm and ≥ 3 mm, respectively.²²⁴ Albandar et al.²³ found that gingival recession was prevalent among Ugandan school attendees, and occurred most frequently at mandibular incisors and canines. Approximately 60% of 20-29 years old Japanese had gingival recession ≥ 1 mm.²⁴¹ In a sample of 20-34 years old Tanzanians without regular dental care, 18% of surfaces had gingival recession, and the teeth most severely affected were the mandibular incisors.²³⁶ Other populations without regular dental care also have shown similar results.^{7,33,150}

Age Group 30 Years and Older

Moderate and advanced levels of gingival recession were common in adults and older age groups. Gingival recession ≥ 3 and ≥ 5 mm were present in 73% and 35% of subjects, and in 27% and 9% of teeth per subject, respectively. Maxillary first molars and mandibular second premolars had the highest frequency of recession. These rates are approximately 3-4 times higher than the prevalence and extent of gingival recession ≥ 3 mm previously reported for the U.S. adult population.¹⁴ However, as discussed above, the U.S. estimates may be significantly underreported due to the use of PRP in the assessment of periodontal measurements. Surveys show that gingival recession also is common in other populations, particularly in older age groups. For instance, a study in Hong Kong found that 22% and 4% of 35-44 years old, and 69% and 26% of 65-74 years old had gingival recession ≥ 4 and ≥ 6 mm, respectively,¹⁰⁸ and all 50+ years old in a group of Japanese subjects had gingival recession ≥ 1 mm.²⁴¹ Among 35-44 and 45-64 years old Tanzanians, gingival recession was observed in 31% and 51% of surfaces, respectively.²³⁶

4.2.2) Risk Assessment Demographic Variables

The pattern and severity of gingival recession were significantly associated with age. The prevalence of recession ≥ 3 mm was 6% subjects in the 14-19 years age group, and increased to 24%, 54%, and 94% subjects in the 20-29, 30-39 and 70+ years groups, respectively. A similar pattern also was seen for the percentages of teeth with recession.

In the age group 14-29 years, no significant differences in the prevalence or severity of gingival recession were found between males and females, or between whites and non-whites. In subjects 30 years of age and older, males showed higher prevalence and extent of gingival recession than females, and the differences were consistent among the various age groups and for various recession thresholds. In addition, gingival recession ≥ 2 mm and ≥ 3 mm were significantly more prevalent in non-whites than in whites. However, the percentages of teeth having recession were comparable in the 2 race groups. Non-whites had higher likelihood of having localized recession (RRR=2.8) than had whites.

A higher prevalence of gingival recession

among older than younger age groups, and in males than in females is in agreement with other studies.^{14,26,33,108,125,150,160,171,203,236,241} A recent national U.S. survey reported significantly higher prevalence and extent of gingival recession in blacks than in whites.¹⁴

Socio-Economic Status

The low socio-economic status group showed higher levels of gingival recession compared to the high socio-economic status group, and this difference was consistent irrespective of the recession threshold or age group. However, socio-economic status was not significantly associated with gingival recession after adjusting for the effect of other factors. No other studies were available for comparison with this population.

Cigarette Smoking

In the age group 14-29 years, moderate/heavy smokers were twice 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 somewhat more likely to have localized recession ≥ 3 mm, and were significantly (RRR=3.0) more likely to have generalized recession ≥ 3 mm than non-smokers.

A few recent studies also have reported significant associations of gingival recession with smoking behaviors.^{26,60,97,160} On the other hand, no significant association was found between smoking and recession in a Tanzanian group,¹⁷¹ or in a 6 months follow-up study in young subjects with minimal periodontal disease.¹⁷⁰

Dental Calculus

In the 14-29 years group, subjects having $>15\%$ of teeth with calculus were 2.3 times more likely to have localized recession ≥ 1 mm, and 3.8 times more likely to have generalized recession ≥ 1 mm than those with $<5\%$ of teeth with calculus. In the 30+ years group, subjects having 25-50% and $>50\%$ of teeth with calculus, respectively, were 40% and 60% more likely to have localized recession ≥ 3 mm, and 2.2 and 6.4 times more likely to have generalized recession ≥ 3 mm than those with $<25\%$ of teeth with calculus.

Previous studies also show significant associations of gingival recession with dental calculus and poor oral hygiene.^{109,125,171,236} However, it also has been shown that gingival recession could occur in populations with good oral

hygiene, and this may be attributed to other factors such as trauma due to inadequate brushing techniques.^{125,128,144,190,203,214}

Dental Visits Pattern

Subjects who have had irregular pattern of dental visits had significantly higher percentage of teeth with recession ≥ 3 mm than subjects with regular visits. However, this effect was not statistically significant in the multivariable model. Similar findings of a significant association in a univariable analysis and no significant effect in a multivariable analysis has been reported elsewhere.¹⁷¹

4.3) Aggressive Periodontitis, Age Group 14-29 Years (Paper V)

4.3.1) Overall Disease Estimates

Destructive periodontal disease was prevalent among the young segment of this study population. CAL ≥ 3 mm affected approximately half of the subjects and 15% of teeth per subject, and CAL ≥ 5 mm was found in one fourth of the subjects and 3% of the teeth per subject. 5.5% of individuals had aggressive periodontitis, and the prevalence of the disease was higher in the 20-29 years than in the 14-19 years groups. Subjects with AgP had multiple teeth affected with attachment loss.

In the present population, 2.5% of 14-19 years old had AgP. In contrast, a survey using large sample of 12-19 years olds from low socio-economic status populations in 3 Brazilian cities reported a prevalence of "localized juvenile periodontitis" ranging between 0.1% and 1.1% (mean=0.3%).²²⁹ However, the latter study used a special initial screening strategy whereby interdental wooden sticks were used to identify subjects with PPD ≥ 5 mm at the proximal surfaces of first molars, who were then examined clinically and radiographically more thoroughly. This screening strategy may partly have contributed to the difference in disease estimates between the two studies.

Gjeramo et al.⁹¹ used bitewing radiographs as a screening method and reported a prevalence of AgP of 3.7% in 15 years old students of a low socio-economic area in Belo Horizonte, Brazil. Albandar et al.¹⁸ also used bitewing radiographs and reported a prevalence of 1.3% among a population of 13 years old schoolchildren of high socio-economic status in São Paulo, Brazil. The samples used in these 2 studies are of uncertain representativity.

The prevalence of AgP in the population of Porto Alegre is somewhat higher than the estimates of disease in most developing countries,^{17,101,124,152} and is significantly higher than what has been reported in developed countries.^{3,21,138,193,194,235} Some other young populations also have been reported to have a high prevalence of CAL; these include New Zealand,²²⁴ Chile,¹⁵⁴ and Uganda.²³

4.3.2) Risk Assessment

Demographic Variables

Subjects 26-29 years of age were 6.2 times more likely to have AgP than those in the 14-19 years group. However, the prevalence of the disease was not significantly higher in the 20-25 than in the 14-19 years groups. The prevalence also was not significantly different between males and females. 6.1% of non-whites and 2.4% of whites were diagnosed with AgP. However, the difference between the 2 groups was not statistically significant. Similarly, the multivariable analyses showed no significant effect of race on prevalence of AgP and CAL in this age group. These findings may be attributed to the relatively small percentage of non-whites in this population, and the inclusion in the multivariable model of pertinent explanatory variables, such as socio-economic status.

A similar finding of a higher prevalence of AgP in older than in younger schoolchildren has been reported by Albandar et al.²¹ Multivariable analyses using a large sample examined in the National Survey of U.S. Children showed a significant association of “localized juvenile periodontitis” with age, and no significant association of “generalized juvenile periodontitis” with age.¹⁴⁷ This and other related findings¹⁵³ suggest that disease classification may influence the relationship between prevalence of disease and age.

It has long been maintained that “juvenile periodontitis” is significantly more prevalent in females than in males.⁴² This, however, was not supported in this study. Inconsistent findings have been reported in other studies. For instance, different studies have shown higher^{11,152,194} or lower^{23,147} prevalence in females than in males, or no significant difference between gender groups.^{21,104,138,193,201,229}

A study in a group of Brazilian schoolchildren of low socio-economic status reported that 5% of males and 2.7% of females had AgP.⁹¹ On the other hand, females were more likely to have

aggressive periodontitis than males in a sample of subjects 15–25 years old seeking dental treatment.⁶⁷ Alveolar bone loss over 3 years was not significantly different in males than in females in another group of Brazilian adolescents.¹⁹ Females in Chile were 40% more likely to have CAL ≥ 3 mm¹⁵⁴ and 7 times more likely to have AgP¹⁵² than males. A study using a group of adolescents from the Netherlands found that CAL ≥ 5 mm was more prevalent among females than males.²³⁵

The gender effect may also be modified by the disease classification and other co-factors. Using multivariable analysis in a large sample of U.S. schoolchildren, L oe & Brown¹⁴⁷ showed that males were more likely to have “generalized juvenile periodontitis” than females. However, for “localized juvenile periodontitis” they reported that the relationship with gender was influenced by race, in that black males were more likely to have “localized juvenile periodontitis” than black females, whereas white females were more likely to have the disease than white males.¹⁴⁷

Only a few other studies have investigated the effect of race/ethnicity on the occurrence of AgP. Two large studies showed significantly higher prevalence of AgP in African-Americans and Hispanics than in whites.^{21,147} In a large sample of schoolchildren in Norway, Asians and other immigrants had higher occurrence of alveolar bone loss than Norwegians.³ Subjects of African or Asian background also had higher occurrence of AgP than Caucasians in a U.K. sample.¹⁹³ No differences were reported between ethnic groups in other European populations.^{138,235}

Socio-Economic Status

The present results show that young Brazilian subjects with low socio-economic status were 4.5 times more likely to have AgP than those in the middle or high socio-economic status groups after adjusting for demographics and other risk indicators. This is in agreement with previous findings of a high level of disease in young groups from Brazilian neighborhoods of low socio-economic status,^{91,229} and a higher prevalence of CAL and AgP in Chilean schoolchildren from low/middle- than in high socio-economic status schools.^{152,154} No association was found between socio-economic status and prevalence of AgP in Uganda students.²³ This association also has been shown in other populations in developing countries¹⁷ and among deprived groups in rich nations.^{3,147,235}

Dental Calculus

AgP was 3.6 times more likely to occur in individuals with $\geq 10\%$ of sites than those with $< 10\%$ of sites with dental calculus. This is in agreement with more recent studies showing higher levels of dental calculus and other local factors in subjects with AgP and other early-onset forms of periodontitis or with progressive tissue loss.^{21,24,25,63,95,216,235} However, the findings are in disagreement with a previously maintained view that “juvenile periodontitis” is not associated with local etiological factors such as dental plaque and calculus.⁴² It is important to note that some of the earlier studies on AgP^{138,193,194} used the classification criteria of Baer⁴² and, accordingly, have excluded individuals with large amounts of plaque and calculus. This may explain the perpetuation of the belief that AgP was not associated with local etiological factors. The present findings also are in accordance with the low level of oral hygiene observed in individuals with AgP reported in another Brazilian sample.²²⁸ Local plaque-retention factors, such as caries lesions and defective restorations, significantly increased the risk for further alveolar bone loss in Brazilian schoolchildren.¹⁹

Cigarette Smoking

AgP was highly prevalent among young individuals who were heavy smokers, compared to non-smokers. In the multivariable model, heavy smokers were 3.1 times more likely to have AgP than non-smokers.

Few studies have addressed the role of smoking in the occurrence of AgP and attachment loss in the < 30 years age groups. Most studies have reported higher levels of CAL and/or radiographic bone loss,^{106,127,142,158,169} and a greater likelihood of showing a generalized than localized AgP¹⁶⁹ among young smokers than non-smokers. No significant association was found between smoking and occur-

rence of CAL in a young Chilean population after adjusting for other risk indicators.¹⁵⁴ However, in the latter study the subjects had a relatively low cigarette smoking exposure, and this may explain the negative findings.

The study of the association of smoking habits with periodontal disease in young populations often is confounded by the complexity of assessing this exposure in young age groups. Smoking exposure is typically measured by means of a questionnaire or an interview. However, cigarette smoking by young subjects is generally regarded as a “socially incorrect” behavior, and this may influence the reliability of the data. In this study, we used adequate interview techniques, and assessed the reproducibility of participants’ response in order to reduce the measurement error (Appendix I).

Alternative methods to assess smoking exposure includes the use of laboratory assays which usually are more objective, and therefore may be more precise. One study assessed the cotinine serum level in a large group of young individuals and found a greater severity of CAL and higher likelihood of generalized than localized AgP in smokers than in non-smokers.²⁰¹

Dental Visits Pattern

No significant association was found between the prevalence of AgP and the pattern and frequency of dental visits. There are little data on the role of professional dental care on the occurrence and progression of periodontal diseases in young subjects. A study of Chilean schoolchildren demonstrated that a higher level of CAL was associated with infrequent pattern of dental visits.¹⁵⁴ Albandar⁸ showed that populations without community dental care had higher occurrence of alveolar bone loss than those that enjoyed regular dental care through community programs.

DISCUSSION

This study is among the very few surveys of representative populations in the continent of South America.⁹⁰ It used a probability sample representative of age, gender and socio-economic cohorts in the Porto Alegre metropolitan area, an urban population in South Brazil (Appendix II and III). We investigated the level of destructive periodontal diseases and gingival recession, and the role of various risk indicators in the occurrence of the disease in this population.

Moderate and severe attachment loss and generalized gingival recession were highly prevalent among adults (Papers I, IV), and aggressive periodontitis was prevalent among young subjects in this Brazilian population (Paper V). Indeed, periodontal diseases were much more prevalent in this study population than have been reported in developed nations and in most studies in developing nations.^{12,14,17,22,36,66,90,205}

Epidemiological studies of periodontal diseases often have used different examination methods and inconsistent disease criteria and/or periodontal disease classifications.¹³¹ Many studies also have used convenience samples, and therefore the representativity of their samples may be questionable. Moreover, other discrepancies also exist, such as differences in the extent, pattern, and causes of tooth loss. In particular, studies in Brazilian populations have been scarce and/or inadequate.⁹⁰ Hence, direct comparisons of findings with other studies may not be warranted.

A clinical examination method that uses a full-mouth protocol and 6 sites per tooth yielded the most accurate assessment of periodontal diseases in the population. However, limited manpower, time, and other resources, as well as other logistical constraints often render more rational the use of a protocol that examines only part of the dentition and/or a few sites per tooth. The results showed that the effect of different PRPs on estimates of attachment loss may vary significantly, and that these differences may also be influenced by certain characteristics of the particular population under study (Paper III). In this population, a protocol that used all teeth (full-mouth) and 3 sites per tooth, the mesiobuccal, midbuccal, and distolingual sites, yielded the least bias in estimates of prevalence of disease as compared to 6 other PRPs (Paper III).

Therefore, it is important that studies that use partial recording systems should assess the magnitude of the bias due to these methods, and preferably also calculate an inflation factor to adjust for the underreporting of disease. In this way the assessment of disease burden may be more precise, and comparisons with other studies be more valid.

It has been suggested that during the past century developed countries have had an epidemic of periodontitis, perhaps attributed to changes in life style exemplified by the establishment of new practices such as smoking behaviors¹¹³ and other environmental changes.¹⁵ Results of recent studies support this view and show that a substantial proportion of severe periodontitis in various populations may be attributed to smoking behaviors.^{13,26,49,87,117,142,221,230} In this Brazilian population, cigarette smoking was a major risk factor and showed a significant association with moderate and severe attachment loss (Paper I). From a public health perspective an important proportion of the burden from periodontal disease in this population may be attributed to cigarette smoking (Paper II). Consequently, smoking cessation should be an integral part of any initiative intended to improve the periodontal status of this and other similar populations. Furthermore, the finding that smokers in the young age group had higher likelihood of having AgP (Paper V) shows that the detrimental effects of smoking on the periodontium are not limited to adults and older age groups. Accordingly, these findings also stress the importance of the establishment and implementation of smoking cessation programs that specifically target young age groups.

Consistent with other surveys,^{3,51,56,76,79,82,86,91,146,147,152,154,168,229,235} these results showed that socio-economic status was significantly associated with periodontal tissue loss in young as well as in older age groups (Paper I, V), and also was associated with generalized gingival recession (Paper IV). Studies suggest that socio-economic status and general health are correlated, and that individuals with poor economy generally have worse systemic health than their better-off counterparts.^{53,239} In addition, groups with low socio-economic status also show a higher frequency of

other detrimental exposures, such as smoking behaviors, poor oral hygiene, and non-compliance with dental care.^{69,76} Poverty may be associated with increased exposure to environmental risk factors and unhealthy behaviors, insufficient knowledge and access to information, as well as inadequate access to health care.^{53,239} This may account for the greater burden of periodontal disease in the low socio-economic status group. Improving knowledge and access to health services for underprivileged groups in developing as well as in developed nations, may lessen the disparities in oral and systemic health and may result in an improvement in quality of life.

The effects of race and ethnicity as risk factors for the occurrence of various diseases have been debated.^{130,165} A high frequency of certain genetic profiles and/or microbiologic and other pathogenic mechanisms has been cited by some as the principal rationale for an inherently higher risk for some diseases in certain racial groups. A second view argues that groups with different socio-economic status, cultural background and societal organization are exposed to different risk factors, and that these dissimilarities should be adjusted for when assessing the relationship between diseases and race. Furthermore, similar to many other cultures, in the Brazilian population there is hardly any segregation of the society based on racial or ethnic background. Consequently, clear distinction between races in this study population was complicated and may be prone to misclassification errors.

The findings showed a higher occurrence of attachment loss and aggressive periodontitis among non-whites than whites (Papers I, V). However, these associations were not statistically significant when other risk indicators were included in the analytical model. This may be due to a number of factors. As stated above, the classification by race in the Brazilian population may be prone to a classification bias which may be higher than in other populations. A second factor may be the relatively small number of non-whites in the present study group. Moreover, in this population the effect of race may be confounded by other explanatory variables,

such as socio-economic status, smoking behavior, and level of dental calculus. Hence, the inclusion of these variables in the multivariable model may have implicitly accounted for the effect of race.

Poor oral hygiene is a major factor in the development and progression of periodontal diseases.^{4,13,29,30,43,56,99,122,143,148,155,172,178,197,199,216}

The level of dental calculus was used as a surrogate for the long-term efficacy of oral hygiene, and the results showed that extensive dental calculus was significantly associated with a higher prevalence and more severe periodontal destruction (Papers II, V). There is also evidence that oral hygiene may be less adequate in smokers than in non-smokers,²⁰⁴ in blacks than in whites, and in males than in females.^{4,12,69} In addition, a poor level of oral hygiene and/or a higher level of dental calculus may also be associated with other risk indicators of periodontal disease, such as irregular pattern of dental care and low socio-economic status.^{69,76}

A large body of evidence show that dental calculus and poor oral hygiene have direct effects leading to inflammatory changes and attachment loss, and also have interactions with other important risk indicators of periodontal diseases. These multiple effects may explain the strong association between calculus and periodontal status in this study. It may be inferred that improving oral hygiene and providing access to dental care may significantly improve the periodontal status of this population.

In the context of the present cross-sectional study, it is important to acknowledge the limitations of risk factor epidemiology in determining causality. In addition, the focus on the individual level is likely to underestimate possible interactions between the effect of major social clusters and the biological mechanisms underlying the pathogenesis of disease. A more integrated study of the risk factors, in which a broad analytical approach is used integrating the strengths of the micro, macro and individual levels is desirable and is likely to improve the understanding of the epidemiology and risk factors of periodontal diseases.

CONCLUDING REMARKS

Based on the findings in this urban Brazilian population, it may be inferred that:

- Moderate and severe clinical attachment loss is common among adults in this population.
- A high percentage of the population has moderate and severe gingival recession.
- Aggressive periodontitis is prevalent among adolescents and young adults, and is more prevalent than in most other populations.
- Partial recording consistently underestimates the prevalence of attachment loss, and the extent of underestimation is dependent on the type of system used and the characteristics of the population surveyed. The use of partial recording systems may explain part of the differences in results between different studies.
- Important risk indicators for destructive periodontal diseases include older age, lower socio-economic status, presence of dental calculus, and smoking behavior.
- Cigarette smoking is an important target in any prevention strategy aimed at reducing the burden of disease, and smoking cessation should be considered for inclusion in all programs designed to prevent or control periodontal diseases.
- The present study establishes a baseline for future monitoring of the trend of periodontal diseases in this Brazilian population. This approach is likely to enlighten many of the unknown factors that play a role in the occurrence of the periodontal diseases in this population.

REFERENCES

1. AAP. The pathogenesis of periodontal diseases. *J Periodontol* 1999;70:457-470.
2. AAP. Diabetes and periodontal diseases. Committee on Research, Science and Therapy. *J Periodontol* 2000;71:664-678.
3. Aass AM, Albandar J, Aasenden R, Tollefsen T, Gjermo P. Variation in prevalence of radiographic alveolar bone loss in subgroups of 14-year-old schoolchildren in Oslo. *J Clin Periodontol* 1988;15:130-133.
4. Abdellatif HM, Burt BA. An epidemiological investigation into the relative importance of age and oral hygiene as determinants of periodontitis. *J Dent Res* 1987;66:13-18.
5. Agholme MB, Dahllof G, Modeer T. Changes of periodontal status in patients with Down syndrome during a 7-year period. *Eur J Oral Sci* 1999;107:82-88.
6. Ainamo J, Ainamo A. Partial indices as indicators of the severity and prevalence of periodontal disease. *Int Dent J* 1985;35:322-326.
7. Akpata ES, Jackson D. The prevalence and distribution of gingivitis and gingival recession in children and young adults in Lagos, Nigeria. *J Periodontol* 1979;50:79-83.
8. Albandar JM. Prevalence of incipient radiographic periodontal lesions in relation to ethnic background and dental care provisions in young adults. *J Clin Periodontol* 1989;16:625-629.
9. Albandar JM. A 6-year study on the pattern of periodontal disease progression. *J Clin Periodontol* 1990;17:467-471.
10. Albandar JM. Some predictors of radiographic alveolar bone height reduction over 6 years. *J Periodontol Res* 1990;25:186-192.
11. Albandar JM. Juvenile periodontitis--pattern of progression and relationship to clinical periodontal parameters. *Community Dent Oral Epidemiol* 1993;21:185-189.
12. Albandar JM. Periodontal diseases in North America. *Periodontol 2000* 2002;29:31-69.
13. Albandar JM. Global risk factors and risk indicators for periodontal diseases. *Periodontol 2000* 2002;29:177-206.
14. Albandar JM, Kingman A. Gingival recession, gingival bleeding, and dental calculus in adults 30 years of age and older in the United States, 1988-1994. *J Periodontol* 1999;70:30-43.
15. Albandar JM, Rams TE. Global epidemiology of periodontal diseases: an overview. *Periodontol 2000* 2002;29:7-10.
16. Albandar JM, Rams TE. Risk factors for periodontitis in children and young persons. *Periodontol 2000* 2002;29:207-222.
17. Albandar JM, Tinoco EM. Global epidemiology of periodontal diseases in children and young persons. *Periodontol 2000* 2002;29:153-176.
18. Albandar JM, Buischi YA, Barbosa MF. Destructive forms of periodontal disease in adolescents. A 3-year longitudinal study. *J Periodontol* 1991;62:370-376.
19. Albandar JM, Buischi YA, Axelsson P. Caries lesions and dental restorations as predisposing factors in the progression of periodontal diseases in adolescents. A 3-year longitudinal study. *J Periodontol* 1995;66:249-254.
20. Albandar JM, Brown LJ, Loe H. Putative periodontal pathogens in subgingival plaque of young adults with and without early-onset periodontitis. *J Periodontol* 1997;68:973-981.
21. Albandar JM, Brown LJ, Loe H. Clinical features of early-onset periodontitis. *J Am Dent Assoc* 1997;128:1393-1399.
22. Albandar JM, Brunelle JA, Kingman A. Destructive periodontal disease in adults 30 years of age and older in the United States, 1988-1994. *J Periodontol* 1999;70:13-29.
23. Albandar JM, Muranga MB, Rams TE. Prevalence of aggressive periodontitis in school attendees in Uganda. *J Clin Periodontol* 2002;29:823-831.
24. Albandar JM, Brown LJ, Brunelle JA, Loe H. Gingival state and dental calculus in early-onset periodontitis. *J Periodontol* 1996;67:953-959.
25. Albandar JM, Kingman A, Brown LJ, Loe H. Gingival inflammation and subgingival calculus as determinants of disease progression in early-onset periodontitis. *J Clin Periodontol* 1998;25:231-237.
26. Albandar JM, Streckfus CF, Adesanya MR, Winn DM. Cigar, pipe, and cigarette smoking as risk factors for periodontal disease and tooth loss. *J Periodontol* 2000;71:1874-1881.
27. Albandar JM, DeNardin AM, Adesanya MR, Diehl SR, Winn DM. Associations between serum antibody levels to periodontal pathogens and early-onset periodontitis. *J Periodontol* 2001;72:1463-1469.
28. Alpagot T, Duzgunes N, Wolff LF, Lee A. Risk factors for periodontitis in HIV patients. *J Periodontol Res* 2004;39:149-157.
29. Amarasena N, Ekanayaka AN, Herath L, Miyazaki H. Tobacco use and oral hygiene as risk indicators for periodontitis. *Community Dent Oral Epidemiol* 2002;30:115-123.
30. Anagnou-Vareltzides A, Diamanti-Kipiotti A, Afentoulidis N, et al. A clinical survey of

- periodontal conditions in Greece. *J Clin Periodontol* 1996;23:758-763.
31. Associação Nacional de Empresas de Pesquisa. Economic Classification Criterion - Brazil. Sao Paulo:Associação Nacional de Empresas de Pesquisa; 1997.
 32. Armitage GC. Development of a classification system for periodontal diseases and conditions. *Ann Periodontol* 1999;4:1-6.
 33. Baelum V. Pattern of periodontal breakdown in adult Tanzanians. *Scand J Dent Res* 1987;95:221-228.
 34. Baelum V, Papapanou PN. CPITN and the epidemiology of periodontal disease. *Community Dent Oral Epidemiol* 1996;24:367-368.
 35. Baelum V, Papapanou PN. CPITN and the epidemiology of periodontal disease. *Community Dent Oral Epidemiol* 1996;24:367-368.
 36. Baelum V, Scheutz F. Periodontal diseases in Africa. *Periodontol 2000* 2002;29:79-103.
 37. Baelum V, Fejerskov O, Manji F. Periodontal diseases in adult Kenyans. *J Clin Periodontol* 1988;15:445-452.
 38. Baelum V, Fejerskov O, Manji F, Wanzala P. Influence of CPITN partial recordings on estimates of prevalence and severity of various periodontal conditions in adults. *Community Dent Oral Epidemiol* 1993;21:354-359.
 39. Baelum V, Manji F, Wanzala P, Fejerskov O. Relationship between CPITN and periodontal attachment loss findings in an adult population. *J Clin Periodontol* 1995;22:146-152.
 40. Baelum V, Chen X, Manji F, Luan WM, Fejerskov O. Profiles of destructive periodontal disease in different populations. *J Periodontal Res* 1996;31:17-26.
 41. Baelum V, Pisuthanakan S, Teanpaisan R, et al. Periodontal conditions among adults in Southern Thailand. *J Periodontal Res* 2003;38:156-163.
 42. Baer PN. The case for periodontosis as a clinical entity. *J Periodontol* 1971;42:516-520.
 43. Bakdash B. Oral hygiene and compliance as risk factors in periodontitis. *J Periodontol* 1994;65:539-544.
 44. Beck JD. Risk revisited. *Community Dent Oral Epidemiol* 1998;26:220-225.
 45. Beck JD, Koch GG. Characteristics of older adults experiencing periodontal attachment loss as gingival recession or probing depth. *J Periodontal Res* 1994;29:290-298.
 46. Beck JD, Koch GG, Rozier RG, Tudor GE. Prevalence and risk indicators for periodontal attachment loss in a population of older community-dwelling blacks and whites. *J Periodontol* 1990;61:521-528.
 47. Beck JD, Elter JR, Heiss G, Couper D, Mauriello SM, Offenbacher S. Relationship of periodontal disease to carotid artery intima-media wall thickness: the atherosclerosis risk in communities (ARIC) study. *Arterioscler Thromb Vasc Biol* 2001;21:1816-1822.
 48. Benichou J. A review of adjusted estimators of attributable risk. *Stat Methods Med Res* 2001;10:195-216.
 49. Bergstrom J, Eliasson S, Dock J. A 10-year prospective study of tobacco smoking and periodontal health. *J Periodontol* 2000;71:1338-1347.
 50. Borrell LN, Burt BA, Gillespie BW, Lynch J, Neighbors H. Periodontitis in the United States: beyond black and white. *J Public Health Dent* 2002;62:92-101.
 51. Borrell LN, Lynch J, Neighbors H, Burt BA, Gillespie BW. Is there homogeneity in periodontal health between African Americans and Mexican Americans? *Ethn Dis* 2002;12:97-110.
 52. Borrell LN, Taylor GW, Borgnakke WS, et al. Factors influencing the effect of race on established periodontitis prevalence. *J Public Health Dent* 2003;63:20-29.
 53. Braveman P, Gruskin S. Poverty, equity, human rights and health. *Bull World Health Organ* 2003;81:539-545.
 54. Brenner H. A potential pitfall in control of covariates in epidemiologic studies. *Epidemiology* 1998;9:68-71.
 55. Brenner H, Blettner M. Controlling for continuous confounders in epidemiologic research. *Epidemiology* 1997;8:429-434.
 56. Brown LJ, Garcia R. Utilization of dental services as a risk factor for periodontitis. *J Periodontol* 1994;65:551-563.
 57. Brown LJ, Albandar JM, Brunelle JA, Loe H. Early-onset periodontitis: progression of attachment loss during 6 years. *J Periodontol* 1996;67:968-975.
 58. Burt BA. Periodontitis and aging: reviewing recent evidence. *J Am Dent Assoc* 1994;125:273-279.
 59. Caldas Jr AF, Marcenes W, Sheiham A. Reasons for tooth extraction in a Brazilian population. *Int Dent J* 2000;50:267-273.
 60. Calsina G, Ramon JM, Echeverria JJ. Effects of smoking on periodontal tissues. *J Clin Periodontol* 2002;29:771-776.
 61. Campos Jr A, Passanezi E, Kim SH, Navarro MFL, Souza SLS. Identification of risk groups to periodontal disease in a population from Bauru city, Sao Paulo. *Rev Fac Odontol Bauru* 1994;2:20-28.
 62. Chen X, Wolff L, Aepli D, et al. Cigarette smoking, salivary/gingival crevicular fluid cotinine and periodontal status. A 10-year longitudinal study. *J Clin Periodontol*

- 2001;28:331-339.
63. Clerehugh V, Worthington HV, Lennon MA, Chandler R. Site progression of loss of attachment over 5 years in 14- to 19-year-old adolescents. *J Clin Periodontol* 1995;22:15-21.
 64. Contreras A, Zadeh HH, Nowzari H, Slots J. Herpesvirus infection of inflammatory cells in human periodontitis. *Oral Microbiol Immunol* 1999;14:206-212.
 65. Corbet E, Wong M, Lin H. Periodontal conditions in adult Southern Chinese. *J Dent Res* 2001;80:1480-1485.
 66. Corbet EF, Zee KY, Lo EC. Periodontal diseases in Asia and Oceania. *Periodontol 2000* 2002;29:122-152.
 67. Cortelli JR, Cortelli SC, Pallos D, Jorge AO. Prevalence of aggressive periodontitis in adolescents and young adults from Vale do Paraiba. *Pesqui Odontol Bras* 2002;16:163-168.
 68. Cortelli SC, Jorge AO, Cortelli JR, Jordan SF, Haraszthy VI. Detection of highly and minimally leukotoxic *Actinobacillus actinomycetemcomitans* strains in patients with periodontal disease. *Pesqui Odontol Bras* 2003;17:183-188.
 69. Davidson PL, Rams TE, Andersen RM. Socio-behavioral determinants of oral hygiene practices among USA ethnic and age groups. *Adv Dent Res* 1997;11:245-253.
 70. der Wiel AB, van Exel E, de Craen AJ, et al. A high response is not essential to prevent selection bias: results from the Leiden 85-plus study. *J Clin Epidemiol* 2002;55:1119-1125.
 71. Diamanti-Kipioti A, Papapanou PN, Moraitaki-Tsami A, Lindhe J, Mitsis F. Comparative estimation of periodontal conditions by means of different index systems. *J Clin Periodontol* 1993;20:656-661.
 72. Diehl SR, Wang Y, Brooks CN, et al. Linkage disequilibrium of interleukin-1 genetic polymorphisms with early-onset periodontitis. *J Periodontol* 1999;70:418-430.
 73. Diehl SR, Wu T, Burmeister JA, et al. Evidence of a substantial genetic basis for IgG2 levels in families with aggressive periodontitis. *J Dent Res* 2003;82:708-712.
 74. Dini EL, Guimaraes LO. Periodontal conditions and treatment needs (CPITN) in a worker population in Araraquara, SP, Brazil. *Int Dent J* 1994;44:309-311.
 75. Dini EL, Foschini AL, Brandao IM. Periodontal conditions in a 7-19-year-old student population in Araraquara, Sao Paulo, Brazil, 1995. *Cad Saude Publica* 1997;13:321-324.
 76. Dolan TA, Gilbert GH, Ringelberg ML, et al. Behavioral risk indicators of attachment loss in adult Floridians. *J Clin Periodontol* 1997;24:223-232.
 77. Douglass CW, Jette AM, Fox CH, et al. Oral health status of the elderly in New England. *J Gerontol* 1993;48:M39-46.
 78. Dowsett SA, Eckert GJ, Kowolik MJ. The applicability of half-mouth examination to periodontal disease assessment in untreated adult populations. *J Periodontol* 2002;73:975-981.
 79. Drury TF, Garcia I, Adesanya M. Socioeconomic disparities in adult oral health in the United States. *Ann N Y Acad Sci* 1999;896:322-324.
 80. Drury TF, Winn DM, Snowden CB, Kingman A, Kleinman DV, Lewis B. An overview of the oral health component of the 1988-1991 National Health and Nutrition Examination Survey (NHANES III-Phase 1). *J Dent Res* 1996;75 Spec No:620-630.
 81. Eid MA, Selim HA, al-Shammery AR. The relationship between chewing sticks (Miswak) and periodontal health. 3. Relationship to gingival recession. *Quintessence Int* 1991;22:61-64.
 82. Elter JR, Beck JD, Slade GD, Offenbacher S. Etiologic models for incident periodontal attachment loss in older adults. *J Clin Periodontol* 1999;26:113-123.
 83. Emrich LJ, Shlossman M, Genco RJ. Periodontal disease in non-insulin-dependent diabetes mellitus. *J Periodontol* 1991;62:123-131.
 84. Flemmig TF. Periodontitis. *Ann Periodontol* 1999;4:32-38.
 85. Flores-de-Jacoby L, Bruchmann S, Mengel R, Zafiroopoulos GG. Periodontal conditions in Rio de Janeiro City (Brazil) using the CPITN. *Community Dent Oral Epidemiol* 1991;19:127-128.
 86. 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;48:96-103.
 87. Gelskey SC. Cigarette smoking and periodontitis: methodology to assess the strength of evidence in support of a causal association. *Community Dent Oral Epidemiol* 1999;27:16-24.
 88. Genco RJ, Ho AW, Grossi SG, Dunford RG, Tedesco LA. Relationship of stress, distress and inadequate coping behaviors to periodontal disease. *J Periodontol* 1999;70:711-723.
 89. Gilbert GH, Heft MW. Periodontal status of older Floridians attending senior activities centers. *J Clin Periodontol* 1992;19:249-255.
 90. Gjermeo P, Rosing CK, Susin C, Oppermann R. Periodontal diseases in Central and South America. *Periodontol 2000* 2002;29:70-78.
 91. Gjermeo 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;11:104-113.
92. Gould W. sg124: Interpreting logistic regression in all its forms. *Stata Technical Bulletin* 2000;53:19-29.
 93. Grau AJ, Becher H, Ziegler CM, et al. Periodontal disease as a risk factor for ischemic stroke. *Stroke* 2004;35:496-501.
 94. Greenland S, Drescher K. Maximum likelihood estimation of the attributable fraction from logistic models. *Biometrics* 1993;49:865-872.
 95. Griffiths GS, Duffy S, Eaton KA, Gilthorpe MS, Johnson NW. Prevalence and extent of lifetime cumulative attachment loss (LCAL) at different thresholds and associations with clinical variables: changes in a population of young male military recruits over 3 years. *J Clin Periodontol* 2001;28:961-969.
 96. Grossi SG, Zambon JJ, Ho AW, et al. Assessment of risk for periodontal disease. I. Risk indicators for attachment loss. *J Periodontol* 1994;65:260-267.
 97. Gunsolley JC, Quinn SM, Tew J, Gooss CM, Brooks CN, Schenkein HA. The effect of smoking on individuals with minimal periodontal destruction. *J Periodontol* 1998;69:165-170.
 98. Haber J, Wattles J, Crowley M, Mandell R, Joshipura K, Kent RL. Evidence for cigarette smoking as a major risk factor for periodontitis. *J Periodontol* 1993;64:16-23.
 99. Haffajee AD, Socransky SS, Lindhe J, Kent RL, Okamoto H, Yoneyama T. Clinical risk indicators for periodontal attachment loss. *J Clin Periodontol* 1991;18:117-125.
 100. Haraszthy VI, Hariharan G, Tinoco EM, et al. Evidence for the role of highly leukotoxic *Actinobacillus actinomycetemcomitans* in the pathogenesis of localized juvenile and other forms of early-onset periodontitis. *J Periodontol* 2000;71:912-922.
 101. Harley AF, Floyd PD. Prevalence of juvenile periodontitis in schoolchildren in Lagos, Nigeria. *Community Dent Oral Epidemiol* 1988;16:299-301.
 102. Harris MI, Flegal KM, Cowie CC, et al. Prevalence of diabetes, impaired fasting glucose, and impaired glucose tolerance in U.S. adults. The Third National Health and Nutrition Examination Survey, 1988-1994. *Diabetes Care* 1998;21:518-524.
 103. Hart TC, Kornman KS. Genetic factors in the pathogenesis of periodontitis. *Periodontol 2000* 1997;14:202-215.
 104. Hart TC, Marazita ML, Schenkein HA, Brooks CN, Gunsolley JG, Diehl SR. No female preponderance in juvenile periodontitis after correction for ascertainment bias. *J Periodontol* 1991;62:745-749.
 105. Hartge P. Raising response rates: getting to yes. *Epidemiology* 1999;10:105-107.
 106. Hashim R, Thomson WM, Pack AR. Smoking in adolescence as a predictor of early loss of periodontal attachment. *Community Dent Oral Epidemiol* 2001;29:130-135.
 107. Hodge P, Michalowicz B. Genetic predisposition to periodontitis in children and young adults. *Periodontol 2000* 2001;26:113-134.
 108. Holmgren CJ, Corbet EF, Lim LP. Periodontal conditions among the middle-aged and the elderly in Hong Kong. *Community Dent Oral Epidemiol* 1994;22:396-402.
 109. Hosanguan C, Ungchusak C, Leelasithorn S, Prasertsom P. The extent and correlates of gingival recession in non-institutionalised Thai elderly. *J Int Acad Periodontol* 2002;4:143-148.
 110. Hosmer D, Lemeshow S. Applied logistic regression. 2nd ed. New York: Jhon Wiley & Sons; 2000: 260-287.
 111. Hubert L. Kappa revisited. *Psychol Bull* 1977;84:289-297.
 112. Hugoson A, Ljungquist B, Breivik T. The relationship of some negative events and psychological factors to periodontal disease in an adult Swedish population 50 to 80 years of age. *J Clin Periodontol* 2002;29:247-253.
 113. Hujoel PP, del Aguila MA, DeRouen TA, Bergstrom J. A hidden periodontitis epidemic during the 20th century? *Community Dent Oral Epidemiol* 2003;31:1-6.
 114. Hunt RJ. The efficiency of half-mouth examinations in estimating the prevalence of periodontal disease. *J Dent Res* 1987;66:1044-1048.
 115. Hunt RJ, Fann SJ. Effect of examining half the teeth in a partial periodontal recording of older adults. *J Dent Res* 1991;70:1380-1385.
 116. Hunt RJ, Levy SM, Beck JD. The prevalence of periodontal attachment loss in an Iowa population aged 70 and older. *J Public Health Dent* 1990;50:251-256.
 117. Hyman JJ, Reid BC. Epidemiologic risk factors for periodontal attachment loss among adults in the United States. *J Clin Periodontol* 2003;30:230-237.
 118. Hyman JJ, Reid BC. Cigarette smoking, periodontal disease: and chronic obstructive pulmonary disease. *J Periodontol* 2004;75:9-15.
 119. Brazilian Institute of Geography and Statistics. Demographics Census 1991 (in Portuguese). Rio de Janeiro: Brazilian Institute of Geography and Statistics; 1991.
 120. Brazilian Institute of Geography and Statistics. Population Census (in Portuguese). Rio de

- Janeiro:Brazilian Institute of Geography and Statistics; 1996.
121. Brazilian Institute of Geography and Statistics. Demographics Census 2000: Characteristics of the Population and Households- General Results (in Portuguese). Rio de Janeiro:Brazilian Institute of Geography and Statistics; 2001.
 122. Ismail AI, Morrison EC, Burt BA, Caffesse RG, Kavanagh MT. Natural history of periodontal disease in adults: findings from the Tecumseh Periodontal Disease Study, 1959-87. *J Dent Res* 1990;69:430-435.
 123. Jeffcoat MK, Hauth JC, Geurs NC, et al. Periodontal disease and preterm birth: results of a pilot intervention study. *J Periodontol* 2003;74:1214-1218.
 124. Jenkins WM, Papapanou PN. Epidemiology of periodontal disease in children and adolescents. *Periodontol 2000* 2001;26:16-32.
 125. Joshipura KJ, Kent RL, DePaola PF. Gingival recession: intra-oral distribution and associated factors. *J Periodontol* 1994;65:864-871.
 126. Kallestal C, Uhlin S. Buccal attachment loss in Swedish adolescents. *J Clin Periodontol* 1992;19:485-491.
 127. Kamma JJ, Nakou M, Baehni PC. Clinical and microbiological characteristics of smokers with early onset periodontitis. *J Periodontol Res* 1999;34:25-33.
 128. Kassab MM, Cohen RE. The etiology and prevalence of gingival recession. *J Am Dent Assoc* 2003;134:220-225.
 129. Kaufman JS, Poole C. Looking back on "causal thinking in the health sciences". *Annu Rev Public Health* 2000;21:101-119.
 130. Kaufman JS, Cooper RS, McGee DL. Socioeconomic status and health in blacks and whites: the problem of residual confounding and the resiliency of race. *Epidemiology* 1997;8:621-628.
 131. Kingman A, Albandar JM. Methodological aspects of epidemiological studies of periodontal diseases. *Periodontol 2000* 2002;29:11-30.
 132. Kingman A, Morrison E, Loe H, Smith J. Systematic errors in estimating prevalence and severity of periodontal disease. *J Periodontol* 1988;59:707-713.
 133. Kleinbaum DG, Kupper LL, Morgenstern H. Epidemiologic research. Belmont, California; 1982.
 134. Korn E, Graubard B. Analysis of health surveys. New York: John Wiley & Sons, Inc; 1999.
 135. Kornman KS, Page RC, Tonetti MS. The host response to the microbial challenge in periodontitis: assembling the players. *Periodontol 2000* 1997;14:33-53.
 136. Krieger N. Epidemiology and the web of causation: has anyone seen the spider? *Soc Sci Med* 1994;39:887-903.
 137. Krieger N, Zierler S. The need for epidemiologic theory. *Epidemiology* 1997;8:212-214.
 138. Kronauer E, Borsa G, Lang NP. Prevalence of incipient juvenile periodontitis at age 16 years in Switzerland. *J Clin Periodontol* 1986;13:103-108.
 139. Last J. A dictionary of epidemiology. 4th ed. New York: Orford University Press, Inc.; 2001.
 140. Lee E, Forthofer R, Lorimor R. Analyzing complex survey data. Sage University Paper series on Quantitative Applications in the Social Sciences, No. 07-071. In. Newbury Park, CA: Sage Pub, Inc.; 1989. p. 16-23.
 141. Levy P, Lemeshow S. Sampling of populations. Methods and applications. 3rd ed. New York: John Wiley & Sons, Inc; 1999:70-79.
 142. Linden GJ, Mullally BH. Cigarette smoking and periodontal destruction in young adults. *J Periodontol* 1994;65:718-723.
 143. Lindhe J, Hamp SE, Loe H. Plaque induced periodontal disease in beagle dogs. A 4-year clinical, roentgenographical and histometrical study. *J Periodontol Res* 1975;10:243-255.
 144. Litonjua LA, Andreana S, Bush PJ, Cohen RE. Toothbrushing and gingival recession. *Int Dent J* 2003;53:67-72.
 145. Locker D. Response and nonresponse bias in oral health surveys. *J Public Health Dent* 2000;60:72-81.
 146. Locker D, Leake JL. Risk indicators and risk markers for periodontal disease experience in older adults living independently in Ontario, Canada. *J Dent Res* 1993;72:9-17.
 147. Loe H, Brown LJ. Early onset periodontitis in the United States of America. *J Periodontol* 1991;62:608-616.
 148. Loe H, Theilade E, Jensen SB. Experimental Gingivitis in Man. *J Periodontol* 1965;36:177-187.
 149. 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;13:431-445.
 150. Loe H, Ånerud Å, Boysen H. The natural history of periodontal disease in man: prevalence, severity and extent of gingival recession. *J Periodontol* 1992;63:489-495.
 151. Lopez NJ, Smith PC, Gutierrez J. Periodontal therapy may reduce the risk of preterm low birth weight in women with periodontal disease: a randomized controlled trial. *J Periodontol* 2002;73:911-924.
 152. Lopez NJ, Rios V, Pareja MA, Fernandez O. Prevalence of juvenile periodontitis in Chile. *J*

- Clin Periodontol* 1991;18:529-533.
153. Lopez R, Baelum V. Classifying periodontitis among adolescents: implications for epidemiological research. *Community Dent Oral Epidemiol* 2003;31:136-143.
 154. Lopez R, Fernandez O, Jara G, Baelum V. Epidemiology of clinical attachment loss in adolescents. *J Periodontol* 2001;72:1666-1674.
 155. Lovdal A, Arno A, Waerhaug J. Incidence of clinical manifestations of periodontal disease in light of oral hygiene and calculus formation. *J Am Dent Assoc* 1958;56:21-33.
 156. Machtei EE, Dunford R, Hausmann E, et al. Longitudinal study of prognostic factors in established periodontitis patients. *J Clin Periodontol* 1997;24:102-109.
 157. Machtei EE, Hausmann E, Dunford R, et al. Longitudinal study of predictive factors for periodontal disease and tooth loss. *J Clin Periodontol* 1999;26:374-380.
 158. Machuca G, Rosales I, Lacalle JR, Machuca C, Bullon P. Effect of cigarette smoking on periodontal status of healthy young adults. *J Periodontol* 2000;71:73-78.
 159. Mariotti A. Dental plaque-induced gingival diseases. *Ann Periodontol* 1999;4:7-19.
 160. Martinez-Canut P, Lorca A, Magan R. Smoking and periodontal disease severity. *J Clin Periodontol* 1995;22:743-749.
 161. McPherson K. Wider "causal thinking in the health sciences". *J Epidemiol Community Health* 1998;52:612-613.
 162. Rio Grande do Sul State Agency for Metropolitan Affairs. Environmental cartographic data of the Metropolitan Area of Porto Alegre (in Portuguese). Porto Alegre:Rio Grande do Sul State Agency for Metropolitan Affairs; 1997.
 163. Miranda LA, Fischer RG, Sztajn bok FR, Figueredo CM, Gustafsson A. Periodontal conditions in patients with juvenile idiopathic arthritis. *J Clin Periodontol* 2003;30:969-974.
 164. Miyazaki H, Ohtani I, Abe N, et al. Periodontal conditions in older age cohorts aged 65 years and older in Japan, measured by CPITN and loss of attachment. *Community Dent Health* 1995;12:216-220.
 165. Morgenstern H. Defining and explaining race effects. *Epidemiology* 1997;8:609-611.
 166. Morita M, Kimura T, Kanegae M, Ishikawa A, Watanabe T. Reasons for extraction of permanent teeth in Japan. *Community Dent Oral Epidemiol* 1994;22:303-306.
 167. Morris AJ, Steele J, White DA. The oral cleanliness and periodontal health of UK adults in 1998. *Br Dent J* 2001;191:186-192.
 168. Ministry of Health. Oral health survey: Brazil, urban area, 1986 (in Portuguese):Ministry of Health; 1988.
 169. Mullally BH, Breen B, Linden GJ. Smoking and patterns of bone loss in early-onset periodontitis. *J Periodontol* 1999;70:394-401.
 170. Muller HP, Stadermann S, Heinecke A. Gingival recession in smokers and non-smokers with minimal periodontal disease. *J Clin Periodontol* 2002;29:129-136.
 171. Mumghamba EGS, Markkanen HA, Honkala E. Risk factors for periodontal diseases in Ilala, Tanzania. *J Clin Periodontol* 1995;22:347-354.
 172. Neely AL, Holford TR, Loe H, Anerud A, Boysen H. The natural history of periodontal disease in man. Risk factors for progression of attachment loss in individuals receiving no oral health care. *J Periodontol* 2001;72:1006-1015.
 173. Newman MG, Socransky SS, Savitt ED, Propas DA, Crawford A. Studies of the microbiology of periodontosis. *J Periodontol* 1976;47:373-379.
 174. 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;62:116-122.
 175. Oliveira JE, Milech A, Franco LJ. The prevalence of diabetes in Rio de Janeiro, Brazil. The Cooperative Group for the Study of Diabetes Prevalence in Rio De Janeiro. *Diabetes Care* 1996;19:663-666.
 176. Ong G, Yeo JF, Bhole S. A survey of reasons for extraction of permanent teeth in Singapore. *Community Dent Oral Epidemiol* 1996;24:124-127.
 177. Owens JD, Dowsett SA, Eckert GJ, Zero DT, Kowolik MJ. Partial-mouth assessment of periodontal disease in an adult population of the United States. *J Periodontol* 2003;74:1206-1213.
 178. Papapanou PN, Wennstrom JL. A 10-year retrospective study of periodontal disease progression. Clinical characteristics of subjects with pronounced and minimal disease development. *J Clin Periodontol* 1990;17:78-84.
 179. Papapanou PN, Wennstrom JL, Grondahl K. A 10-year retrospective study of periodontal disease progression. *J Clin Periodontol* 1989;16:403-411.
 180. Papapanou PN, Wennstrom JL, Johnsson T. Extent and severity of periodontal destruction based on partial clinical assessments. *Community Dent Oral Epidemiol* 1993;21:181-184.
 181. Papapanou PN, Lindhe J, Sterrett JD, Eneroth L. Considerations on the contribution of ageing to loss of periodontal tissue support. *J Clin Periodontol* 1991;18:611-615.
 182. Parra B, Slots J. Detection of human viruses in periodontal pockets using polymerase chain

- reaction. *Oral Microbiol Immunol* 1996;11:289-293.
183. Payne JB, Reinhardt RA, Nummikoski PV, Patil KD. Longitudinal alveolar bone loss in postmenopausal osteoporotic/osteopenic women. *Osteoporos Int* 1999;10:34-40.
 184. Pearce N. Traditional epidemiology, modern epidemiology, and public health. *Am J Public Health* 1996;86:678-683.
 185. Preus HR, Anerud A, Boysen H, Dunford RG, Zambon JJ, Loe H. The natural history of periodontal disease. The correlation of selected microbiological parameters with disease severity in Sri Lankan tea workers. *J Clin Periodontol* 1995;22:674-678.
 186. Ramfjord SP. Indices for prevalence and incidence of periodontal disease. *J Periodontol* 1959;30:51-59.
 187. Reinhardt RA, Payne JB, Maze CA, Patil KD, Gallagher SJ, Mattson JS. Influence of estrogen and osteopenia/osteoporosis on clinical periodontitis in postmenopausal women. *J Periodontol* 1999;70:823-828.
 188. Robertson PB, Walsh M, Greene J, Ernster V, Grady D, Hauck W. Periodontal effects associated with the use of smokeless tobacco. *J Periodontol* 1990;61:438-443.
 189. Rockhill B, Newman B, Weinberg CR. Use and misuse of population attributable fractions. *Am J Public Health* 1998;88:15-19.
 190. Sangnes G, Gjermo P. Prevalence of oral soft and hard tissue lesions related to mechanical toothcleansing procedures. *Community Dent Oral Epidemiol* 1976;4:77-83.
 191. Savitz DA. In defense of black box epidemiology. *Epidemiology* 1994;5:550-552.
 192. Savitz DA. The alternative to epidemiologic theory: whatever works. *Epidemiology* 1997;8:210-212.
 193. Saxby MS. Juvenile periodontitis: an epidemiological study in the west Midlands of the United Kingdom. *J Clin Periodontol* 1987;14:594-598.
 194. Saxen L. Prevalence of juvenile periodontitis in Finland. *J Clin Periodontol* 1980;7:177-186.
 195. Scannapieco FA. Position paper of The American Academy of Periodontology: periodontal disease as a potential risk factor for systemic diseases. *J Periodontol* 1998;69:841-850.
 196. Scannapieco FA, Ho AW. Potential associations between chronic respiratory disease and periodontal disease: analysis of National Health and Nutrition Examination Survey III. *J Periodontol* 2001;72:50-56.
 197. Schatzle M, Loe H, Burgin W, Anerud A, Boysen H, Lang NP. Clinical course of chronic periodontitis. I. Role of gingivitis. *J Clin Periodontol* 2003;30:887-901.
 198. Schatzle M, Loe H, Lang NP, et al. Clinical course of chronic periodontitis. III. Patterns, variations and risks of attachment loss. *J Clin Periodontol* 2003;30:909-918.
 199. Schei O, Wærhaug J, Lovdal A, Arno A. Alveolar bone loss as related to oral hygiene and age. *J Periodontol* 1959;30:7-16.
 200. Schenkein HA, Van Dyke TE. Early-onset periodontitis: systemic aspects of etiology and pathogenesis. *Periodontol 2000* 1994;6:7-25.
 201. Schenkein HA, Gunsolley JC, Koertge TE, Schenkein JG, Tew JG. Smoking and its effects on early-onset periodontitis. *J Am Dent Assoc* 1995;126:1107-1113.
 202. Schwartz S, Susser E, Susser M. A future for epidemiology? *Annu Rev Public Health* 1999;20:15-33.
 203. Serino G, Wennstrom JL, Lindhe J, Eneroth L. The prevalence and distribution of gingival recession in subjects with a high standard of oral hygiene. *J Clin Periodontol* 1994;21:57-63.
 204. Sheiham A. Periodontal disease and oral cleanliness in tobacco smokers. *J Periodontol* 1971;42:259-263.
 205. Sheiham A, Netuveli GS. Periodontal diseases in Europe. *Periodontol 2000* 2002;29:104-121.
 206. Ship JA, Beck JD. Ten-year longitudinal study of periodontal attachment loss in healthy adults. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 1996;81:281-290.
 207. Shrout P, Fleiss J. Intraclass correlations: uses in assessing rater reliability. *Psychol Bull* 1979;86:420-428.
 208. Skrabanek P. The emptiness of the black box. *Epidemiology* 1994;5:553-555.
 209. Slade GD, Spencer AJ. Periodontal attachment loss among adults aged 60+ in South Australia. *Community Dent Oral Epidemiol* 1995;23:237-242.
 210. Slots J. The predominant cultivable organisms in juvenile periodontitis. *Scand J Dent Res* 1976;84:1-10.
 211. Slots J. The predominant cultivable microflora of advanced periodontitis. *Scand J Dent Res* 1977;85:114-121.
 212. Slots J, Ting M. Actinobacillus actinomycetemcomitans and Porphyromonas gingivalis in human periodontal disease: occurrence and treatment. *Periodontol 2000* 1999;20:82-121.
 213. Slots J, Contreras A. Herpesviruses: a unifying causative factor in periodontitis? *Oral Microbiol Immunol* 2000;15:277-280.
 214. Smith RG. Gingival recession. Reappraisal of an enigmatic condition and a new index for monitoring. *J Clin Periodontol* 1997;24:201-205.
 215. Stang A, Jockel KH. Studies with low response proportions may be less biased than studies

- with high response proportions. *Am J Epidemiol* 2004;159:204-210.
216. Suda R, Cao C, Hasegawa K, Yang S, Sasa R, Suzuki M. 2-year observation of attachment loss in a rural Chinese population. *J Periodontol* 2000;71:1067-1072.
 217. Susser M. Does risk factor epidemiology put epidemiology at risk? Peering into the future. *J Epidemiol Community Health* 1998;52:608-611.
 218. Susser M, Susser E. Choosing a future for epidemiology: II. From black box to Chinese boxes and eco-epidemiology. *Am J Public Health* 1996;86:674-677.
 219. Susser M, Susser E. Choosing a future for epidemiology: I. Eras and paradigms. *Am J Public Health* 1996;86:668-673.
 220. Taylor GW, Burt BA, Becker MP, Genco RJ, Shlossman M. Glycemic control and alveolar bone loss progression in type 2 diabetes. *Ann Periodontol* 1998;3:30-39.
 221. Teng HC, Lee CH, Hung HC, et al. Lifestyle and psychosocial factors associated with chronic periodontitis in Taiwanese adults. *J Periodontol* 2003;74:1169-1175.
 222. Teng YT, Taylor GW, Scannapieco F, et al. Periodontal health and systemic disorders. *J Can Dent Assoc* 2002;68:188-192.
 223. Thomson WM, Williams SM. Partial- or full-mouth approaches to assessing the prevalence of and risk factors for periodontal disease in young adults. *J Periodontol* 2002;73:1010-1014.
 224. Thomson WM, Hashim R, Pack AR. The prevalence and intraoral distribution of periodontal attachment loss in a birth cohort of 26-year-olds. *J Periodontol* 2000;71:1840-1845.
 225. Thomson WM, Slade GD, Beck JD, Elter JR, Spencer AJ, Chalmers JM. Incidence of periodontal attachment loss over 5 years among older South Australians. *J Clin Periodontol* 2004;31:119-125.
 226. Timmerman MF, Van der Weijden GA, Abbas F, et al. Untreated periodontal disease in Indonesian adolescents. Longitudinal clinical data and prospective clinical and microbiological risk assessment. *J Clin Periodontol* 2000;27:932-942.
 227. Timmerman MF, Van der Weijden GA, Arief EM, et al. Untreated periodontal disease in Indonesian adolescents. Subgingival microbiota in relation to experienced progression of periodontitis. *J Clin Periodontol* 2001;28:617-627.
 228. Tinoco EM, Sivakumar M, Preus HR. The distribution and transmission of *Actinobacillus actinomycetemcomitans* in families with localized juvenile periodontitis. *J Clin Periodontol* 1998;25:99-105.
 229. Tinoco EM, Beldi MI, Loureiro CA, et al. Localized juvenile periodontitis and *Actinobacillus actinomycetemcomitans* in a Brazilian population. *Eur J Oral Sci* 1997;105:9-14.
 230. Tomar SL, Asma S. Smoking-attributable periodontitis in the United States: findings from NHANES III. National Health and Nutrition Examination Survey. *J Periodontol* 2000;71:743-751.
 231. Tomita NE, Chinellato LE, Pernambuco RA, Lauris JR, Franco LJ. [Periodontal conditions and diabetes mellitus in the Japanese-Brazilian population]. *Rev Saude Publica* 2002;36:607-613.
 232. Tonetti MS, Mombelli A. Early-onset periodontitis. *Ann Periodontol* 1999;4:39-53.
 233. Trovik TA, Klock KS, Haugejorden O. Trends in reasons for tooth extractions in Norway from 1968 to 1998. *Acta Odontol Scand* 2000;58:89-96.
 234. U.S. Department of Health and Human Services. National Center for Health Statistics. Centers for Disease Control and Prevention. Analytic and reporting guidelines: The Third National Health and Nutrition Examination Survey, NHANES III (1988-94). Hyattsville, Maryland: U.S. Department of Health and Human Services. National Center for Health Statistics. Centers for Disease Control and Prevention; 1996.
 235. Van der Velden U, Abbas F, Van Steenberghe TJ, et al. Prevalence of periodontal breakdown in adolescents and presence of *Actinobacillus actinomycetemcomitans* in subjects with attachment loss. *J Periodontol* 1989;60:604-610.
 236. van Palenstein Helderma WH, Lembariti BS, van der Weijden GA, van 't Hof MA. Gingival recession and its association with calculus in subjects deprived of prophylactic dental care. *J Clin Periodontol* 1998;25:106-111.
 237. Vignarajah S. Various reasons for permanent tooth extractions in a Caribbean population--Antigua. *Int Dent J* 1993;43:207-212.
 238. Vineis P. Causality in epidemiology. *Soz Praventivmed* 2003;48:80-87.
 239. Wagstaff A. Poverty and health sector inequalities. *Bull World Health Organ* 2002;80:97-105.
 240. Walter SD. The estimation and interpretation of attributable risk in health research. *Biometrics* 1976;32:829-849.
 241. Yoneyama T, Okamoto H, Lindhe J, Socransky SS, Haffajee AD. Probing depth, attachment loss and gingival recession. Findings from a clinical examination in Ushiku, Japan. *J Clin Periodontol* 1988;15:581-591.

Appendix

QUALITY ASSURANCE

Interview

A pilot version of the questionnaire contained open-ended and close-ended questions and was developed and evaluated prior to the start of the survey. Detailed interviews with 20 subjects of various age, gender, race and socio-economic backgrounds were performed. The questionnaire was then refined and included mainly close-ended questions, and was re-evaluated through interviews with 30 new subjects. Some questions were eliminated because no reliable answers could be obtained.

Interview consistency was improved through training of interviewer and standardization of procedures. The interviewer was trained to follow the following rules:

- Avoid stating own views
- Follow the sequence of questions in the questionnaire form
- Attempt to use the same wording as stated in the questionnaire form
- Attain uniformity in asking questions and recording answers
- Avoid probing for different answers than what the participants actually stated
- Exercise permissive attitude.

In order to minimize error during the fieldwork, all interview and clinical data forms were reviewed by the participating dentists before moving the examination center to the next sector. When missing interview data were identified, attempts were made to re-interview the subject to gather the missing information. Unattainable data were scored as missing.

Clinical Examinations

One of the 4 examiners (C.S.) was an experienced periodontist and served as the “gold standard” examiner. The intra-examiner reproducibility of the standard examiner in assessing periodontal measurements was assessed. The standard examiner then trained and calibrated the other 3 examiners in performing the clinical measurements in 2 phases at two different time points: prior to and 3 months after the start of the fieldwork. Initially, clinical parameters, measurement instruments, and the correct assessment technique were reviewed and discussed. After a demonstration of the clinical examination by the standard examiner, the other 3 examiners performed a supervised examination on patients. Fifty-seven subjects were examined in the first training/calibration phase (before the start of study), and 51 subjects in the second (3 months after commencement of fieldwork). During the fieldwork, the 4 examiners followed a quality control protocol designed to minimize systematic and random measurement errors. The protocol involved standard examination environment and methodology, standard equipment, and detailed written instructions for clinical procedures.

NON-RESPONSE AND MISSING INFORMATION

In the present study 65.1% of eligible individuals were interviewed and clinically examined. The following steps were taken to increase the response rate for the interview and clinical examinations:

- a) Letters explaining the aims of the study were sent to eligible participants in advance.
- b) A dentist visited the selected households and explained the aims of the study encouraging participation of eligible persons.
- c) Flexible examination schedules were adopted to suit the needs of each household/individual.
- d) A follow-up system was used to reach and encourage individuals who refused to participate or did not attend a scheduled examination.
- e) A written report describing the oral health status of the examined participant was provided.
- f) Participants were made aware of any significant oral health problems and were referred to one of two university hospitals (Lutheran University, Canoas, or the Federal University of Rio Grande do Sul, Porto Alegre Brazil).

One area sector, out of the 30 selected, was not sampled because the public administrative supervisor did not grant our request to access the residential areas in that sector. In the remaining 29 sectors, response from 142 (15.4%) households could not be achieved. The main reasons for non-response in these households were that the house residents were not at home, or that access was denied. Multiple attempts were made to contact nonresponding households during the time period the examination team was in the same area.

The total number of non-respondents was 849 (34.9%) individuals. Among these, 636 (26.1%) individuals were not at home, 127 (5.2%) individuals refused to participate, 26 (1.1%) individuals were disabled and 60 (2.5%) individuals who participated in the interview, did not participate in the clinical examination.

Every effort was made to ensure that all participants answered the core questions of the questionnaire. This contributed to a very low frequency of missing values for these variables, including age, gender, race, socio-economic status, smoking status and general health. Of the whole study group, 5 subjects were examined clinically for various oral health parameters, but their clinical periodontal status could not be assessed due to their uncontrollable spastic movements.

Acceptable response rates are very difficult to achieve and different figures have been proposed in the literature.^{105,145} Furthermore, it has been demonstrated that high response rates do not guarantee small selection bias and studies with low participation will not necessarily provide biased estimates.^{70,215} In the present study the overall response rate was 65%, in contrast 74.1% of subjects selected for the oral examination component of the NHANES III were interviewed and examined.^{80,234} It is important to acknowledge that the mentioned U.S. survey employed strategies not used in the present study such as extensive outreach and publicity, and personal incentives like cash payments.^{80,234}

Non-response Data Analysis

Subsequent to the completion of the examinations, a simple random sample of 339 (39.9%) subjects was selected out of 849 eligible subjects who either had refused to participate or were unavailable during the normal

survey schedule. Attempts were made to contact these subjects by telephone in order to collect data for the non-response analysis. Individuals who did not own a telephone were contacted at work or through another family member, or by a neighbor who had a telephone. Of the 339 subjects selected for the interview, 50 (14.7%) subjects and their household were not available on 2 telephone call attempts, and an additional 18 (5.3%) subjects refused to be interviewed.

Non-response data was obtained for 271 (79.9%) subjects. Of these, 127 subjects were present and agreed to the telephone interview. The other 144 subjects were not available on 2 telephone call attempts, and the non-response data were therefore obtained through a first-degree relative living in the same household. The information collected included the subject's gender, age, education, dental care visits, and income level. In addition, information about the number of teeth present was obtained from the 127 subjects who were interviewed by telephone. The results of the non-response study are shown in Table 2.

Table 2. Percentage of subjects (n=127) who were personally interviewed by telephone, by the stated reason for not participating in the survey.

Reason for non-response	n	%
Away from home when the survey took place	48	37.8
Not interested	41	32.3
Did not have time	19	14.9
Did not know enough about the study	9	7.1
Afraid of the clinical examination	7	5.5
Other reasons	3	2.4
Total	127	100.0

The non-respondents had a mean age of 35.2 (SD: 13.3) years, 51.3% were males, and 90.8% were whites. In contrast, the mean age of the study group was 38 (SD: 17.4) years, 45.3% were males, and 82.5% whites. In the non-respondent and study groups, respectively, 7.4% and 22.3% of subjects had 4 or fewer years of education, 22.5% and 40.0% of subjects had 5 to 8 years, and 70.1% and 37.8% had more than 8 years. This suggests that the non-respondents were similar to the study group in their mean age, but included somewhat higher percentages of males and whites, and had a higher number of years of education. Given the discrepancy in some of the demographic features and the level of education between the study participants and subjects that did not wish to participate, a weight variable was introduced to minimize the bias in the estimation of population parameters (for more information, see Appendix III).

Assessment of the Bias Due to Non-response

The Double Sampling methodology¹⁴¹ was used to estimate the bias due to non-response. Data about tooth loss was used to estimate the impact of non-response in the present study. Tooth loss data gathered from the study sample, and corresponding data gathered from the non-respondents by telephone interview were used to calculate the double sampling estimate (\bar{x}_{dub}) which was considered the most accurate estimate of the true population parameter.

$$\bar{x}_{dub} = \frac{(n_1 \bar{x}_1 + n_2 \bar{x}_2)}{n_1 + n_2}$$

where:

n_1 is the number of subjects examined in the survey (1,586 individuals).

n_2 is the number of subjects not responding to the survey (849 individuals).

\bar{x}_1 is the estimate of parameter among respondents that were examined in the survey.

\bar{x}_2 is the estimate of the corresponding parameter among non-respondents who were interviewed by telephone.

Three parameters were assessed: prevalence of tooth loss and edentulism, and mean tooth loss. Bias due to non-response is defined as

$$Bias_{\bar{x}} = \left(\frac{n_2}{n_1} \right) \times (\bar{x}_1 - \bar{x}_2)$$

The estimate of the magnitude of bias due to non-response in this study was small, amounting to 2.6% and 1.3% for the prevalence of tooth loss and edentulism, respectively, and 0.8 teeth for mean tooth loss (Table 3).

Table 3. Estimates of the bias in tooth loss due to non-response

	Prevalence of tooth loss (%)	Prevalence of edentulism (%)	Mean tooth loss
Estimates among respondents*	73.6	7.6	7.7
Estimates among non-respondents	66.1	3.9	5.3
True population estimates (\bar{x}_{dub})	71.0	6.3	6.9
Bias \bar{x}	-2.6	-1.3	-0.8

* Not accounting for the complex sampling design (assuming simple random sampling)

Assuming that the double sampling estimates are the less biased estimates for tooth loss in the present population, a comparison between these estimates and those obtained after considering the complex sample design and using the sample weight could provide some insights into the ability of the present weighing scheme to adjust for sampling inaccuracies. The comparison between the weighted estimates and those obtained by the double sampling strategy showed relatively small differences in the mean tooth loss and prevalence of tooth loss and edentulism. It is important to acknowledge that the use a self-reported number of teeth by those that did not participate in the survey is prone to error. Nevertheless, it is unlikely that such inaccuracy would be of a great magnitude, and the information regarding edentulism is very likely to be fairly accurate. Therefore, it seems reasonable to conclude that the bias due to non-response in the present study did not jeopardize the results in a great extent.

SAMPLING WEIGHT AND DESIGN EFFECT

The distributions of the study sample by gender, age, and education level were compared to corresponding distributions in the target population which were provided by IBGE. The difference between the sample and the population, in the proportions of subjects by various gender and age groups ranged between 0.2 and 2.6 percentage points (Table 1). There is at present no current data about the distribution of the Porto Alegre population by race. However, comparison of the sample distribution by race and pre-existing data revealed no major differences (Table 4). Contrasting the distributions of the sample and the target population by the level of education showed that the largest difference (4.5% points) was in the percentage of males with ≤ 4 years of education (Table 5)

Table 4. Distribution of the sample and target population according to race.

Race/ ethnicity*	Sample		Population
	n	%	%
White	1309	82.5	86.7
Non-white	277	17.5	13.3
Total	1586	100.0	100.0

* IBGE – National Household Survey (PNAD), Porto Alegre Metropolitan area – Table 262, 2001.

Table 5. Distribution of the sample and target population according to gender and years of education.

Education (years)	Sample				Target population*	
	Male		Female		Male†	Female‡
	n	%	n	%	%	%
0 – 4	124	7.8	228	14.4	12.3	14.4
5 – 8	312	19.7	322	20.3	19.9	20.9
≥ 9	283	17.9	316	19.9	15.3	17.3
Total	719	45.4	866	54.6	47.4	52.6

* IBGE – Population census (Contagem da População), Porto Alegre Metropolitan area, 1996.

† N=1,241,926

‡ N=1,375,868

Analysis of the non-response data showed a somewhat higher proportion of males, whites and individuals with more years of education among those that did not participate in the study. A weight variable was therefore used to adjust for the potential bias in the population estimates.¹³⁴ The calculation of this weight variable used census information provided by IBGE.¹²⁰

The sample weight adjusted for the probability of selection and population distribution according to age,

gender and education. Probability of selection was calculated separately for the two economic strata and the population was divided by the number of individuals sampled in each area (base weight=N/n). This procedure also permitted achieving the expansion weight. The distribution of the population (poststratification) was calculated using the 1996 census information for the metropolitan area of Porto Alegre. The sample and the population were divided into various subgroups of males/females, 14-19/20-29/30-39/40-49/50-59/60-69/70+ years of age, and <4/5-8/>9 years of education. Some cells had small numbers of subjects. Therefore, subjects 60 years and older were collapsed into one age group. The final sample weight variable was calculated by multiplying the base weight with the post-stratification adjustment, yielding 34 sample weights. The largest sample weight was 4623, and the smallest was 769. No constraints were imposed on the values that the sample weight could have.

The post-stratification adjustment is an attempt to make the composition of the sample similar to the composition of the target population. This procedure is expected to improve the precision of the estimates, and may reduce non-response and sample selection bias to the extent that it is related to the demographic composition.^{140,141} Post-stratification has been characterized as a robust technique for estimation, offering some protection against extreme sample configurations, non-response and other problems in sample selection.^{140,141}

Design Effect

The use of a simple random sampling strategy is not always a feasible survey design due to lack of sampling frame, logistic constraints, and other limitations. Therefore, in this study a multistage sampling design was used. The use of complex survey design may affect estimates of parameter variance. This occurs because there is a higher correlation (i.e. intra-cluster correlation) between units (i.e. subjects) sampled in a given cluster (i.e. geographical area, or area sector) than expected if a simple random sample was drawn. This correlation increases homogeneity yielding greater standard errors and wider confidence intervals. In this study, the magnitude of the design effect on the parameter variance was estimated relative to a simple random sample. The calculated design effect for the overall prevalence of CAL ≥5mm was 1.28 and for the overall percentage of teeth with CAL ≥5mm was 1.12. It has been suggested that this ratio could be interpreted to indicate how much should the present complex design sample be inflated in order to achieve the same precision using a simple random sample.¹⁴¹ For comparison, analysis of the NHANES III indicated that the average design effect ranged between 1.2 and 1.3,²³⁴ which is of about the same magnitude as observed for important variables in the present study.

$$Design\ effect = \frac{\hat{V}(\hat{\theta})_{dsg}}{\hat{V}(\hat{\theta})_{srs}}$$

where:

$\hat{V}(\hat{\theta})_{dsg}$ is the design-based estimate of variance

$\hat{V}(\hat{\theta})_{srs}$ is the estimate of the variance obtained from a similar hypothetical survey conducted using simple random sampling

Paper I

Periodontal Attachment Loss in an Urban Population of Brazilian Adults: Effect of Demographic, Behavioral, and Environmental Risk Indicators

Cristiano Susin,*†‡ Caroline F. Dalla Vecchia,† Rui V. Oppermann,†§ Ola Haugejorden,† and Jasim M. Albandar*

Background: There is little information about the occurrence and risk factors of periodontal diseases in developing countries. This study describes the clinical attachment loss (CAL) in an adult Brazilian population and performs a risk assessment of demographic, behavioral, and environmental exposures.

Methods: A representative sample of 853 dentate individuals (age: 30 to 103 years) was selected by a multistage probability sampling method. The subjects had a full-mouth clinical examination of six sites per tooth and were interviewed using a structured written questionnaire.

Results: Seventy-nine percent (79%) and 52% of the subjects and 36% and 16% of the teeth per subject had CAL ≥ 5 and ≥ 7 mm, respectively. A multivariable model showed that 40 to 49 and ≥ 50 years olds had 3.0 and 5.9 times higher risk for moderate CAL and 7.4 and 25.4 times higher risk for severe CAL, compared to the 30 to 39 years olds. Moderate cigarette smokers had a significantly higher risk for moderate (relative risk ratio [RRR] = 2.1) and severe CAL (RRR = 3.4), and heavy smokers had a higher risk for moderate (RRR = 3.0) and severe CAL (RRR = 8.2) compared to non-smokers. A significantly higher risk for severe CAL was also present in males (RRR = 1.6), subjects with low (RRR = 1.8) or medium socioeconomic status (RRR = 1.6), and those with a history of irregular dental visits (RRR = 2.1). Diabetic status and race did not show significant associations with CAL after adjusting for other effects.

Conclusions: This Brazilian population had a high occurrence of attachment loss. A population-based strategy that includes the establishment of prevention and health promotion programs targeting high-risk groups is highly desirable for controlling the high occurrence of attachment loss in this population. *J Periodontol* 2004;75:1033-1041.

KEY WORDS

Brazilians; periodontal ligament attachment loss/epidemiology; public health programs; risk factors; smoking/adverse effects.

Periodontal diseases are a group of chronic inflammatory diseases that affect the supporting tissues of teeth and share common clinical manifestations. Periodontitis affects a relatively high percentage of the adult population in developed¹⁻³ and developing countries.⁴⁻⁷ In addition to its effect on function and esthetics, periodontitis has been associated with certain systemic diseases including cardiovascular diseases,⁸ adverse pregnancy outcome,⁹ bacterial respiratory diseases,¹⁰ diabetes mellitus,¹¹ and other diseases.

Prevalence of periodontal disease varies significantly in different regions of the world,^{12,13} and there are indications that the disease may be more prevalent in developing than in developed nations. A national survey in the United States used a partial recording system and estimated that 19.9% and 7.3% of subjects aged 30 to 90 years had clinical attachment loss (CAL) ≥ 5 and ≥ 7 mm, respectively.^{2,3} A national survey in the United Kingdom used the Community Periodontal Index (CPI) and estimated that 42% of 35 to 44 year olds and 70% of 55 to 64 year olds had CAL >3.5 mm.¹ Other studies reported prevalence rates of CAL ≥ 4 mm amounting to 56% to 61% among 35 to 44 year olds and 48% to 55% among 65 to 74 year olds in Southern China,⁶ and 90% among subjects 35 years and older in Kenya.⁴

Several risk factors and risk indicators have been associated with the occur-

* Department of Periodontology, Temple University School of Dentistry, Philadelphia, PA.

† Department of Odontology, University of Bergen, Bergen, Norway.

‡ Department of Periodontology, Lutheran University of Brazil, Canoas, Brazil.

§ Department of Periodontology, Federal University of Rio Grande do Sul, Porto Alegre, Brazil.

rence of destructive forms of periodontal diseases.¹³ Strong evidence suggests that cigarette smoking¹⁴ and diabetes mellitus¹⁵ are important risk factors for CAL. Other factors, including age, gender, race, socioeconomic status, and specific subgingival bacteria, have been associated with periodontal diseases but their role as risk factors of disease has not been fully substantiated.¹³

There is little information about the occurrence of periodontal diseases in developing countries, and the data are particularly sparse for Latin America and Brazil.¹⁶ Moreover, few studies have addressed the relationship of periodontal attachment loss with potential risk factors or risk indicators using a valid population-based study design.

This study was carried out to assess the occurrence of periodontal attachment loss in a sample representative of a large segment of the adult urban population in the Brazilian state of Rio Grande do Sul and to assess the association of demographic, behavioral, and environmental variables with the occurrence of attachment loss in this population.

MATERIALS AND METHODS

Study Design

This study was a cross-sectional survey. The target population was adults aged 30 years and older living in the metropolitan area of Porto Alegre in the Brazilian state of Rio Grande do Sul, located in the southern part of Brazil, neighboring Argentina and Uruguay. The survey covered 14 major municipalities from the Porto Alegre metropolitan area.

Study Sample

The total study sample included 974 individuals, age range 30 to 103 years (Table 1), of whom 121 were edentulous.

Table 1.

Age and Gender Distribution of Study Sample and Target Population

Age (years)	Study Sample				Target Population	
	Males		Females		Males	Females
	Number	%	Number	%	N = 773,739 %	N = 898,047 %
30-39	137	14.1	160	16.4	17.5	18.9
40-49	109	11.2	151	15.5	13.2	14.5
50-59	91	9.3	109	11.2	7.9	9.1
60-69	58	6.0	69	7.1	4.9	6.5
≥70	33	3.4	57	5.9	2.8	4.8
Total	428	43.9	546	56.1	46.3	53.7

Percentages were estimated based on the 1996 population census.^{17,18}

The periodontal findings reported pertain to the 853 dentate subjects (388 [45.5%] males and 465 [54.5%] females; 686 [80.4%] white and 167 [19.6%] non-white). Data for 10 subjects were not complete for some variables and were excluded. The mean tooth loss was 9.2 (SD: 7.2); ranging from 5.2 (SD: 5.0) among 30 to 39 year olds to 16.2 (SD: 6.9) for persons ≥70 years.

Sampling Procedures

The study sample was drawn from a larger sample representative of subjects aged 14 years and older among the population of Porto Alegre, which was derived using a multistage probability sampling method and based on information provided by Rio Grande do Sul state government agency for metropolitan affairs (METROPLAN) and the Brazilian Institute of Geography and Statistics (IBGE). Using area maps, the Porto Alegre metropolitan area was divided into 90 geographic areas 10 km² each. Using the 1991 census data¹⁹ and other relevant municipal information,²⁰ these geographic areas were stratified into 13 (14.4%) high-income and 77 (85.6%) low-income status areas. Low-income geographic areas were defined as those in which more than 40% of the heads of the households had a monthly income less than two standard Brazilian salaries (about \$180), and high-income areas were those with a higher level of income. Within each of these two income strata, primary sampling units (PSU) were randomly selected with a probability proportional to size and using a sampling frame of these PSUs. A total of 11 geographic areas were selected, two (18.2%) areas with high and nine (81.8%) areas with low-income status.

The second stage consisted of selecting area sectors within each geographic area, which are defined by IBGE as map areas comprising approximately 300 households each. The sectors were selected randomly within each geographic area with the number proportional to the number of sectors in each area. Thirty (3.5%) of the 846 eligible sectors were selected. Permission and/or support to conduct the study were granted by key community, religious, and/or administrative leaders in 29 of the sectors.

The third stage included selecting households within each of the 29 sectors. It was estimated that approximately 25 households were needed per sector to provide a sufficient number of subjects for the sample. In each sector, a starting point for the selection of households was established on area maps that were provided by the IBGE. Households were sampled consecutively beginning with the next block after the starting point and until the preset number of households was reached.

Consenting household members who were 14 years of age or older were examined and subjects ≥30 years were included in this study. Exclusion criteria were presence of diseases/conditions that may pose health

risks to the participant or examiner or that may interfere with the clinical examination. Hence, subjects were excluded if they were diagnosed with psychiatric problems, or intoxicated with alcohol or drugs. Individuals requiring a prophylactic regimen of antibiotics were provided with the appropriate medicine before the clinical examination.

Operational Procedures

The clinical examinations were performed in a mobile examination unit consisting of a trailer equipped with a complete dental unit, comprising a dental chair, light, compressor, and other basic amenities. The unit was moved from one location to the next according to the survey schedule. Four periodontists assisted by two dental assistants performed the clinical examinations. The fieldwork was completed between June and December 2001. Letters explaining the aims of the study and soliciting participation were sent to households. A few days later, a dentist visited the households and provided more information on the study and encouraged participation. Eligible subjects who consented to participation were interviewed to gather demographic, socioeconomic status, oral health, and other health-related data using a structured written questionnaire.

Clinical Examination

Trained dental assistants recorded the data on prepared record sheets. All permanent fully erupted teeth, excluding third molars, were examined with a manual periodontal probe^{||} color coded at 1, 2, 3, 5, 7, 8, 9, and 10 mm. Six sites per tooth were assessed in the mesio-buccal, mid-buccal, disto-buccal, disto-lingual, mid-lingual, and mesio-lingual sites.

Probing depth was defined as the distance from the free gingival margin to the bottom of the pocket/sulcus. Gingival recession was defined as the distance from the cemento-enamel junction (CEJ) to the free gingival margin and was assigned a negative sign if the gingival margin was located coronal to the CEJ. Clinical attachment loss (CAL) was defined as the distance from the CEJ to the bottom of the pocket/sulcus, and was calculated as the sum of the probing depth and gingival recession measurements. Measurements were made in millimeters, rounded to the lower whole millimeter.

Sample Size

Considering the lack of information about the prevalence of periodontal disease in the target population, we assumed the "worst case scenario" (i.e., 50% prevalence) in calculating the sample size, and used a $\pm 3\%$ precision level for the 95% confidence interval for the reporting of results. We estimated that this design would yield approximately 30% inefficiency. The sample size calculation used standard formulas for simple random sampling²¹ and adjusted for the design effect.²² It was estimated

that the required sample size was approximately 1,400 subjects.

Ethical Considerations

The study protocol was reviewed and approved by the following committees: Research Ethics Committee, Federal University of Rio Grande do Sul, Porto Alegre, Brazil; the National Commission on Ethics in Research, Ministry of Health, Brazilia, Brazil; and Ethics in Medical Research Committee, University of Bergen, Bergen, Norway. Subjects who agreed to participate signed an informed consent form. At the conclusion of the study the participants were provided with a written report detailing their oral status and any diagnosed mucosal lesions. Patients with diagnosed pathological conditions were advised to seek specialist consultation and treatment.

Non-Response Analysis

A total of 2,435 subjects were eligible for examination in this survey. Of these, 1,586 (65.1%) were clinically examined, including 974 subjects in the age group 30+ years, and 612 subjects <30 years old. Among those not examined, 127 (5.2%) refused to participate, 26 (1.1%) were unable to attend the examination site because of a physical disability, 60 (2.5%) were interviewed but refused to be examined, and 636 (26.1%) were not at home. Subsequent to the examinations, a random sample of 339 (39.9%) subjects was selected from 849 eligible subjects who either refused to participate or were not available during the normal survey schedule. Attempts were made to contact the selected subjects by telephone to collect data for the non-response analysis. Fifty (14.7%) subjects and their households were not available on two telephone call attempts and 18 (5.3%) refused to be interviewed.

Data were obtained for the 271 (79.9%) subjects, and these are referred to in the text as the non-respondents. Of these, 127 subjects agreed to the telephone interview. The other 144 were not available after two telephone call attempts and the non-response data were obtained through a first-degree relative living in the same household. The information collected included the gender, age, education, dental care visits, and income level. Information on the number of teeth present was collected for the 127 subjects who participated in a telephone interview.

The non-respondents were similar to the study group in the mean age, but included a somewhat higher percentages of males and whites and had more years of education.

Measurement Reproducibility

At two time points, before and 3 months after the start of the study, the examiners were trained and calibrated in performing the clinical measurements. The examination team followed a quality control protocol aimed

^{||} PCP10-SE, Hu-Friedy Inc., Chicago, IL.

at reducing systematic and random measurement errors and to quantify what error remained. The protocol involved standard examination environment and methodology, standard equipment, and detailed written instructions for clinical procedures.

Assessment of measurement reproducibility used replicate periodontal measurements performed during the fieldwork. The examiner with the most clinical experience served as the gold standard examiner. A total of 57 subjects, divided into four groups ranging from eight to 20 subjects, were used for the reproducibility assessment. In one of the groups, the replicate measurements consisted of repeated measurements by the gold standard examiner. In the remaining three groups, the replicate measurements were made by one examiner and the gold standard examiner. Measurement reproducibility at the subject level was assessed by the intraclass correlation coefficient²³ and weighted kappa, and at the site level by the weighted Kappa.²⁴ The intraclass correlation coefficient for mean CAL ranged between 0.95 and 0.99 and for extent scores of CAL ≥ 5 mm and ≥ 7 mm ranged between 0.80 and 0.98. The weighted kappa (± 1 mm) at subject level prevalence (maximum attachment loss) were between 0.69 and 1.00. The weighted kappa (± 1 mm) at site level ranged between 0.65 and 0.87.

Data Analysis

Prevalence was defined as the percentage of individuals having at least one tooth with the condition, and extent was defined as the percentage of teeth per person having at least one site with the condition.

A weight variable was used in the data analysis to minimize the bias in the population parameter estimation²² which may arise due to the sample non-response. The calculation of the weight variable was based on census information provided by IBGE.¹⁷

The race variable was scored as white or non-white. Only two categories of race were used because reliable criteria to classify Brazilian subjects into blacks and mulattos were not available, and this population included only a small percentage of other ethnic groups.

Socioeconomic status was scored by combining information about family income using a standard Brazilian classification (CCEB), and each individual's level of education. High socioeconomic status was defined as having ≥ 9 years of education and being in the upper two tertiles of the CCEB classification, or having 5 to 8 years of education and being in the highest tertile of the CCEB classification. Low socio-economic status was defined as having 1 to 4 years of education and being in the lowest two tertiles of the CCEB classification, or having 5 to 8 years of education and being in the lowest tertile of the CCEB classification. Individuals who had higher economic status and education than the low socioeconomic status group, but

less than the high group were classified as having medium socioeconomic status.

Exposure to cigarette smoking was calculated for current and former smokers. Number of cigarettes consumed per day was multiplied by the number of years of habit, and divided by 20 (one pack) to calculate the total number of packs of cigarettes consumed in a lifetime. The subjects were classified into four groups: non-smokers, light (one to 2,734 packs), moderate (2,735 to 7,300 packs), and heavy smokers ($\geq 7,300$ packs). The smoking cut-points were selected according to tertiles among current and former smokers.

The study subjects were classified according to the self-reported frequency and reasons for dental visits during the last 5 years. Individuals who had visited a dentist on a regular basis for maintenance care were classified as having regular dental visits. Subjects who had visited a dentist only for emergency dental treatment, or had not visited a dentist during the last 5 years were classified as having irregular dental visits.

Data analysis was performed using a statistical software program[¶] and survey commands that take into account survey design including stratification, clustering, and weighting and robust variance estimation. A weight variable was used to adjust for the probability of selection and deviations in the sample distributions from the target population distribution by age, gender, and education.^{17,22} Pair-wise comparisons of crude estimates were carried out using the Wald test.²² The chosen level of statistical significance was 5%, and the 95% confidence intervals (CI) were calculated.

Survey multinomial logistic regression was used to model the relationship between different degrees of attachment loss and potential risk indicators. The dependent variable was occurrence of moderate or severe attachment loss. Moderate and severe attachment loss were defined as subjects with CAL ≥ 5 mm in 15% to 50% of the teeth or in $>50\%$ of the teeth, respectively. Subjects with levels of attachment loss below the moderate attachment loss category were defined as having slight or no attachment loss.

The data were analyzed by univariable and multivariable models using logistic regression analyses. In both models, the multinomial logistic regression method²⁵ was used to compare the moderate and severe attachment loss groups, separately, with the slight/no attachment loss group. Preliminary analysis was performed using a univariable model. Only exposures showing in the univariable analyses associations with $P \leq 0.25$ were included in the multivariable model.²⁵ The contribution of each variable to the model was assessed by means of the Wald statistic. Confounding and interactions were evaluated. In the multinomial logistic model the estimate of risk in the population is reported as relative risk ratio (RRR) which

¶ Stata version 7.0 for Windows, Stata Corporation, College Station, TX.

is equivalent to the odds ratio in the binary logistic regression analysis. No significant interactions were found among the independent variables. Eight hundred forty-three (843) subjects were included in the multivariable analysis.

RESULTS

Approximately 97%, 79%, and 52% of the subjects and 68%, 36%, and 16% of the teeth per subject had CAL

≥3 mm, ≥5 mm, and ≥7 mm, respectively (Table 2). Both the percentage of subjects, and the percentage of teeth per subject with CAL increased with increasing age regardless of the CAL threshold assessed.

Males had a significantly higher prevalence of CAL and frequency of teeth with CAL than females, particularly in comparisons that involved the more pronounced thresholds of attachment loss (Table 3). Non-whites had a significantly (*P* <0.001) higher prevalence of CAL

≥5 mm than whites. However, the two races showed comparable percentages of subjects with slight (≥3 mm) and severe (≥7 mm) CAL, and similar percentages of teeth with various thresholds of CAL. Maxillary molars were the teeth most frequently affected with CAL; 77% of maxillary first molars in persons aged 50 years or older had CAL ≥5 mm (Fig. 1).

A consistent pattern of relationship was observed between attachment loss and socioeconomic status (Fig. 2). In the low socioeconomic status group, 83% of the subjects and 41% of the teeth had CAL ≥5 mm, whereas the corresponding figures in the high socioeconomic status group were 74% subjects and 28% teeth. A similar trend was also seen for the higher thresholds of CAL.

Cigarette smoking status had a significant relationship with the prevalence and extent of attachment loss (Fig. 3). Moderate and heavy smokers had markedly increased prevalence and higher percentages of teeth with CAL compared to light and non-smokers, particularly in the high thresholds attachment loss.

Individuals who reported having diabetes mellitus had a significantly higher percentage of teeth with CAL ≥3 mm (77.8% versus 67.3%, *P* <0.05) and ≥5 mm (49.6% versus 34.8%, *P* <0.05) than those self-reported as non-diabetic, although no other statistically significant differences were observed. The group that reported regular dental visits had significantly lower

Table 2.

Percentage of Subjects (prevalence) and Percentage of Teeth Per Subject (extent) by Attachment Loss and Age

Attachment Loss	Age (years)											
	30-39		40-49		50-59		60-69		≥70		Total*	
	%	SE	%	SE	%	SE	%	SE	%	SE	%	SE
Prevalence												
≥3 mm	93.9	1.7	100.0	0.0	99.4	0.6	100.0	0.0	100.0	0.0	97.4	0.6
≥4 mm	85.3	1.6	97.1	1.2	98.3	0.8	100.0	0.0	100.0	0.0	92.9	0.8
≥5 mm	64.3	2.4	84.6	2.2	94.4	2.3	92.3	2.4	98.0	2.2	79.2	1.5
≥6 mm	46.0	2.5	72.1	3.1	79.6	4.0	80.0	3.1	93.7	2.6	64.4	1.8
≥7 mm	32.0	1.6	62.1	3.3	65.3	4.5	70.7	3.2	80.6	7.8	51.9	1.2
Extent												
≥3 mm	51.1	2.9	74.2	2.4	80.5	2.1	88.8	2.8	93.5	1.7	67.9	1.7
≥4 mm	31.9	1.5	57.4	2.8	66.2	1.9	73.5	2.5	85.1	1.6	51.1	1.1
≥5 mm	18.1	0.9	41.9	3.1	48.3	1.7	55.2	2.9	71.9	2.5	35.7	1.0
≥6 mm	9.4	0.5	28.2	3.1	31.9	1.5	38.2	3.5	52.6	3.4	23.1	0.9
≥7 mm	5.3	0.3	20.5	2.9	22.3	1.2	27.4	2.8	37.5	3.5	15.9	0.8

* Total percentages were calculated using a weight variable based on population census information.

Table 3.

Percentage of Subjects (prevalence) and Percentage of Teeth Per Subject (extent) by Attachment Loss, Gender, and Race

Attachment Loss	Gender					Race				
	Males		Females		<i>P</i>	Whites		Non-Whites		<i>P</i>
	%	SE	%	SE		%	SE	%	SE	
Prevalence										
≥3 mm	98.3	0.8	96.7	1.0	0.23	97.1	0.7	98.8	1.0	0.18
≥4 mm	94.3	1.1	91.6	1.4	0.18	91.8	0.9	97.4	1.5	0.006
≥5 mm	83.6	2.0	75.3	1.2	0.0003	76.8	1.4	89.2	2.3	0.0003
≥6 mm	71.8	2.0	57.7	1.8	0.0001	61.7	1.9	75.3	3.1	0.002
≥7 mm	58.8	1.8	45.8	1.2	0.0001	51.0	1.8	55.9	2.6	0.22
Extent										
≥3 mm	71.1	2.6	65.1	1.4	0.03	67.3	1.9	70.3	2.0	0.21
≥4 mm	55.8	2.3	46.8	0.8	0.007	50.4	1.2	53.6	2.0	0.17
≥5 mm	40.8	1.8	31.1	1.0	0.001	35.2	1.0	37.7	2.0	0.28
≥6 mm	27.8	1.3	18.8	1.1	0.0002	22.3	1.1	26.3	2.1	0.12
≥7 mm	20.0	1.3	12.2	1.0	0.0006	15.4	0.9	17.9	2.1	0.29

prevalence and extent of CAL ≥ 7 mm than the group with irregular dental visits (36% versus 56% subjects, and 8% versus 18% teeth, $P < 0.01$).

In this population, 42.2%, 28.4%, and 30.4% of the subjects had slight/no loss, moderate, and severe attach-

ment loss, respectively. The univariable analysis showed that subjects in the age groups 40 to 49 and >50 years, and those who were moderate or heavy smokers were at a significantly higher risk for having moderate or severe attachment loss than subjects of younger age, and non-smokers (Table 4). Males, diabetics, subjects with irregular dental visits, and those with low or medium socioeconomic level had a significantly higher risk for having severe attachment loss than females, non-diabetics, and subjects with regular dental care and high socioeconomic status. Race was not significantly associated with attachment loss.

The multivariable analysis showed that subjects in the age groups 40 to 49 and >50 years had a significantly higher risk for having moderate (RRR = 3.0 and 5.9) and severe CAL (RRR = 7.4 and 25.4) compared to subjects in the 30 to 39 years group (Table 5). Moderate and heavy smokers had a significantly higher risk for moderate (RRR = 2.1 and 3.0) and severe CAL (RRR = 3.4 and 8.2) compared to non-smokers. There also was a significantly higher risk for severe CAL in males than in females (RRR = 1.6), and in subjects with low (RRR = 1.8) or medium socioeconomic status (RRR = 1.6),

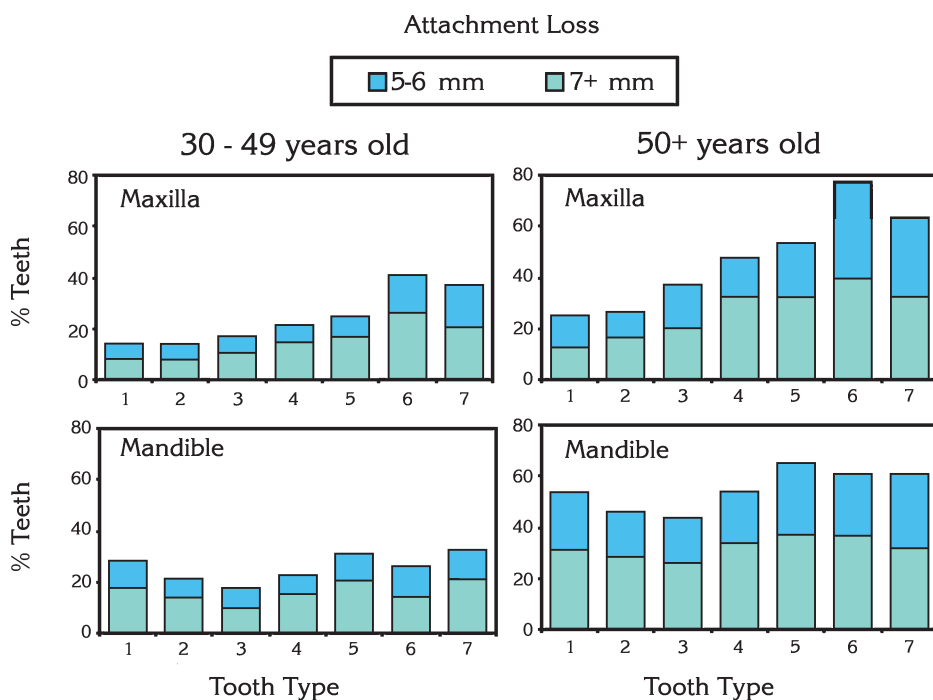


Figure 1.

Percentage of teeth by various thresholds of attachment loss, tooth type, and age group. Central incisor: 1; lateral incisor: 2; canine: 3; first premolar 4; second premolar: 5; first molar: 6; second molar: 7.

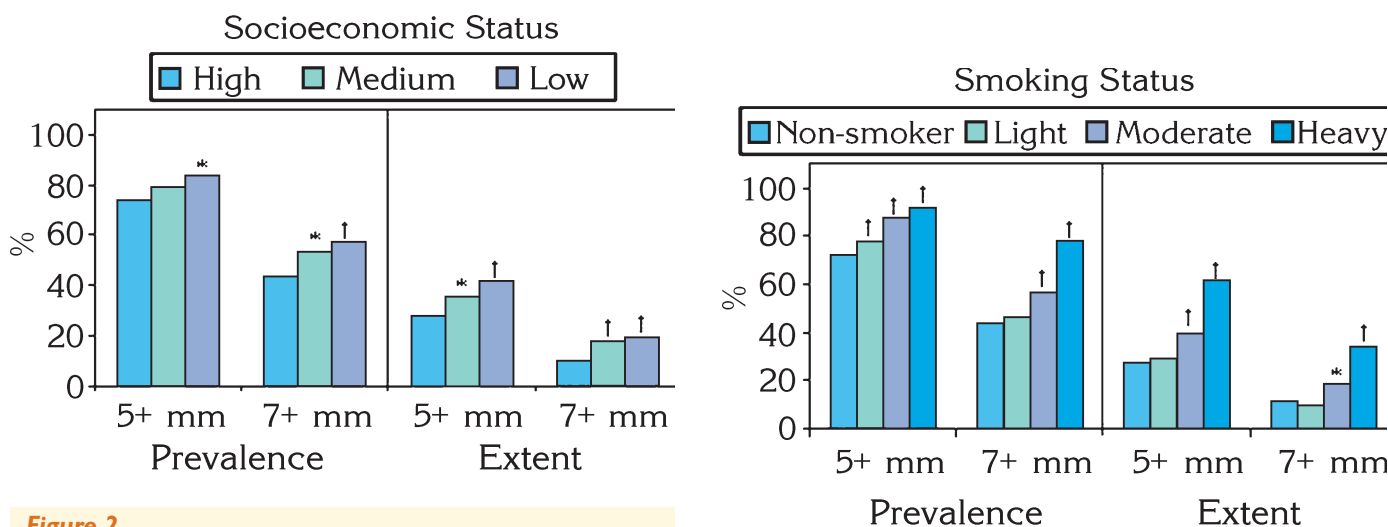


Figure 2.

The percentage of subjects (prevalence) and the percentage of teeth per subject (extent) with attachment loss by socioeconomic status. * $P < 0.05$; † $P < 0.01$, compared to the high socioeconomic status group by means of Wald test.

Figure 3.

Prevalence and extent of attachment loss by smoking status. * $P < 0.05$; † $P < 0.01$, compared to non-smokers by means of Wald test.

Table 4.
Univariable Model of the Effect of Demographic, Behavioral, and Other Variables on the Occurrence of Attachment Loss

Variable	N	Attachment Loss					
		Moderate			Severe		
		RRR	95% CI		RRR	95% CI	
Age							
30-39 years	294	1.0			1.0		
40-49 years	253	2.9 [†]	1.5 5.5		7.4 [†]	4.8 11.2	
50+ years	301	5.0 [†]	3.4 7.4		19.0 [†]	13.9 26.0	
Gender							
Female	385	1.0			1.0		
Male	463	1.2	0.8 1.9		1.9 [†]	1.3 2.8	
Race							
White	681	1.0			1.0		
Non-white	167	1.1	0.7 1.6		1.0	0.7 1.4	
Socioeconomic status							
High	271	1.0			1.0		
Medium	235	1.5	0.9 2.5		1.7*	1.0 2.8	
Low	342	1.4	0.8 2.3		2.2 [†]	1.6 3.1	
Smoking							
Non-smokers	419	1.0			1.0		
Light	147	1.0	0.6 1.5		1.1	0.6 1.9	
Moderate	139	1.7*	1.1 2.8		2.5 [†]	1.6 3.8	
Heavy	143	2.9*	1.3 6.5		9.0 [†]	5.8 14.0	
Dental visits							
Regular	188	1.0			1.0		
Irregular	655	1.3	0.9 1.9		2.9*	1.4 6.2	
Diabetic status							
Non-diabetic	789	1.0			1.0		
Diabetic	59	1.7	0.8 3.5		3.3*	1.1 10.6	

* $P < 0.05$. † $P < 0.01$.

or having irregular dental visits (RRR = 2.1). Diabetic status and race did not show significant associations with CAL in this multivariable model.

DISCUSSION

The results show that attachment loss is prevalent in the adult urban population in the Brazilian state of Rio Grande do Sul. Approximately three of four subjects aged >30 years had at least one tooth with CAL ≥5 mm, and more than half of the subjects had one or more teeth with CAL ≥7 mm.

A recent systematic review¹⁶ found only a few published reports on the periodontal status of the Brazilian adult population. A national survey conducted in 1986

Table 5.
Multivariable Model of the Effect of Demographic, Behavioral, and Other Variables on the Occurrence of Attachment Loss

Variable	Attachment Loss					
	Moderate			Severe		
	RRR	95% CI		RRR	95% CI	
Age						
30-39 years	1.0			1.0		
40-49 years	3.0 [†]	1.6 5.8		7.4 [†]	5.0 11.1	
50+ years	5.9 [†]	4.0 8.6		25.4 [†]	19.0 33.7	
Gender						
Female	1.0			1.0		
Male	1.2	0.8 1.8		1.6*	1.0 2.5	
Socioeconomic status						
High	1.0			1.0		
Medium	1.5	1.0 2.4		1.6 [†]	1.3 2.1	
Low	1.3	0.8 2.2		1.8 [†]	1.3 2.6	
Smoking						
Non-smoker	1.0			1.0		
Light	1.1	0.7 1.9		1.4	0.6 3.2	
Moderate	2.1 [†]	1.4 3.2		3.4 [†]	2.6 4.4	
Heavy	3.0 [†]	1.6 5.8		8.2 [†]	5.5 12.2	
Dental visits						
Regular	1.0			1.0		
Irregular	1.1	0.8 1.7		2.1*	1.1 4.0	

* $P < 0.05$; † $P < 0.01$.

in Brazil²⁶ used the Community Periodontal Index of Treatment Needs (CPITN) methodology to assess the periodontal status and estimated that 5.2% and 7.4% of the subjects in the age groups 35 to 44 and 50 to 59 years had one or more teeth with probing depth of ≥5.5 mm (CPITN 4). Other surveys of Brazilian sub-populations^{27,28} have also used the CPITN method. However, comparison between these surveys and the present study is not feasible since the CPITN method is based on a partial recording system and it does not measure attachment loss.

Attachment loss among U.S. adults was assessed in the Third National Health and Nutrition Examination Survey (NHANES III) during 1988-1994 and reported that 19.9% and 7.3% of American adults aged 30 to 90 years had CAL ≥5 mm and ≥7 mm, respectively.^{2,3} In this study we estimated that the corresponding prevalence rates in our Brazilian population aged 30+ years were 79.2% and 51.9% of subjects, which are 4- and 7-fold higher than for the U.S. population, respectively. Similarly, the mean percentage of teeth

per subject with attachment loss was considerably higher in this population than in the U.S. adult population: 35.7% versus 5.9% had CAL ≥ 5 mm, and 15.9% versus 1.8% had CAL ≥ 7 mm. It should be noted, however, that the NHANES III survey used a partial recording system consisting of a half-mouth examination and examined two sites per tooth. Partial recordings underestimate periodontal disease status, and the amount of underestimation varies depending on the type of system.²⁹ For this reason, and without the use of an accurate inflation factor, direct comparison of the present findings with those of the NHANES III survey may not be rational.

A national survey of United Kingdom adults was conducted in 1998¹ and reported that 42% of 35 to 44 years olds and 70% of 55 to 64 years olds had CAL >3.5 mm. These prevalence estimates were significantly lower than the corresponding rates in the present study which were 92.2% and 99.2% subjects, respectively. The U.K. survey used the CPI method to assess attachment loss, and as such it has some of the inherent limitations of a partial recording system.

In a survey among adults in Southern China, Corbet et al.⁶ used the CPI index and estimated that 56% to 61% of 35 to 44 year olds had CAL ≥ 4 mm and 48% and 55% of 65 to 74 year olds in urban and rural areas, respectively, had CAL ≥ 6 mm. The corresponding rates in our population were 92.2% of 35 to 44 year olds with CAL ≥ 4 mm and 88.3% of 65 to 74 year olds with CAL ≥ 6 mm, which are higher than those reported for the population in Southern China. On the other hand, Baelum et al.⁴ studied Kenyan adults and estimated that 90% of subjects 35 years and older had CAL ≥ 4 mm, which is similar to the prevalence in our population. Furthermore, they reported that 35% of 35 to 44 year olds, 55% of 45 to 54 year olds, and 80% of 55 to 64 year olds had CAL ≥ 7 mm. The corresponding rates in the present study were 44.4%, 66.1%, and 66.7%, respectively. Comparable prevalence and extent of attachment loss to our population also have been reported in a recent study in a rural population in Southern Thailand.⁵

Tooth loss was highly prevalent in the present population, and edentulism steadily increased after 40 years of age. Since tooth loss decreases the number of teeth available for periodontal assessment, it is possible that it would affect the estimates of CAL especially in the older age group.^{2,30}

The present results showed that age was an important predictor of the occurrence of moderate and severe attachment loss. The percentage of teeth with CAL ≥ 5 mm increased 4-fold, from 18% to 72%, and the prevalence increased 1.5 times, from 64% to 98%, between the age groups 30 to 39 and 70+ years. Univariable and multivariable analytical models confirmed that age is an important predictor of the severity of attachment loss even after adjusting for other pertinent

variables. This corroborates findings of other studies showing a significant association of age with periodontal disease severity.^{3,31,32}

Our findings also are consistent with previous studies showing a significant effect of cigarette smoking on the occurrence of periodontal diseases.^{6,14,31,33-35} Using a univariable model we found that heavy smokers had 2.9 and 9 times higher chance of having moderate and severe attachment loss, whereas moderate smoking showed moderate effects (RRR = 1.7 and 2.5, respectively). Notably, the effects of smoking on attachment loss were not significantly reduced after adjusting for other risk indicators and heavy smoking was still a significant risk factor for moderate and severe CAL (RRR = 3.0 and 8.2), and the same was true for moderate smoking (RRR = 2.1 and 3.4). Light smoking was not associated with a significantly increased risk for CAL.

In this population, males showed a higher prevalence and more teeth with CAL and had a 60% higher risk for severe CAL than females. This is similar to findings of other studies in developed^{1,2,31,33} and developing countries.⁴⁻⁶ Non-whites had somewhat higher prevalence of CAL than whites in this study. However, race did not show a significant effect on the severity or the percentage of teeth per subject with CAL. Albanadar et al.² adjusted for age and gender and reported a significantly higher prevalence and extent of attachment loss in African-Americans and Mexican-Americans than in whites. However, Hyman and Reid³³ did not observe an association between race and CAL after adjusting for other important risk indicators.

Other factors that showed statistically significant risk effects for attachment loss included low to medium socioeconomic status and irregular pattern of dental visits. This is consistent with studies in South American populations showing an association between low socioeconomic status and poor periodontal health,^{26,36} and with studies of the relationship of dental visits with severe CAL in the U.S.³³ and Chinese⁶ subpopulation. However, a study among U.K. adults did not find a significant association.¹ Because of the particular features of this population, the cut-off points used in the present study for certain predictors may not be directly comparable to other studies.

Epidemiologic data are important and can be very useful for the improvement of public health. The identification of high-risk groups is one potential strategy that has been recommended for disease prevention. Prediction models can be used to identify environmental and other exposures that may have causal or other associations with disease. However, it should be recognized that cross-sectional studies can suggest associations, whereas causality can only be established using prospective study design.¹³

This study described an adult population with a high level of attachment loss and assessed the magnitude

of risk due to different exposures. The results show that aging and a moderate to heavy cigarette smoking habit may significantly increase the risk for moderate and severe attachment loss. Males, subjects with low or moderate socioeconomic status, and those with an irregular pattern of dental visits are also at a significantly higher risk for attachment loss than the rest of the population. Using a combination of these factors may allow the definition of high-risk groups, which may be targeted for public health intervention.

ACKNOWLEDGMENT

Funding for this project was provided by the Foundation for Post-Graduate Education (CAPES), Ministry of Education, Brazilia, DF, Brazil (grant number 1614/99-1).

REFERENCES

- Morris AJ, Steele J, White DA. The oral cleanliness and periodontal health of UK adults in 1998. *Br Dent J* 2001; 191:186-192.
- Albandar JM, Brunelle JA, Kingman A. Destructive periodontal disease in adults 30 years of age and older in the United States, 1988-1994. *J Periodontol* 1999;70:13-29.
- Albandar JM. Periodontal diseases in North America. *Periodontol 2000* 2002;29:31-69.
- Baelum V, Fejerskov O, Manji F. Periodontal diseases in adult Kenyans. *J Clin Periodontol* 1988;15:445-452.
- Baelum V, Pisuihanakan S, Teanpaisan R, et al. Periodontal conditions among adults in Southern Thailand. *J Periodontol Res* 2003;38:156-163.
- Corbet E, Wong M, Lin H. Periodontal conditions in adult Southern Chinese. *J Dent Res* 2001;80:1480-1485.
- Papapanou PN. Epidemiology of periodontal diseases: An update. *J Int Acad Periodontol* 1999;1:110-116.
- Slade GD, Ghezzi EM, Heiss G, Beck JD, Riche E, Offenbacher S. Relationship between periodontal disease and C-reactive protein among adults in the Atherosclerosis Risk in Communities study. *Arch Intern Med* 2003; 163:1172-1179.
- Lopez NJ, Smith PC, Gutierrez J. Periodontal therapy may reduce the risk of preterm low birth weight in women with periodontal disease: A randomized controlled trial. *J Periodontol* 2002;73:911-924.
- Scannapieco FA, Ho AW. Potential associations between chronic respiratory disease and periodontal disease: Analysis of National Health and Nutrition Examination Survey III. *J Periodontol* 2001;72:50-56.
- Grossi SG, Skrepcinski FB, DeCaro T, et al. Treatment of periodontal disease in diabetics reduces glycated hemoglobin. *J Periodontol* 1997;68:713-719.
- Albandar JM, Rams TE. Global epidemiology of periodontal diseases: An overview. *Periodontol 2000* 2002;29:7-10.
- Albandar JM. Global risk factors and risk indicators for periodontal diseases. *Periodontol 2000* 2002;29:177-206.
- Gelskey SC. Cigarette smoking and periodontitis: Methodology to assess the strength of evidence in support of a causal association. *Community Dent Oral Epidemiol* 1999;27:16-24.
- Taylor GW. Bidirectional interrelationships between diabetes and periodontal diseases: An epidemiologic perspective. *Ann Periodontol* 2001;6:99-112.
- Gjerme P, Rosing CK, Susin C, Oppermann R. Periodontal diseases in Central and South America. *Periodontol 2000* 2002;29:70-78.
- Brazilian Institute of Geography and Statistics. Population Census (in Portuguese). Rio de Janeiro, Brazil; 1996.
- Brazilian Institute of Geography and Statistics. Demographic Census 2000: Characteristics of the Population and Households – General Results (in Portuguese). Rio de Janeiro, Brazil; 2001.
- Brazilian Institute of Geography and Statistics. Demographic Census 1991 (in Portuguese). Rio de Janeiro, Brazil; 1999.
- Rio Grande do Sul State Agency for Metropolitan Affairs. Environmental Cartographic Data of the Metropolitan Area of Porto Alegre (in Portuguese). Porto Alegre, Brazil; 1997.
- Levy P, Lemeshow S. *Sampling of Populations. Methods and Applications*, 3rd ed. New York: John Wiley & Sons, Inc; 1999:70-79.
- Korn E, Graubard B. *Analysis of Health Surveys*. New York: John Wiley & Sons, Inc; 1999.
- Shrout P, Fleiss J. Intraclass correlations: Uses in assessing rater reliability. *Psychol Bull* 1979;86:420-428.
- Hubert L. Kappa revisited. *Psychol Bull* 1977;84:289-297.
- Hosmer D, Lemeshow S. *Applied Logistic Regression*, 2nd ed. New York: John Wiley & Sons; 2000:260-287.
- Ministério da Saúde. Levantamento epidemiológico em saúde bucal: Brasil, zona urbana, 1986 (in Portuguese): Ministério da Saúde; 1988.
- Dini EL, Guimaraes LOC. Periodontal conditions and treatment needs (CPITN) in a worker population in Araraquara, SP. *Brazil Dent J* 1994;44:309-311.
- Flores-de-Jacoby L, Bruchmann S, Mengel R, Zafropoulos GG. Periodontal conditions in Rio de Janeiro City (Brazil) using the CPITN. *Community Dent Oral Epidemiol* 1991;19:127-128.
- Kingman A, Albandar JM. Methodological aspects of epidemiological studies of periodontal diseases. *Periodontol 2000* 2002;29:11-30.
- Schuller AA, Thomsen IO, Holst D. Adjusting estimates of alveolar bone loss for missing observations: Developing and testing a general model. *J Dent Res* 1999;78:661-666.
- Grossi SG, Zambon JJ, Ho AW, et al. Assessment of risk for periodontal disease. I. Risk indicators for attachment loss. *J Periodontol* 1994;65:260-267.
- Burt BA. Periodontitis and aging: Reviewing recent evidence. *J Am Dent Assoc* 1994;125:273-279.
- Hyman JJ, Reid BC. Epidemiologic risk factors for periodontal attachment loss among adults in the United States. *J Clin Periodontol* 2003;30:230-237.
- Albandar JM, Streckfus CF, Adesanya MR, Winn DM. Cigar, pipe, and cigarette smoking as risk factors for periodontal disease and tooth loss. *J Periodontol* 2000; 71:1874-1881.
- Bergstrom J, Eliasson S, Dock J. A 10-year prospective study of tobacco smoking and periodontal health. *J Periodontol* 2000;71:1338-1347.
- 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;48:96-103.

Correspondence: Dr. Jasim M. Albandar, Department of Periodontology, Temple University School of Dentistry, 3223 N. Broad St., Philadelphia, PA 19140. Fax: 215/707-7616; e-mail: jasim.albandar@temple.edu.

Accepted for publication November 25, 2003.

Paper II

Periodontal attachment loss attributable to cigarette smoking in an urban Brazilian population

Cristiano Susin^{1,2,3},
Rui V. Oppermann^{3,4},
Ola Haugejorden² and
Jasim M. Albandar¹

¹Department of Periodontology, Temple University School of Dentistry, Philadelphia, PA, USA; ²Department of Odontology, University of Bergen, Bergen, Norway; ³Department of Periodontology, Lutheran University of Brazil, Canoas, Brazil; ⁴Department of Periodontology, Federal University of Rio Grande do Sul, Porto Alegre, Brazil

Susin C, Oppermann RV, Haugejorden O, Albandar JM: Periodontal attachment loss attributable to cigarette smoking in an urban Brazilian population. *J Clin Periodontol* 2004; doi: 10.1111/j.1600-051X.2004.00588.x. © Blackwell Munksgaard, 2004.

Abstract

Aims: The present study estimated the percentages of cases with severe periodontal attachment loss (PAL) attributable to cigarette smoking in a representative adult urban population in southern Brazil.

Methods: A representative sample comprising 853 dentate individuals (age: 30–103 years) was selected by a multistage, probability sampling method. A full-mouth clinical examination of six sites per tooth was performed and an interview using a structured written questionnaire was undertaken. Cases were defined as individuals with $\geq 30\%$ teeth with PAL ≥ 5 mm. A multivariate logistic regression analysis for complex surveys was performed, and adjusted for age, gender, race, socioeconomic status and dental calculus.

Results: The prevalence of cases in this population was 49.7%, or 739,000 subjects. Overall, 50.9% of this adult population, or approximately 757,000 subjects have had a lifetime exposure to cigarette smoking. Multivariate analysis showed that heavy and moderate smokers had a significantly higher risk for PAL ≥ 5 mm than non-smokers (odds ratio = 3.6, 2.0, respectively) after adjusting for the above covariates. We estimated that the number of moderate and heavy smokers with $\geq 30\%$ teeth with PAL ≥ 5 mm might be reduced by approximately 28% and 48%, respectively, had they not smoked cigarettes. We project that a smoking cessation program could result in a reduction in the number of cases by up to 12% in this population, or approximately 90,000 potential cases.

Conclusion: Cigarette smoking was strongly associated with severe attachment loss in this population. A significant percentage of cases may have been prevented if smoking cessation interventions had been implemented. The results support the implementation of population-based smoking cessation programs to reduce the prevalence of severe attachment loss in populations with high level of smoking exposure.

Key words: attributable fraction; attributable risk; periodontal attachment loss; periodontal disease/epidemiology; risk factors; smoking

Accepted for publication 2 February 2004

Smoking has been recognized as one of the major risk factors for a number of diseases in humans, and implicated in a substantial proportion of the global burden of these diseases (Ezzati et al. 2002). Smoking has also been associated with periodontal disease pathogenesis (Gelskey 1999, Albandar 2002) and a significant increase in risk for periodontitis (Albandar et al. 2000, Tomar & Asma 2000, Hyman & Reid 2003).

Despite the wide popularity of using estimates of relative risk and odds ratio (OR) as the basis for assessing the association between smoking and periodontal diseases, neither of these two methods take into consideration the prevalence of the exposures in the population (Walter 1976, Benichou 2001). In addition, the OR may not be a good estimate of relative risk when disease outcome is high. An exposure that highly increases the person's risk

for a certain disease or condition may be of limited public health importance if only a small percentage of cases are attributed to this exposure. Hence, a better appreciation of the population impact of a given exposure should also incorporate an inference of the number of cases that may be attributed to the exposure (Walter 1976, Ezzati et al. 2002).

The concept of population attributable fraction (PAF) was introduced in the

1950s to estimate how much of the disease burden could be attributed to a given risk factor, or may be prevented by its elimination or reduction. Various terms have been used to refer to the frequency of cases attributable to a given exposure, including the terms attributable risk, etiologic fraction and excess fraction (Kleinbaum et al. 1982, Rockhill et al. 1998a). Some of these terms have been criticized for their implicit causality, and it has therefore been suggested that the term attributable fraction may be preferable (Rockhill et al. 1998a).

Few studies have attempted to estimate the fraction of destructive periodontal diseases that could be attributed to smoking in different populations. Analysis of the NHANES III data suggests that a significant proportion of attachment loss in the American population may be attributed to cigarette smoking (Tomar & Asma 2000, Hyman & Reid 2003). However, little data are available from other populations. The aim of the present study was to estimate the number and percentage of cases with severe attachment loss attributable to cigarette smoking in a representative adult urban population in southern Brazil.

Material and Methods

Population

The target population of the present study was adults aged 30 years and older living in the metropolitan area of Porto Alegre in the Brazilian state of Rio Grande do Sul. This state is located in the southern part of Brazil, neighboring Argentina and Uruguay. The present survey covered 14 major municipalities from the Porto Alegre metropolitan area with about 3 million inhabitants.

Study design

The study sample included 974 individuals with an age range of 30–103 years, and comprised 388 (45.5%) males and 465 (54.5%) females, 686 (80.4%) whites and 167 (19.6%) non-whites. Table 1 shows the distribution of subjects by demographic and other important variables in the sample and the target population. The study group comprised 853 dentate and 121 edentulous subjects. Data for five subjects were not complete for some variables, and were excluded from the analysis.

Table 1. Sociodemographic and other characteristics of dentate individuals in the study population

Variables	Dentate sample, <i>N</i>	Dentate population		Individuals with PAL ≥ 5 mm in $\geq 30\%$ of the teeth	
		%	<i>N</i> (thousands)	%	SE
Age (years)					
30–39	294	40.4	601	22.3	1.7
40–49	253	30.4	451	57.5	3.6
50–59	175	16.6	247	65.4	3.0
60–69	84	8.6	127	73.0	4.1
70+	42	4.1	60	91.7	3.7
Gender					
male	385	47.4	705	54.9	3.7
female	463	52.6	782	40.5	1.4
Race					
White	681	80.3	1,195	46.2	1.4
non-White	167	19.7	292	51.7	4.0
Socioeconomic status					
low	342	43.0	639	54.4	2.5
medium	235	26.5	394	46.1	4.4
high	271	30.5	453	38.4	2.0
Smoking					
non-smokers	419	49.1	731	37.6	1.5
light	147	17.9	266	39.6	3.8
moderate	139	17.1	254	55.5	3.7
heavy	143	15.9	237	76.1	3.8
Supragingival calculus (%)					
<25	347	41.6	619	23.8	2.5
25–50	254	30.0	446	49.1	2.2
>50	247	28.4	423	79.9	1.9

Total dentate adults population = 1,487,025 subjects.
PAL, periodontal attachment loss; SE, standard error.

The study sample was drawn from a larger sample representative of subjects aged 14 years and older among the population of Porto Alegre. A representative, multistage, probability sample was derived based on information provided by Rio Grande do Sul state government agency for metropolitan affairs (METROPLAN) and the Brazilian Institute of Geography and Statistics (IBGE). Using area maps, the Porto Alegre metropolitan area was divided into 90 geographic areas 10 km² each. Using the 1991 census data (IBGE 1991) and other relevant municipal information (METROPLAN 1997) these geographic areas were stratified into 13 (14.4%) high-income, and 77 (85.6%) low-income status areas. Low-income geographic areas were defined as areas in which more than 40% of the head of the households had a monthly income ≤ 2 standard Brazilian salaries (about US\$ 180), and high-income areas were those with a higher level of income. Within each of these two income strata, primary sampling units (PSUs) were selected randomly with a

probability proportional to size and using a sampling frame of these PSUs. A total of 11 geographic areas were selected, and included two (18.2%) areas with high, and nine (81.8%) areas with low-income status.

The second stage consisted of selecting area sectors within each geographic area. The area sectors have been defined by IBGE as map areas comprising approximately 300 households each. The sectors were selected randomly within each geographic area, and the number of sectors selected was proportional to the number of sectors in each area. Thirty (3.5%) sectors were selected, out of a total of 846 eligible sectors. Approvals for conducting the study were sought separately in each sector from key community, religious and/or administrative leaders. Permission and/or support were granted to access 29 of these sectors, whereas permission to access one sector was denied.

The third stage included selecting households within each of the 29 sectors. It was estimated that approxi-

mately 25 households were needed per sector to provide a sufficient number of subjects in the sample. In each sector, a starting point for the selection of households was established on area maps and was provided independently by the IBGE. Households were sampled consecutively beginning with the next block after the starting point and until the preset number of households was reached.

Consenting household members who were 14 years of age or older were examined, and subjects 30 years or older were included in this study. Exclusion criteria were presence of diseases/conditions that may pose health risks to the participant or examiner, or that may interfere with the clinical examination. Hence, subjects were excluded if they were diagnosed with psychiatric problems, or intoxicated with alcohol or drugs. Individuals requiring a prophylactic regimen of antibiotics were provided with the appropriate medicine before the clinical examination.

Interviews and clinical examination

Three interviewers performed the interviews using a structured written questionnaire. The interviewers were trained before the study, and used standardized procedures to increase consistency. The clinical examinations were performed in a mobile examination unit consisting of a trailer equipped with a complete dental unit, comprising a dental chair, light, compressor and other basic amenities. The examination unit was moved from one examination location to the next according to the survey schedule. Four dentists and two dental assistants completed the fieldwork between June and December 2001. Letters were sent to households and explained the aims of the study and solicited participation. A few days later, one dentist visited the households and provided more information about the study and encouraged participation. Eligible subjects who consented to participation were interviewed to gather demographic, socioeconomic, oral health and other health-related data.

Trained dental assistants recorded the data on prepared record sheets. All permanent fully erupted teeth, excluding third molars, were examined with a manual periodontal probe (PCP10-SE, Hu-Friedy Mfg. Co. Inc., Chicago, IL, USA) color coded at 1, 2, 3, 5, 7, 8, 9,

10 mm. Six sites per tooth were assessed in the mesiobuccal, midbuccal, distobuccal, distolingual, midlingual and mesiolingual sites.

Probing depth was defined as the distance from the free gingival margin to the bottom of the pocket/sulcus. Gingival recession was defined as the distance from the cemento-enamel junction (CEJ) to the free gingival margin, and this assessment was assigned a negative sign if the gingival margin was located coronal to the CEJ. Periodontal attachment loss (PAL) was defined as the distance from the CEJ to the bottom of the pocket/sulcus, and was calculated as the sum of the probing depth and gingival recession measurements. Measurements were made in millimeters and were rounded to the lower whole millimeter.

Ethical considerations

The study protocol was reviewed and approved by the following committees: Research Ethics Committee, Federal University of Rio Grande do Sul, Porto Alegre, Brazil; the National Commission on Ethics in Research, Ministry of Health, Brasilia, Brazil and Ethics in Medical Research Committee, University of Bergen, Bergen, Norway. Subjects who agreed to participate signed an informed consent form. At the conclusion of the study the participants were provided with a written report detailing their oral status and any diagnosed mucosal lesions. Patients with diagnosed pathological conditions were advised to seek specialist consultation and treatment.

Non-response analysis

In the whole population, including individuals aged 14 years and older, 2435 individuals were eligible for the survey. Of these, 1586 (65.1%) subjects were clinically examined. Among those who did not participate, 127 (5.2%) refused to participate, 26 (1.1%) were unable to attend the examination site because of a physical disability, 60 (2.5%) were interviewed but refused to be examined and 636 (26.1%) were not at home. Subsequent to the completion of the examinations, a random sample of 339 (39.9%) subjects was selected out of 849 eligible subjects who either refused to participate or were not available during the normal survey

schedule. Attempts were made to contact the selected subjects by telephone in order to collect data for the non-response analysis. Of the 339 subjects selected for interview, 50 (14.7%) subjects and their households were not available on two telephone call attempts, and an additional 18 (5.3%) subjects refused to be interviewed.

Non-response data were obtained for 271 (79.9%) subjects, and these will be referred to in the text as the non-respondents. Of these, 127 subjects were present and agreed to the telephone interview. The other 144 subjects were not available on two telephone call attempts, and the non-response data were therefore obtained through a first-degree relative living in the same household. The information collected included the subject's gender, age, education, dental care visits and income level. In addition, information about the number of teeth present was collected for the 127 subjects who were present during the telephone interview.

The mean age of the non-respondents group was 35.2 years, and included 51.3% males and 48.7% females, and 90.8% whites and 9.2% non-whites. In contrast, the mean age of the study group was 38 years, and included 45.3% males and 54.7% females, and 82.5% whites and 17.5% non-whites. By the number of years of education, the non-respondents and respondents groups, respectively, included 7.4% and 22.3% subjects with 4 or fewer years, 22.5% and 40.0% subjects with 5–8 years, and 70.1% and 37.8% subjects with more than 8 years of education. This suggests that the non-respondents were similar to the study group in the mean age, but included somewhat higher percentages of males and whites, and had a higher number of years of education than the study participants. A weight variable was used in the data analysis to minimize the bias in the population parameter estimation (Korn & Graubard 1999), which may arise because of the sample non-response. The calculation of the weight variable was based on census information provided by IBGE (IBGE 1996).

Measurement reproducibility

The examiners were trained and calibrated in performing the clinical measurements before and during the field examinations. The examination team followed a quality control protocol that

was aimed at reducing systematic and random measurement errors and to quantify what error remained. The protocol involved standard examination environment and methodology, standard equipment and detailed written instructions for clinical procedures.

Assessment of measurement reproducibility used replicate periodontal measurements performed during the fieldwork. One examiner with the most clinical experience served as the “gold standard” examiner. A total of 57 subjects, divided into four groups ranging from eight to 20 subjects, were used for the reproducibility assessment. In one of the groups, the replicate measurements consisted of repeated measurements by the gold standard examiner. In each of the remaining three groups, the replicate measurements were made by one examiner and the gold standard examiner. Measurement reproducibility at the subject level was assessed by the intraclass correlation coefficient (Shrout & Fleiss 1979) and weighted κ , and at the site level by the weighted κ (Hubert 1977). The intraclass correlation coefficients for mean PAL ranged between 0.95 and 0.99, and for extent scores of PAL ≥ 5 and ≥ 7 mm ranged between 0.80 and 0.98. The weighted κ 's (± 1 mm) of the prevalence of PAL ≥ 5 mm were between 0.69 and 1.00, and at site level ranged between 0.65 and 0.87. The intraclass correlation coefficients for supragingival calculus ranged between 0.73 and 0.98 at the site level, and between 0.66 and 0.99 at the tooth level.

In order to assess the reliability of the self-reported smoking variable, 79 subjects of the study sample were re-interviewed a second time by the gold standard examiner. The second interview was made 1–4 days after the first. The unweighted κ for smoking (categorized as non-smokers, light, moderate and heavy smokers) was 0.92.

Data analysis

The mean tooth loss in this dentate population was 9.2 teeth. At a given tooth, attachment loss was scored as the maximum of attachment loss measurements at six sites per tooth. The outcome variable was presence of severe attachment loss, defined as subjects with PAL ≥ 5 mm in $\geq 30\%$ of the teeth. Exposure to cigarette smoking was calculated for current and former smokers.

Number of cigarettes consumed per day was multiplied by the number of days of habit, and divided by 20 (one pack) to calculate the total number of packs of cigarettes consumed in a lifetime. Smokers were classified using smoking thresholds selected according to tertiles among current and former smokers into heavy (>7300 packs), moderate (2735–7300 packs), light (1–2734 packs) and non-smokers (<1 pack). The four smoking categories are comparable with a consumption of >20 pack years (or = 1 pack/day for >20 years), 7.5–20 pack years (or ~ 1 pack/day for 7.5–20 years), 0.1–7.4 pack years (or ~ 1 pack/day for 0.1–7.4 years) and <0.1 pack years, respectively. The classification by smoking status did not differentiate between current and former smokers.

The race of the subject was scored as “White” or “non-White”, with blacks, mulattos and other ethnic groups scored as “non-Whites”. Socioeconomic status was scored by combining information about family economy using a standard Brazilian economy classification (CCEB) and the level of education of the individual. High socioeconomic status was defined as having 9 years of education and being in the upper two tertiles of the CCEB economy classification, or having 5–8 years of education and being in the highest tertile of the CCEB classification. Low socioeconomic status was defined as having 1–4 years of education, and being in the lowest two tertiles of the CCEB classification, or having 5–8 years of education and being in the lowest tertile of the CCEB classification. Individuals who had higher economy and education than the low socioeconomic group, but less than the high group were classified as having a medium socioeconomic status. Based on tertiles of the percentage of sites with supragingival dental calculus the subjects were grouped into three groups: $<25\%$, 25–50% and $>50\%$ sites.

Attributable fraction among exposed subjects (AF_{exp}) estimates the absolute excess risk for an outcome variable associated with a given exposure, i.e. the fraction of exposed cases that would not have occurred if exposure had not occurred (Kleinbaum et al. 1982, Rockhill et al. 1998a, Szklo & Nieto 2000). In this study, attributable fraction among smokers estimates the absolute excess risk for severe attachment loss because of smoking, or the fraction of

smokers who would not have severe attachment loss if smoking had not occurred. Percent attributable fraction among exposed ($\%AF_{\text{exp}}$) simply converts the attributable fraction among exposed into the percentage of smokers with severe attachment loss because of smoking. PAF is the proportion of reduction of attachment loss risk that could be achieved by eliminating smoking from the population while other risk factors remain unchanged. Percentage population attributable fraction ($\%PAF$) converts the PAF into percentage of subjects with severe attachment loss which is preventable, in the entire population. The following formulas (Szklo & Nieto 2000) were used:

- $AF_{\text{exp}} = p_1 - p_2$,
- $\%AF_{\text{exp}} = \frac{(p_1 - p_2)}{p_1} \times 100$,
- $PAF = p_0 - p_2$,
- $\%PAF = \frac{(p_0 - p_2)}{p_0} \times 100$,

where p_0 is the probability of having severe attachment loss among all subjects; p_1 is the probability of having severe attachment loss among smokers in each smoking category; p_2 is the probability of having severe attachment loss among non-smokers.

The present analysis took into account the design of the survey, including stratification, clustering and weighting. A logistic model for complex survey was used to predict the probability of the outcome, expected prevalence and number of cases. The estimates were adjusted for age, socioeconomic status, gender, race and presence of supragingival calculus. After the initial model was calculated, the exposure effect, i.e. smoking, was removed from the dataset by resetting the covariate to zero, and the probability of the outcome in the logistic model was predicted again. The resulting estimates are the predicted probability of the outcome if the exposure had been removed. Summing these probabilities gives the expected prevalence and number of cases of disease if the exposure was absent or removed from the population (Greenland & Drescher 1993, Benichou 2001).

Results

Overall, 50.9% of this adult population, or approximately 757,000 individuals have been exposed to cigarette smoking

Table 2. Estimated ORs (crude and adjusted) with 95% CI and the attributable fractions (%AF_{exp} and %PAF) because of smoking on the occurrence of attachment loss[†]

	Crude OR	95% CI	Adjusted OR [†]	95% CI	%AF _{exp}	%PAF
non-smoker	1.0		1.0			
light	1.1	0.7–1.7	1.2	0.7–2.2	0.6	1.2
moderate	2.1**	1.4–3.1	2.0**	1.4–2.9	27.7	4.2
heavy	5.6**	3.5–9.0	3.6**	2.2–6.0	48.1	6.8
overall						12.2

%AF_{exp}, percentage attributable fraction among exposed; %PAF, percentage population attributable fraction; OR, odds ratio; CI, confidence interval.

[†]Defined as periodontal attachment loss ≥ 5 mm in $\geq 30\%$ of the teeth.

[‡]Adjusted for age, gender, race, socioeconomic status, and supragingival calculus.

** $p < 0.01$.

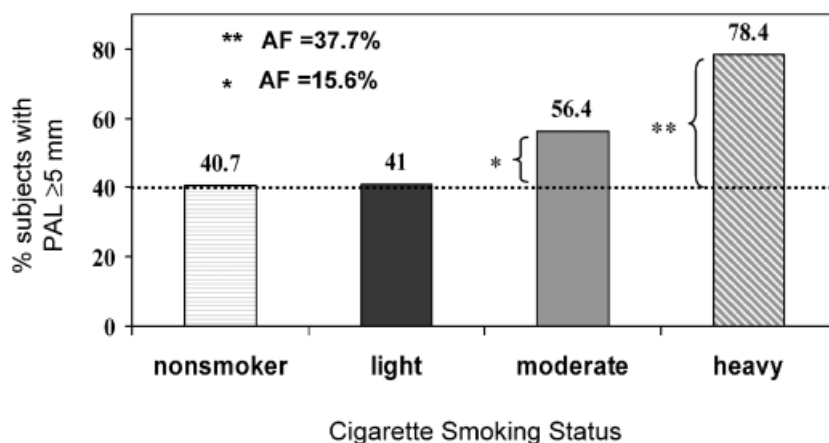


Fig. 1. Percentage of subjects with attachment loss ≥ 5 mm at $\geq 30\%$ of the teeth, adjusted for age, gender, race, socioeconomic status, and dental calculus; and the attributable fraction because of smoking (attributable fraction, AF), by smoking status.

(Table 1), and 49.7%, or 739,000 subjects had PAL ≥ 5 mm at $\geq 30\%$ of the teeth. PAL ≥ 5 mm was significantly more prevalent among heavy ($p = 0.0001$) and moderate ($p = 0.002$) smokers than among non-smokers (Table 1), and was not significantly different in light smokers compared with non-smokers ($p > 0.05$).

Univariate analysis showed that heavy (OR = 5.6) and moderate smokers (OR = 2.1) had higher probability of having severe attachment loss than non-smokers. Multivariate analysis showed that heavy and moderate smokers had higher risk for severe attachment loss than non-smokers (OR = 3.6, 2.0, respectively) after adjusting for age, gender, race, socioeconomic status and dental calculus (Table 2).

The percentage of subjects with severe attachment loss, adjusted for the covariates, was positively correlated with smoking status (Fig. 1). The attributable fraction of attachment loss because of cigarette smoking was

37.7% and 15.6% among heavy and moderate smokers, respectively, and only 0.3% among light smokers. Approximately 28% and 48% of the cases of severe attachment loss could be prevented among moderate and heavy smokers, respectively (Table 2).

In the whole population, 6.1%, or 90,400 individuals had PAL ≥ 5 mm at $\geq 30\%$ of the teeth, attributable to cigarette smoking (Fig. 2). Light smoking contributed only 0.6% to the overall occurrence of PAL ≥ 5 mm in $\geq 30\%$ of the teeth, whereas moderate and heavy smoking, respectively, had 2.1% and 3.3% attributable fraction in the population. We project approximately a 12% decrease in the percentage of subjects having $\geq 30\%$ teeth with PAL ≥ 5 mm if cigarette smoking was completely eliminated in this population, and a larger number of cases may be prevented among heavy smokers than moderate or light smokers (Table 2).

The percentage of subjects with severe attachment loss attributable to

smoking in the population was similar in the 30–39 and 40–49 years old groups, and considerably lower in the 50+ years old group (Table 3). The percentage of PAL attributable to smoking among heavy smokers was considerably higher among individuals 30–39 years old compared with individuals 50+ years old (71.0% versus 27.7%).

Discussion

Half of the subjects in this urban adult Brazilian population have been exposed to cigarette smoking. In addition, half of the population had $\geq 30\%$ of their teeth showing PAL ≥ 5 mm. We estimate that the number of moderate and heavy smokers with $\geq 30\%$ teeth with PAL ≥ 5 mm may be reduced by approximately 28% and 48%, respectively, if these individuals had not been smokers. We also project that a smoking cessation program could result in a reduction in the percentage of cases by up to 12% in this population, or approximately 90,000 potential cases. Clearly, the projected number of preventable cases will depend on the success of the smoking cessation program.

Two recent studies (Tomar & Asma 2000, Hyman & Reid 2003) used data from the NHANES III survey and estimated a higher potential reduction in the percentage of attachment loss cases in the US population than was found in this study. Tomar & Asma (2000) defined cases as subjects with one or more periodontal sites that had a probing depth as well as PAL ≥ 4 mm, and estimated that 41.9% and 10.9% of the cases were attributable to current and former smoking, respectively. Among current smokers, 74.8% of the cases could be attributed to smoking, while among former smokers the percentage of cases was 40.5%. On the other hand, Hyman & Reid (2003) defined cases as the 10% of the population with the greatest mean attachment loss within each age group, and estimated that the smoking attributable fraction for US current smokers was 82% and 84% cases in the 20–49 years and 50+ years old groups, respectively. They also estimated that the attributable fractions for the whole US population (smokers and non-smokers) were 60% and 47% cases in the two respective age groups. The differences in the estimates of PAF between the two studies appear to be related to the definition of cases,

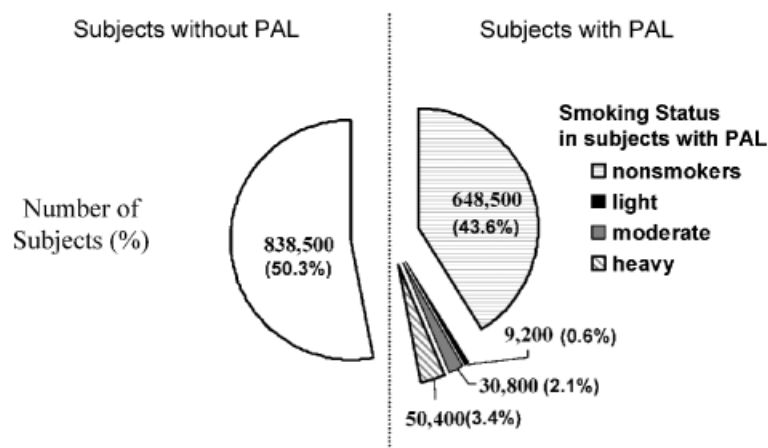


Fig. 2. Population attributable risk (%) because of smoking, and the number of subjects with attachment loss ≥ 5 mm at $\geq 30\%$ of the teeth, by smoking status.

Table 3. Estimated odds ratios (crude and adjusted) with 95% CI and the attributable fractions (%AF_{exp} and %PAF) due to smoking on the occurrence of attachment loss[†], by age group

	Crude OR	95% CI	Adjusted OR [†]	95% CI	%AF _{exp}	%PAF
30–39 years old						
non-smoker	1.0		1.0			
light	1.4	0.4–4.3	1.0	0.3–3.0	21.8	–0.3
moderate	2.4	1.0–6.3	1.5	0.4–5.3	50.2	5.3
heavy	6.1**	2.0–18.7	2.4*	1.1–5.4	71.0	8.3
overall						13.3
40–49 years old						
non-smoker	1.0		1.0			
light	1.5	0.8–2.7	1.3	0.7–2.3	18.5	1.5
moderate	3.5**	2.1–5.8	2.5*	1.1–5.7	41.5	5.8
heavy	6.9**	3.3–14.4	4.2*	1.4–12.6	51.7	8.9
overall						16.2
50+ years old						
non-smoker	1.0		1.0			
light	1.0	0.4–2.3	1.2	0.5–3.2	–2.2	0.8
moderate	2.2	0.6–7.3	2.0	0.7–5.5	18.4	1.8
heavy	4.6**	2.5–8.5	2.5**	1.4–4.5	27.7	2.7
overall						5.3

%AF_{exp}, percentage attributable fraction among exposed; %PAF, percentage population attributable fraction; OR, odds ratio; CI, confidence interval.

[†]Defined as periodontal attachment loss ≥ 5 mm in $\geq 30\%$ of the teeth.

[†]Adjusted for age, gender, race, socioeconomic status, and supragingival calculus.

* $p < 0.05$.

** $p < 0.01$.

as well as the thresholds of smoking exposures used in the two studies. A recent study used a case–control design and adjusted for important risk indicators of periodontal disease, and it estimated that 12% of chronic periodontitis cases could be attributed to smoking (Teng et al. 2003), which is similar to the finding of this study.

For a given exposure, the magnitude of the PAF in the population is directly related to the degree of association between the exposure and the outcome (measured by one of three methods: the

probability of having the disease, relative risk or OR), and the prevalence of exposure (Benichou 2001). The percentage of Brazilians who had smoked in the present population was 50.9%. Similarly, it has been estimated that 51.1% of the American population had been exposed to cigarette smoking (Tomar & Asma 2000). On the other hand, the overall association between smoking and periodontal disease was somewhat weaker in this study population (OR = 1.9) than was reported in the American population (OR = 2.7). The latter differ-

ence may explain part of the disparity in the estimated smoking attributable fraction in the two populations.

In multifactorial diseases the contribution of all plausible and potential risk factors should be investigated, since the estimated value of PAF may be influenced by the study design, including the types of covariates used in the model. The estimated PAF measures the reduction in PAL which could be achieved, given that all other factors remain unchanged (Greenland & Drescher 1993, Rockhill et al. 1998a, Benichou 2001).

The multivariate approach used in the present study included covariates with variable degrees of associations with periodontal diseases. Our analytical model adjusted for the effect of supragingival calculus, as a measure of oral hygiene, when assessing the association between smoking and PAL. In contrast, previous studies did not adjust for this variable. Notably, in the present analysis, excluding the calculus variable from the analytical model resulted in an increase in the estimate of %PAF from 12.2% to 19.8%. Hence, it is likely that the higher estimates of %PAF reported by other studies may also be attributed to the lack of adjustment for dental calculus.

In the present analysis, we predicted the number of exposed individuals (smokers) with or without severe attachment loss, and these estimates were used in the calculation of the PAF estimates (Greenland & Drescher 1993, Benichou 2001). In contrast, Hyman & Reid (2003) and Tomar & Asma (2000) used the prevalence of smoking in their populations and the respective OR for smoking, in the calculation of PAF estimates. The latter method is based on the assumption that the OR is an approximation of the relative risk when the prevalence of disease is low (<10%) (Zhang & Yu 1998, Szklo & Nieto 2000, Eide & Heuch 2001). However, this Brazilian population had a relatively high prevalence of severe attachment loss, and the assumption used in previous studies may, therefore, not be valid. The difference in the analytical approach between this and the two other studies may also have contributed to some of the difference in the PAF estimates between these studies.

It has been suggested that analytical models which involve attributable fraction estimation should include only variables that are causally associated with the disease and that are modifiable

through prevention and intervention (Rockhill et al. 1998a, b, Szklo & Nieto 2000, Eide & Heuch 2001). Surveys may provide valuable data about the occurrence of disease and prevalence of potential risk factors in populations, but they do not provide proof of causality (Albandar & Rams 2002). Moreover, this study design may provide important information needed to calculate PAF values, not feasible with other study designs (Walter 1976).

For smoking, current knowledge suggests that there is a strong association between this variable and destructive periodontal diseases, and enough evidence does exist to characterize smoking as a true risk factor of these diseases (Gelskey 1999, Albandar 2002). In this regard, our findings are consistent with other studies showing a significant effect of cigarette smoking on the occurrence of periodontal diseases (Grossi et al. 1994, Gelskey 1999, Albandar et al. 2000, Bergstrom et al. 2000, Corbet et al. 2001, Hyman & Reid 2003).

Most studies that have addressed the relationship between smoking and periodontitis have been based on a self-reported assessment of tobacco consumption. Self-reporting may be influenced by cultural and social factors, and the effects of smoking on health may also be influenced by individual variations because of differences in metabolism, depth of inhalation, and nicotine concentration in cigarettes. An alternative approach to self-reporting may include the assessment of specific metabolites, such as cotinine, which are present in serum following tobacco consumption. The assessment of metabolites, however, may measure smoking levels in current smokers only (Scott et al. 2001, Spiekerman et al. 2003). Furthermore, the self-reported assessments showed a very high level of reproducibility in this study population ($\kappa = 0.92$).

While interesting from a conceptual point of view, the complete elimination of an exposure is often an unattainable public health goal, whereas a reduction in the prevalence and severity of exposure is a more realistic objective (Rockhill et al. 1998a, b). Hence, a decline in the smoking PAF and the overall prevalence of severe attachment loss in the population may be expected if preventive interventions were applied. Moreover, since exposure to a risk factor is cumulative in nature, cessation

of exposure should not reduce the risk in previously exposed individuals to the same level observed in those that have never been exposed (Szklo & Nieto 2000). Evidently, prevention of periodontitis in former smokers cannot be achieved by means of a smoking cessation program. Nevertheless, inclusion of former smokers in the analysis is useful in the calculation of the total burden of disease that may be attributed to smoking.

A multidisciplinary approach is probably the most appropriate strategy for the prevention of periodontal diseases. Consequently, targeting exposures that also are risk factors for systemic diseases may have a better chance of success, and may also enhance the benefits and effectiveness of public health interventions (Ezzati et al. 2002). Since smoking is also an important risk factor for other diseases, a common risk factor approach would be to include periodontal diseases in ongoing or planned intervention campaigns designed to prevent smoking-related diseases (Sheiham & Watt 2000).

Cigarette smoking was strongly associated with severe attachment loss in this study population, and a significant percentage of cases might have been prevented if smoking cessation interventions had been implemented. The results suggest a need for population-based smoking cessation programs in an attempt to reduce the incidence of severe attachment loss in populations with high level of smoking exposure.

Acknowledgments

Funding for this project was provided by Foundation for Post-graduate Education (CAPES), Ministry of Education, Brazil, Grant number 1614/99-1.

References

- Albandar, J. M. (2002) Global risk factors and risk indicators for periodontal diseases. *Periodontology 2000* **29**, 177–206.
- Albandar, J. M. & Rams, T. E. (2002) Global epidemiology of periodontal diseases: an overview. *Periodontology 2000* **29**, 7–10.
- Albandar, J. M., Streckfus, C. F., Adesanya, M. R. & Winn, D. M. (2000) Cigar, pipe, and cigarette smoking as risk factors for periodontal disease and tooth loss. *Journal of Periodontology* **71**, 1874–1881.
- Benichou, J. (2001) A review of adjusted estimators of attributable risk. *Statistical Methods in Medical Research* **10**, 195–216.
- Bergstrom, J., Eliasson, S. & Dock, J. (2000) A 10-year prospective study of tobacco smok-

- ing and periodontal health. *Journal of Periodontology* **71**, 1338–1347.
- Corbet, E., Wong, M. & Lin, H. (2001) Periodontal conditions in adult Southern Chinese. *Journal of Dental Research* **80**, 1480–1485.
- Eide, G. E. & Heuch, I. (2001) Attributable fractions: fundamental concepts and their visualization. *Statistical Methods in Medical Research* **10**, 159–193.
- Ezzati, M., Lopez, A. D., Rodgers, A., Hoorn, S. V. & Murray, C. J. L. (2002) Selected major risk factors and global and regional burden of disease. *The Lancet* **360**, 1347–1360.
- Gelskey, S. C. (1999) Cigarette smoking and periodontitis: methodology to assess the strength of evidence in support of a causal association. *Community Dentistry and Oral Epidemiology* **27**, 16–24.
- Greenland, S. & Drescher, K. (1993) Maximum likelihood estimation of the attributable fraction from logistic models. *Biometrics* **49**, 865–872.
- Grossi, S. G., Zambon, J. J., Ho, A. W., Koch, G., Dunford, R. G., Machtei, E. E., Norderyd, O. M. & Genco, R. J. (1994) Assessment of risk for periodontal disease. I. Risk indicators for attachment loss. *Journal of Periodontology* **65**, 260–267.
- Hubert, L. (1977) Kappa revisited. *Psychological Bulletin* **84**, 289–297.
- Hyman, J. J. & Reid, B. C. (2003) Epidemiologic risk factors for periodontal attachment loss among adults in the United States. *Journal of Clinical Periodontology* **30**, 230–237.
- IBGE (1991) *Censo demográfico 1991*. Instituto Brasileiro de Geografia e Estatística.
- IBGE (1996) *Contagem da população*. Instituto Brasileiro de Geografia e Estatística.
- Kleinbaum, D. G., Kupper, L. L. & Morgenstern, H. (1982) *Epidemiologic Research. Principles and Quantitative Methods*. New York: John Wiley & Sons.
- Korn, E. & Graubard, B. (1999) *Analysis of Health Surveys*. New York: John Wiley & Sons, Inc.
- Metroplan (1997) *Dados cartográficos sobre a situação ambiental da Região Metropolitana de Porto Alegre - Levantamento do quadro ambiental da RMPA*. Fundação de Planejamento Metropolitano e Regional – Secretaria da Coordenação e Planejamento do Estado do Rio Grande do Sul.
- Rockhill, B., Newman, B. & Weinberg, C. R. (1998a) Use and misuse of population attributable fractions. *American Journal of Public Health* **88**, 15–19.
- Rockhill, B., Weinberg, C. R. & Newman, B. (1998b) Population attributable fraction estimation for established breast cancer risk factors: considering the issues of high prevalence and unmodifiability. *American Journal of Epidemiology* **147**, 826–833.
- Scott, D. A., Palmer, R. M. & Stapleton, J. A. (2001) Validation of smoking status in clinical research into inflammatory periodontal disease. *Journal of Clinical Periodontology* **28**, 715–722.

- Sheiham, A. & Watt, R. G. (2000) The common risk factor approach: a rational basis for promoting oral health. *Community Dentistry and Oral Epidemiology* **28**, 399–406.
- Shrout, P. & Fleiss, J. (1979) Intraclass correlations: uses in assessing rater reliability. *Psychological Bulletin* **86**, 420–428.
- Spiekerman, C. F., Hujoel, P. P. & DeRouen, T. A. (2003) Bias induced by self-reported smoking on periodontitis-systemic disease associations. *Journal of Dental Research* **82**, 345–349.
- Szklo, M. & Nieto, F. J. (2000) *Epidemiology. Beyond the Basics*. Maryland: Aspen Publishers, Inc.
- Teng, H. C., Lee, C. H., Hung, H. C., Tsai, C. C., Chang, Y. Y., Yang, Y. H., Lu, C. T., Yen, Y. Y. & Wu, Y. M. (2003) Lifestyle and psychosocial factors associated with chronic periodontitis in Taiwanese adults. *Journal of Periodontology* **74**, 1169–1175.
- Tomar, S. L. & Asma, S. (2000) Smoking-attributable periodontitis in the United States: findings from NHANES III. National Health and Nutrition Examination Survey. *Journal of Periodontology* **71**, 743–751.
- Walter, S. D. (1976) The estimation and interpretation of attributable risk in health research. *Biometrics* **32**, 829–849.
- Zhang, J. & Yu, K. F. (1998) What's the relative risk? A method of correcting the odds ratio in cohort studies of common outcomes. *Journal of the American Medical Association* **280**, 1690–1691.

Address:
Jasim M. Albandar
Department of Periodontology
Temple University School of Dentistry
3223 North Broad Street
Philadelphia, PA 19140, USA
Fax: +1 215 707 7616
E-mail: albandar@temple.edu

Paper III

Effect of Partial Recording Protocols on Estimates of Prevalence of Periodontal Disease

Cristiano Susin,^{*†‡} Albert Kingman,[§] Jasim M. Albandar^{*}

Aims: The aim of this study was to assess the degree of underreporting in the estimates of prevalence of clinical attachment loss due to different partial recording protocols (PRPs) in epidemiological studies, and to derive a correction factor to adjust for this bias.

Methods: The study sample included 1,460 dentate persons 14–103 years old who were examined clinically to assess the clinical attachment loss at 6 sites per tooth. Seven PRPs based on full-mouth or half-mouth designs were assessed, and the bias and sensitivity in the assessment of attachment loss prevalence for these protocols were assessed.

Results: All partial protocols underestimated the prevalence of attachment loss. Bias estimates for any full-mouth PRP were smaller than those for the corresponding site-combination PRPs for the half-mouth design. The PRP using the mesiobuccal (MB), midbuccal (B), and distolingual (DL) sites of teeth in all 4 quadrants showed the smallest bias and highest sensitivity of prevalence estimates among the 7 PRPs evaluated, uniformly across the range of attachment loss severity level. The 3-site PRP incorporating the DL site produced less bias than the 3-site PRP including the distobuccal (DB) site. There was a 3%-12% gain in sensitivity for 2-5mm attachment loss thresholds for the 3-site half-mouth PRPs compared with the 2-site MB, B half-mouth PRP.

Conclusions: The bias in the assessment of attachment loss is influenced by the partial recording design and the type and number of sites assessed, and is also influenced by the severity of attachment loss in the study population. These factors should be considered when selecting a partial recording method in large surveys. *J Periodontol* 2005;76:

KEY WORDS

Periodontal diseases/diagnosis, partial recording, periodontal attachment loss, bias.

* Periodontal Diagnostics Research Laboratory, Department of Periodontology, Temple University School of Dentistry, Philadelphia, USA

† Faculty of Dentistry, University of Bergen, Bergen, Norway

‡ Department of Periodontology, Lutheran University of Brazil, Canoas, Brazil

§ Biostatistics Core, National Institute of Dental and Craniofacial Research, National Institutes of Health, Bethesda, Maryland, USA

A protocol which consists of full-mouth clinical periodontal assessments made on 6 sites per tooth is at present the gold-standard method for the assessment of the periodontal disease status of subjects and is routinely used in the periodontology clinic. However, the use of this protocol in large surveys may not be feasible. Full-mouth examinations require a lot of resources and are time and labor consuming. In addition, this method could trigger patient and examiner fatigue, and this may potentially increase measurement errors and increase dropout rates.

Various partial recording protocols (PRP) have been recommended to overcome some of the problems associated with full-mouth examinations. Some of the widely used methods are based on clinical measurements made on multiple sites around the circumference of index teeth.¹⁻⁴ A second approach uses a random half-mouth scheme, and conducts measurements on selected sites, typically 2-3, per tooth. The latter method has been used routinely in national^{5,6} and regional^{7,8} surveys in the USA. In addition, partial recordings using full-mouth examinations have also been used.⁹⁻¹²

Kingman and Albandar¹² studied the effect of PRPs on the estimates of periodontal disease prevalence in a sample of young subjects diagnosed with early-onset

forms of periodontal disease including aggressive and chronic periodontitis, and a group of matched controls selected in a national survey. The study showed that PRPs based on periodontal measurements of 2 or 3 buccal sites and random half-mouth may significantly underestimate the true prevalence of clinical attachment loss and probing depth in a population.

The aims of this study were to assess the degree of underreporting in prevalence estimates of clinical attachment loss obtained in epidemiological studies by employing specific PRPs, and to derive estimated correction factors to adjust for this bias.

MATERIAL AND METHODS

This study used a representative sample of subjects 14–103 years old (mean: 38.0, SD: 17.4 years) living in 14 major municipalities which constitute the metropolitan area of Porto Alegre in the Brazilian state of Rio Grande do Sul. The sample included 1,460 dentate persons drawn by means of a multistage probability sampling method. A detailed description of the sampling method and the target population is provided elsewhere.¹³

The subjects were examined clinically in a mobile examination center consisting of a trailer equipped with a complete dental unit, and the center was moved from one examination location to the next according to the survey schedule. Four dentists and two dental assistants conducted the fieldwork. All permanent fully erupted teeth, excluding third molars, were examined with a manual periodontal probe (PCP10-SE, Hu-Friedy Mfg. Co. Inc., Chicago, USA) color coded at 1,2,3,5,7,8,9,10 mm. Six sites per tooth were assessed in the mesiobuccal (MB), midbuccal (B), distobuccal (DB), distolingual (DL), midlingual (L), and mesiolingual (ML) sites.

Probing depth was defined as the distance from the free gingival margin to the bottom of the pocket/sulcus. Gingival recession was defined as the distance from the cemento-enamel junction (CEJ) to the free gingival margin, and this assessment was assigned a negative sign if the gingival margin was located coronal to the CEJ. Clinical attachment loss was defined as the distance from the CEJ to the bottom of the pocket/sulcus, and was calculated as the sum of the probing depth and gingival recession measurements. Measurements were made in millimeters and were rounded to the lower whole millimeter.

Measurement reproducibility

At two time points, before and 3 months after the start of the study, the examiners were trained and calibrated in performing the clinical measurements. The examination team followed a quality control

protocol designed to minimize systematic and random measurement errors and to quantify what error remained. The protocol involved standard examination environment and methodology, standard equipment, and detailed written instructions for clinical procedures.

Assessment of measurement reproducibility used replicate periodontal measurements performed during the fieldwork. One examiner with the most clinical experience served as the “gold standard” examiner. A total of 57 subjects, divided into four groups ranging from 8 to 20 subjects, were used for the reproducibility assessment. In one of the groups, the replicate measurements consisted of repeated measurements by the gold standard examiner. In each of the remaining 3 groups, one examiner and the gold standard examiner conducted replicate measurements. Measurement error was estimated for the gold standard examiner.¹² Measurement reproducibility at the subject level was assessed by the intraclass correlation coefficient¹⁴ and weighted kappa, and at the site level by the weighted Kappa.¹⁵

Partial recording protocols

Seven PRPs were assessed in this study:

- a) Mesiobuccal, and midbuccal measurements on all teeth (MB-B, full-mouth).
- b) Mesiobuccal, midbuccal, and distobuccal measurements on all teeth (MB-B-DB, full-mouth).
- c) Mesiobuccal, midbuccal, and distolingual measurements on all teeth (MB-B-DL, full-mouth).
- d) Mesiobuccal, and midbuccal measurements on all teeth in one maxillary and one mandibular, randomly selected quadrants (MB-B, half-mouth).
- e) Mesiobuccal, midbuccal, and distobuccal measurements on all teeth in one maxillary and one mandibular, randomly selected quadrants (MB-B-DB, half-mouth).
- f) Mesiobuccal, midbuccal, and distolingual measurements on all teeth in one maxillary and one mandibular, randomly selected quadrants (MB-B-DL, half-mouth).
- g) Mesiobuccal, midbuccal, distobuccal, distolingual, midlingual, and mesiolingual sites on all teeth in one maxillary and one mandibular, randomly selected quadrants (6 sites, half-mouth).

Data analysis

Prevalence was defined as the percentage of individuals having at least one tooth with a given threshold of clinical attachment loss.^{12,16} Absolute bias was defined as the difference in prevalence

estimates for a specific PRP and that determined by the full-mouth evaluation (FM), which consisted of 6 measurements made on all teeth. That is, Bias = PRP estimate – FM score (Fig. 1). Prevalence estimates derived from PRPs are inherently non-positive, i.e. underestimates of true prevalence. Sensitivity was defined as the proportion of diseased persons who have a positive test, and was calculated as the ratio of the prevalence of clinical attachment loss using a given partial system, relative to the true prevalence. Specificity was defined as the proportion of disease-free persons who have a negative test, and for the present PRPs the specificities were 100%.^{12,16} An inflation factor was derived to adjust for the underestimation of prevalence measurements, and was calculated as the inverse function of sensitivity. Statistical analysis was performed using SAS*.

RESULTS

Measurement error for the gold standard was 0.24 mm. The gold standard showed a high reproducibility of attachment loss measurements, with 91.1% of the measured sites were within ± 1 mm for the repeated assessments (intra-examiner agreement). The weighted kappa (± 1 mm) of the site measurements was 0.87. The weighted kappa (± 1 mm) at subject level prevalence (maximum clinical attachment loss) was 1.00. Compared to the gold standard, the 3 other examiners showed inter-examiner site measurements percentage agreement (within ± 1 mm) between 77.9% and 80.7%, and site-level weighted kappas ranging between 0.65 and 0.71. The weighted kappa (± 1 mm) at subject level prevalence (maximum clinical attachment loss) ranged between 0.69 and 0.92.

There were large differences among the estimates of attachment loss prevalence produced by the 7 PRPs (Table 1). The degree of underestimation is displayed in Figure 2 (the negative of the bias is presented to facilitate presentation) for the 7 PRPs. PRPs based on full-mouth produced smaller underestimates for attachment loss prevalence than the comparable half-mouth site-combination PRPs. Additionally, the type and number of sites evaluated also affected the amount of underestimation. For protocols that used only 2 or 3 sites, the system that used MB, B, DL yielded the largest prevalence rates, for both the half-mouth and full-mouth design. In most PRPs, the bias in the prevalence estimates was highest for moderate (4-7 mm) attachment loss (Fig. 2). A PRP based on the MB, B, DL, full-mouth performed the best among the 7 PRPs evaluated,

uniformly across the severity of attachment loss used to define disease.

Different recording systems also showed varying sensitivities for assessment of attachment loss prevalences (Table 2). Generally, better sensitivities were seen in systems that used full-mouth than half-mouth design, and in 3-site systems that used DL rather than DB sites.

We calculated an inflation factor to adjust for the bias in attachment loss prevalence. The magnitude of inflation was positively correlated with the severity of attachment loss, and the correlation was stronger for the half-mouth than full-mouth methods (Fig. 3). In addition, the inflation factor was considerably smaller for the full-mouth than half-mouth methods, and for 3-site protocols that included DL rather than DB measurements. The MB, B, DL full-mouth protocol showed the smallest inflation factor among all 7 PRPs assessed.

The data were also analyzed separately for each of the 4 examiners. The results showed some variability among the different examiners, although the trends were similar to the pooled data.

DISCUSSION

The results suggest that partial recording protocols (PRPs) that use full-mouth measurements produce less bias in reporting of attachment loss prevalence than systems using similar sites and random half-mouth design. Furthermore, among 2 and 3-site PRPs based on full-mouth and half-mouth systems, the 3-site: mesiobuccal (MB), midbuccal (B), distolingual (DL) protocol performed the best for full-mouth and half-mouth classes of PRPs tested, separately. Overall, among the 7 PRPs assessed in this study, the 3-site MB, B, DL protocol based on teeth in all 4 quadrants showed the smallest bias in prevalence estimates.

This study also showed that the magnitude of the PRPs bias in assessing disease prevalence is a function of the underlying prevalence. Therefore, the bias due to a given system may increase with the increase in the severity of attachment loss within the population. This suggests that the performance of different systems will, to some extent, be affected by the characteristics of the population. Consequently, a given diagnostic system may perform differently in populations of different demographics such as sex, ethnicity, and age.

In evaluating the usefulness of diagnostic methods, including different PRPs, both the sensitivity and specificity are important criteria, and should be taken in consideration when selecting a suitable system. Since the specificity of a PRP is necessarily 100% we can restrict our attention to a comparison of PRP sensitivities. Although we derive

* SAS, version 9.0 for Windows, SAS Institute, Inc., Cary, NC

estimates for sensitivity and thus also the inflation factors, across the range of attachment level severity, we suggest the use of 80% threshold for an adequate PRP in an epidemiological survey. This insures that the inflation factor will not exceed 1.25 and thus the degree of underestimation for disease prevalence will be minimized. In table 2, adequate performance with sensitivity values of 80% or larger were highlighted. The table shows that the limitations of various PRPs are a function of disease level. Our study results showed that the choice of a suitable 3-site half-mouth PRP will be adequate for estimating attachment loss prevalence in the range of 2 mm to 4 mm, and rapidly declines for larger values. If one is willing to employ a 3-site full-mouth PRP adequate performance can be achieved for the range 2 mm to 6 mm, or possibly to 8 mm with the MB, B, DL protocol.

The present results corroborate earlier findings reported by Kingman and Albandar¹² showing that full-mouth protocols have higher sensitivity than random half-mouth protocols, and that a system that uses the MB, B, DB half-mouth sites shows lower bias in estimates of disease prevalence than a system that uses only the MB, B half-mouth sites. These findings differ in that we show that the 3-site MB, B, DL protocol performs better than the 3-site MB, B, DB protocol generally. Notably, comparison of similar 3-site PRPs in the two studies suggests that partial protocols generally showed higher sensitivities in the present study population for various thresholds of attachment loss. This may be due to the nature of the type of periodontal disease among the population studied by Kingman and Albandar¹² or the higher prevalence of periodontal disease in this population.

A recent study used a random half-mouth protocol of 6 sites per tooth and reported high sensitivity for estimates of attachment loss prevalence.^{1,9} In the present study this protocol also showed high sensitivity for assessing prevalence rates. However, this PRP showed a somewhat lower sensitivity and higher bias than a protocol consisting of 3 sites, the MB, B, DL full-mouth protocol. Thus, one would do better by evaluating 3-sites for all 4 quadrants than use 6-sites per tooth on a half-mouth basis.

Direct comparisons of the present findings with other studies are difficult since most studies have assessed probing depth rather than attachment loss,^{2,4,17-19} or used fewer than 6 sites per tooth when assessing the true prevalence of attachment loss.^{16,20,21} Moreover, previous studies have used small samples,^{1,9,20} included individuals with certain demographics characteristics,^{1,16,17,19,20} or used convenience samples.^{9,16,21} In contrast, the present study used a large representative sample which permits

higher precision of estimates. However, the reported findings may pertain to a particular Brazilian population, and the validity of the findings will need to be substantiated for other populations.

In this study we derived an inflation factor to adjust for the inaccuracies in the estimates of attachment loss prevalence. The findings showed a higher percentage inflation is needed for half-mouth than full-mouth protocols that use similar sites, and for methods that included the DB rather than the DL sites. In addition, the magnitude of inflation correlated positively with disease severity, and for most protocols it increased sharply for attachment loss thresholds above 3 mm. Notably, however, PRP methods that were based on full-mouth measurements at 3 sites per tooth required only $\leq 19\%$ inflation of their estimates of attachment loss for thresholds ≤ 6 mm (Fig. 3).

In large surveys, limited resources, including manpower, funds, and time are among the main rationales for not using a measurement protocol of full-mouth and six sites per tooth. In addition, other fieldwork logistics constrains may influence the choice of the partial recording method used. A careful consideration of these factors should be undertaken to select a suitable diagnostic method that shows satisfactory precision.

ACKNOWLEDGMENT

Funding for this project was provided by Foundation for Post-Graduate Education (CAPES), Ministry of Education, Brasilia, DF, Brazil. Grant number 1614/99-1.

REFERENCES

1. Dowsett SA, Eckert GJ, Kowolik MJ. The applicability of half-mouth examination to periodontal disease assessment in untreated adult populations. *J Periodontol* 2002;73:975-981.
2. Diamanti-Kipiotti A, Papapanou PN, Moraitaki-Tsami A, Lindhe J, Mitsis F. Comparative estimation of periodontal conditions by means of different index systems. *J Clin Periodontol* 1993;20:656-661.
3. Baelum V, Papapanou PN. CPITN and the epidemiology of periodontal disease. *Community Dent Oral Epidemiol* 1996;24:367-368.
4. Baelum V, Fejerskov O, Manji F, Wanzala P. Influence of CPITN partial recordings on estimates of prevalence and severity of various periodontal conditions in adults. *Community Dent Oral Epidemiol* 1993;21:354-359.
5. Albandar JM, Brunelle JA, Kingman A. Destructive periodontal disease in adults 30 years of age and older in the United States, 1988-1994. *J Periodontol* 1999;70:13-29.

6. Albandar JM, Kingman A. Gingival recession, gingival bleeding, and dental calculus in adults 30 years of age and older in the United States, 1988-1994. *J Periodontol* 1999;70:30-43.
7. Hunt RJ, Levy SM, Beck JD. The prevalence of periodontal attachment loss in an Iowa population aged 70 and older. *J Public Health Dent* 1990;50:251-256.
8. Gilbert GH, Heft MW. Periodontal status of older Floridians attending senior activities centers. *J Clin Periodontol* 1992;19:249-255.
9. Owens JD, Dowsett SA, Eckert GJ, Zero DT, Kowolik MJ. Partial-mouth assessment of periodontal disease in an adult population of the United States. *J Periodontol* 2003;74:1206-1213.
10. Fox CH, Jette AM, McGuire SM, Feldman HA, Douglas CW. Periodontal disease among New England elders. *J Periodontol* 1994;65:676-684.
11. Beck JD, Koch GG, Rozier RG, Tudor GE. Prevalence and risk indicators for periodontal attachment loss in a population of older community-dwelling blacks and whites. *J Periodontol* 1990;61:521-528.
12. Kingman A, Albandar JM. Methodological aspects of epidemiological studies of periodontal diseases. *Periodontol* 2000 2002;29:11-30.
13. Susin C, Dalla Vecchia CF, Oppermann RV, Haugejorden O, Albandar JM. Periodontal attachment loss in an urban population of Brazilian adults: the effect of demographic, behavioral and environmental risk indicators. *J Periodontol* 2004;75:1033-1041.
14. Shrout P, Fleiss J. Intraclass correlations: uses in assessing rater reliability. *Psychol Bull* 1979;86:420-428.
15. Hubert L. Kappa revisited. *Psychol Bull* 1977;84:289-297.
16. Kingman A, Morrison E, Loe H, Smith J. Systematic errors in estimating prevalence and severity of periodontal disease. *J Periodontol* 1988;59:707-713.
17. Hunt RJ. The efficiency of half-mouth examinations in estimating the prevalence of periodontal disease. *J Dent Res* 1987;66:1044-1048.
18. Ainamo J, Ainamo A. Partial indices as indicators of the severity and prevalence of periodontal disease. *Int Dent J* 1985;35:322-326.
19. Hunt RJ, Fann SJ. Effect of examining half the teeth in a partial periodontal recording of older adults. *J Dent Res* 1991;70:1380-1385.
20. Thomson WM, Williams SM. Partial- or full-mouth approaches to assessing the prevalence of and risk factors for periodontal disease in young adults. *J Periodontol* 2002;73:1010-1014.
21. Papapanou PN, Wennstrom JL, Johnsson T. Extent and severity of periodontal destruction based on partial clinical assessments. *Community Dent Oral Epidemiol* 1993;21:181-184.

Correspondence:

Dr. Jasim M. Albandar, Department of Periodontology, Temple University School of Dentistry, 3223 North Broad St., Philadelphia, PA 19140, USA. Fax: 1-215-707-7616; email: Jasim.Albandar@temple.edu

Accepted for publication June 2, 2004.

Table 1.
Prevalence estimates for different thresholds of periodontal attachment loss, by type of partial recording method.

Attachment loss (mm)	Full-mouth				Random half-mouth			
	6 sites*	MB-B	MB-B-DB	MB-B-DL	6 sites	MB-B	MB-B-DB	MB-B-DL
≥1	96.4	92.1	93.4	94.2	92.5	87.6	89.3	90.2
≥2	89.1	82.9	85.4	85.5	85.1	76.4	79.7	80.7
≥3	78.8	71.3	73.8	75.3	74.0	65.4	68.6	69.8
≥4	69.3	59.5	63.7	65.1	63.4	51.8	55.5	57.4
≥5	55.8	44.7	49.8	51.4	50.0	37.3	41.5	44.2
≥6	43.6	32.5	36.6	38.9	36.2	24.7	28.3	31.4
≥7	33.9	23.3	26.0	30.0	27.0	17.0	19.5	22.8
≥8	24.9	15.5	18.1	20.8	18.3	11.3	13.1	15.5
Maximum number of sites per subject	168	56	84	84	84	28	42	42

* True prevalence

Table 2.
The sensitivity of different partial recording systems in reporting the prevalence of various severity of attachment loss.*

Attachment loss (mm)	Full-mouth				Random half-mouth		
	MB-B	MB-B-DB	MB-B-DL	6 sites	MB-B	MB-B-DB	MB-B-DL
≥1	0.96	0.97	0.98	0.96	0.91	0.93	0.94
≥2	0.93	0.96	0.96	0.96	0.86	0.89	0.91
≥3	0.90	0.94	0.95	0.94	0.83	0.87	0.89
≥4	0.86	0.92	0.94	0.92	0.75	0.80	0.83
≥5	0.80	0.89	0.92	0.90	0.67	0.74	0.79
≥6	0.75	0.84	0.89	0.83	0.57	0.65	0.72
≥7	0.69	0.77	0.88	0.80	0.50	0.58	0.67
≥8	0.62	0.73	0.84	0.73	0.45	0.53	0.62

*Highlighted cells have ≥80% sensitivity

$$\text{Absolute bias} = \text{Prevalence}_{\text{PRP}} - \text{TruePrevalence}$$

$$\text{Sensitivity} = \frac{\text{Prevalence}_{\text{PRP}}}{\text{TruePrevalence}}$$

$$\text{Inflation factor} = \frac{\text{True Prevalence}}{\text{Prevalence}_{\text{PRP}}}$$

$$\text{Specificity} = \frac{\text{Number of Nondiseased}_{\text{PRP}}}{\text{Total Number of Nondiseased}}$$

Figure 1. Calculation of the absolute bias, sensitivity, specificity, and inflation factor. (PRP= partial recording protocol)

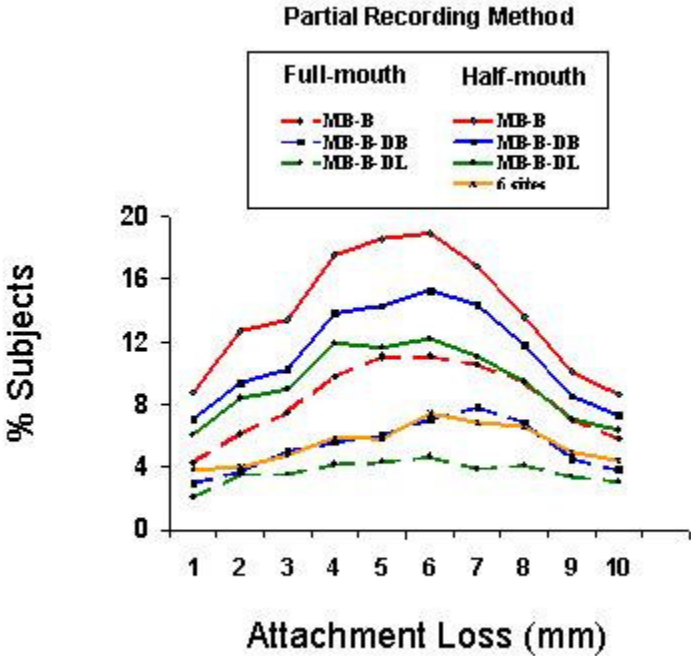


Figure 2. Estimates of the underestimation in the prevalence of attachment loss (absolute bias) using 7 different partial recording protocols, by severity of attachment loss. The negative value of the bias is shown here to facilitate presentation. (MB: mesiobuccal, B: midbuccal, DB: distobuccal, DL: distolingual)

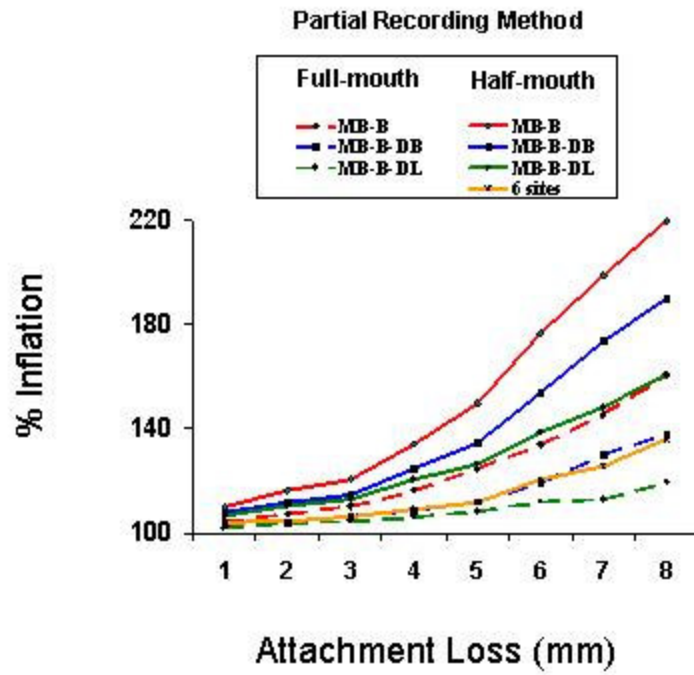


Figure 3. The percentage inflation of the estimated prevalences of attachment loss using 7 different partial recording protocols, by severity of attachment loss. (MB: mesiobuccal, B: midbuccal, DB: distobuccal, DL: distolingual)

Paper IV

Gingival recession: epidemiology and risk indicators in a representative urban Brazilian population

Cristiano Susin,*†‡ Alex N. Haas,§ Rui V. Oppermann,‡§ Ola Haugejorden,† Jasim M. Albandar*

Background: Gingival recession is a common manifestation of periodontal disease, but is also associated with other risk factors. A few studies have investigated the epidemiology and risk factors of this condition. This study describes the epidemiology of gingival recession in a representative, urban Brazilian population, and assesses various risk indicators.

Methods: A representative sample comprising 1460 subjects was selected using a multi-stage, probability, cluster sampling strategy. The subjects were interviewed using a structured questionnaire and had a full-mouth clinical examination in a mobile examination center.

Results: 51.6% and 22.0% of the individuals, and 17.0% and 5.8% of teeth per individual showed gingival recession ≥ 3 mm and ≥ 5 mm, respectively. The prevalence, extent, and severity of recession correlated with age. Recession showed a nonlinear relationship with age, with 25-50 years olds showing the highest level of recession among the age groups. Males aged ≥ 30 years showed significantly higher prevalence and extent of gingival recession than females. The percentage of teeth with recession was significantly higher in the low than in the high socio-economic status groups irrespective of age, and in subjects ≥ 30 years of age with irregular dental care than in subjects with regular care. Using a multivariable model, cigarette smoking and presence of supragingival calculus were the factors most significantly associated with localized and generalized recession, whereas gender, dental visits and socio-economic status were not significant risk indicators.

Conclusion: The high level of gingival recession in this Brazilian population may be primarily related to destructive periodontal disease, and is significantly associated with a high level of supragingival dental calculus and cigarette smoking. Population-based programs aimed at the prevention of periodontal diseases may reduce the prevalence of severe gingival recession in this and similar populations.

KEY WORDS

Periodontal disease/epidemiology, gingival recession, risk factors, smoking, dental calculus

Gingival recession is clinically manifested by an apical displacement of the gingival tissues, leading to root surface exposure, which often causes nuisances such as poor esthetics,^{1,2} increased susceptibility for root caries³ and dentine hypersensitivity.⁴ The mechanism by which gingival recession occurs is not well understood but it seems to be inflammatory in nature. The main etiological factors of this condition are the accumulation of dental plaque biofilm with the resulting inflammatory periodontal diseases, and mechanical trauma due to faulty oral hygiene technique.^{1,2,5,6} Several other risk factors have been postulated to play a role in the occurrence of recession, including aging, alveolar bone dehiscence, high frenum attachment, and smoking.^{1,2,7-11}

Gingival recession is a common manifestation in most populations. It is estimated that more than half of the United States adult population have recession, and on average about a quarter of the dentition is affected.¹² However, representative information about the occurrence and risk factors of gingival recession in other populations

* Department of Periodontology, Temple University School of Dentistry, Philadelphia, USA.

† Department of Odontology, University of Bergen, Bergen, Norway.

‡ Department of Periodontology, Lutheran University of Brazil, Canoas, Brazil.

§ Department of Periodontology, Federal University of Rio Grande do Sul, Porto Alegre, Brazil.

is limited. Notably, many surveys have used the Community Periodontal Index of Treatment Needs (CPITN) for the assessment of periodontal diseases, and this may have contributed to the scarcity of data about this subject since this index does not measure recession.

There is evidence that periodontal diseases are prevalent in Latin America.¹³ This may suggest that also gingival recession may be prevalent in the Latin America populations. This study is a part of a larger survey to study the oral health of the urban population in the Brazilian state of Rio Grande do Sul. The aims of this study were to: 1) assess the prevalence, extent and severity of gingival recession in a sample representative of a large segment of this urban population, and 2) assess the association of potential risk indicators with the occurrence of gingival recession in this population.

MATERIAL AND METHODS

Population

The target population of the present study was individuals aged 14 years and older living in an urban area with more than 3 million inhabitants and comprising 14 major municipalities in the metropolitan area of Porto Alegre. Porto Alegre is the capital of the Brazilian state of Rio Grande do Sul, which is located in the southern part of Brazil, neighboring Argentina and Uruguay.

Study design

A representative sample of the target population was derived using a multistage probability sampling method¹⁴ using information provided by Rio Grande do Sul State Government Agency for Metropolitan Affairs (METROPLAN)¹⁵ and the Brazilian Institute of Geography and Statistics (IBGE).¹⁶ Primary sampling units (PSU) were selected randomly from geographic areas that had been stratified by income level. The PSUs were selected with a probability proportional to size and using a sampling frame of these PSUs. Area sectors were then selected randomly within each geographic area, and the number of sectors selected was proportional to the number of sectors in each area. Households were sampled consecutively within the selected sectors. More detailed information about the study design is provided elsewhere.²³

Eligible household members who were 14 years of age or older and who agreed to participate in the survey were included in the study. Exclusion criteria were presence of diseases/conditions that may pose health risks to the participant or examiner, or that may

interfere with the clinical examination. These criteria included subjects who were diagnosed with psychiatric problems, or intoxicated with alcohol or drugs. Individuals requiring a prophylactic regimen of antibiotics were provided with the appropriate medicine before the clinical examination.

Study sample

924 households in the 29 randomly selected sectors were eligible for inclusion in the survey, and 142 (15.4%) of these were not accessible. At least three attempts on different days were made to examine the eligible household members while the examination team was in the same residential area.

The number of individuals 14 years and older who were eligible for the survey was 2,435. A total of 1,646 (67.6%) subjects were interviewed, of whom 1,586 (65.1%) subjects were examined clinically. The study subjects had an age range of 14 to 103 years (mean: 37.9, SD: 13.3 years), and comprised 719 (45.3%) males and 867 (54.7%) females, 1,309 (82.5%) whites and 277 (17.5%) non-whites. The distribution of the participants and the corresponding target population by gender and age groups is shown in Table 1. The study group comprised 1,465 dentate and 121 edentulous subjects. In this study we report gingival recession findings of 1,460 dentate persons for whom relevant clinical data were available. The overall mean tooth loss, excluding third molars, was 7.6 (SD:8.9); ranging from 0.5 (SD:1.5) among 14-19 years-old to 16.2 (SD: 6.9) for persons 70 years of age and older.

Among the subjects who did not participate in the survey, 636 (26.1%) were not at home, 127 (5.2%) refused to participate, 60 (2.5%) were interviewed but refused to be examined, and 26 (1.1%) were unable to attend the examination site because of an impairing physical condition.

Interview and clinical examinations

Interviews and clinical examinations of subjects were performed in a mobile examination unit consisting of a trailer equipped with a complete dental unit, comprising a dental chair, light, compressor, and other basic amenities. The examination unit was moved from one examination location to the next according to the survey schedule. Four dentists and two dental assistants performed the fieldwork between June and December, 2001. Letters were mailed to eligible households explaining the aims of the study and soliciting participation. In addition, a member of the examination team visited the

households and provided more information about the study and encouraged participation. Eligible subjects who consented to participate were interviewed to gather demographic, socio-economic, oral health and other health-related data using a structured written questionnaire.

Gingival recession was defined as the distance from the cemento-enamel junction (CEJ) to the free gingival margin (FGM) and was measured using a manual periodontal probe* color coded at 1,2,3,5,7,8,9 and 10 mm. All measurements were made in millimeters and were rounded to the lower whole millimeter. All permanent fully erupted teeth, excluding third molars, were examined, and the measurements were made at 6 sites per tooth, the mesiobuccal, midbuccal, distobuccal, distolingual, midlingual, and mesiolingual sites. Gingival recession was scored as zero if the FGM was located at the CEJ, and was assigned a negative sign if the FGM was coronal to the CEJ.

Ethical considerations

The study protocol was reviewed and approved by the following committees: Research Ethics Committee, Federal University of Rio Grande do Sul, Porto Alegre, Brazil; the National Commission on Ethics in Research, Ministry of Health, Brasilia, Brazil; Ethics in Medical Research Committee, University of Bergen, Bergen, Norway. Subjects who agreed to participate signed an informed consent form. At the conclusion of the study the participants were provided with a written report detailing their oral status and any diagnosed mucosal lesions. Patients with diagnosed pathological conditions were advised to seek specialist consultation and treatment.

Non-reponse analysis

Subsequent to the completion of the examinations, a random sample of 339 (39.9%) subjects was selected out of 849 eligible subjects who either refused to participate or were not available during the normal survey schedule.²³

The mean age of the nonrespondents group was 35.2 years, and included 51.3% males and 48.7% females, and 90.8% whites and 9.2% nonwhites. In contrast, the mean age of the study group was 38 years, and included 45.3% males and 54.7% females, and 82.5% whites and 17.5% nonwhites. When classified by the number of years of education, the nonrespondents and respondents groups included 7.4% and 22.3% subjects with 4 or

fewer years, 22.5% and 40.0% subjects with 5 to 8 years, and 70.1% and 37.8% subjects with more than 8 years of education, respectively. This suggests that the nonrespondents were similar to the study group in the mean age, but included somewhat higher percentages of males and whites, and had a higher education than the study participants. Any bias in the population parameter estimates¹⁷ which could arise due to the non-reponse was reduced by using a weight variable. The calculation of the weight variable was based on Census information provided by IBGE.¹⁸

Measurements reproducibility

At two time points, before and 3 months after the start of the study, the examiners were trained and calibrated in performing the clinical measurements. The examination team followed a quality control protocol that involved standard examination environment and methodology, standard equipment, and detailed written instructions for clinical procedures. The protocol was aimed at reducing systematic and random measurement errors and to quantify what error remained.

Assessment of measurement reproducibility used replicate periodontal measurements performed during the fieldwork. One examiner with the most clinical experience served as the "gold standard" examiner. A total of 57 subjects, divided into four groups ranging from 8 to 20 subjects, were used for the reproducibility assessment. In one of the groups, the replicate measurements consisted of repeated measurements by the gold standard examiner. In each of the remaining 3 groups, the replicate measurements were made by one examiner and the gold standard examiner. Gingival recession measurements reproducibility at the subject level was assessed by the intraclass correlation coefficient,¹⁹ and at the site level by the weighted Kappa.²⁰ The intraclass correlation coefficient for mean recession for the different examiners ranged between 0.96 and 0.99, and for extent of recession ≥ 3 mm ranged between 0.66 and 0.98. The weighted kappa (± 1 mm) at the subject level prevalence (maximum recession) was between 0.63 and 1.00. The weighted kappa values (± 1 mm) at site level ranged between 0.71 and 0.90. The intraclass correlation coefficient for supragingival calculus ranged between 0.73 and 0.98 at the site level, and between 0.66 and 0.99 at the tooth level.

Data analysis

Prevalence was defined as the percentage of individuals having at least one tooth with the condition, and extent was defined as the

* PCP10-SE, Hu-Friedy Inc., Chicago, USA

percentage of teeth per person having at least one site with the condition. The mean gingival recession was calculated and was used as a measure of the condition severity in the population. Severity was also assessed as thresholds of gingival recession.

Blacks and mulattos were combined into a “non-Whites” group as reliable criteria to distinguish between them were not available. The race of the subject was scored as “White” or “non-White”, since the study population included only a small percentage of other ethnic groups. Socio-economic status was scored by combining information about family economy using a standard Brazilian economy classification (CCEB) and the level of education of the individual. High socio-economic status was defined as having ≥ 9 years of education and being in the upper two tertiles of the CCEB economy classification, or having 5-8 years of education and being in the highest tertile of the CCEB classification. Low socio-economic status was defined as having 1-4 years of education, and being in the lowest two tertiles of the CCEB classification, or having 5-8 years of education and being in the lowest tertile of the CCEB classification. Individuals who had higher economy and education than the low socio-economic status group, but less than the high group were classified as having a middle socio-economic status.

The subjects were classified according to their self-reported frequency and reasons for dental visits during the last 5 years. Individuals who had visited a dentist on a regular basis for maintenance care were classified as having a regular dental care. Subjects who had visited a dentist only for emergency dental treatment, or had not visited a dentist during the last 5 years were classified as not receiving regular dental care. Most participants in this study claimed using a toothbrush regularly at least once a day, and this information was therefore not used in the present analysis.

The total exposure to cigarette smoking was calculated for current and former smokers combined, and was made separately for the younger (14-29 years) and older (30+ years) age cohorts. The total number of packs of cigarettes consumed in a life time was calculated as the number of cigarettes consumed per day, multiplied by number of days of habit, divided by 20 (1 pack). Individuals 14-29 years old were classified into 3 groups: non-smokers, light (1 – 912 packs) and moderate/heavy smokers (>912 packs). Subjects 30 years and older were classified into 4 groups: non-smokers, light (1

- 2734 packs), moderate (2735 - 7300 packs), and heavy smokers (>7300 packs). Presence of supragingival dental calculus was categorized into three categories according to the percentage of sites with calculus. For individuals 14-29 years the three categories were: $<5\%$, 5 to 15% and $>15\%$. Individuals 30 years and older were divided in: $<25\%$, 25% to 50% and $>50\%$.

Data analysis was performed by STATA software[†] and using survey commands that take into account the survey design, including stratification, clustering, and weighting and robust variance estimation. A weight variable was used to adjust for the probability of selection and deviations in the sample distributions from the target population distribution by age, gender and education.^{17,18} Pairwise comparisons of crude estimates were carried out using the Wald test.¹⁷ The chosen level of statistical significance was 5%, and the 95% confidence intervals (CI) were calculated.

Multinomial Logistic Regression Analyses for Complex Survey was used to model the relationship between gingival recession and potential explanatory variables. A new variable was calculated and used as the dependent variable in the analysis, based on the extents of gingival recession ≥ 1 mm for individuals 14 to 29 years old, and the extent of recession ≥ 3 mm for individuals 30 years and older. Hence, the subjects were scored as having localized or generalized recession if between 1% and 15%, or $\geq 16\%$ of the teeth were affected, respectively. Individuals without ≥ 1 mm recession (14-29 years olds) or ≥ 3 mm recession (≥ 30 years olds) were used as the reference groups in the models. In both analyses, the multinomial logistic regression method¹⁷ was used to assess the contribution of the independent variables to the probability of occurrence of localized or generalized gingival recession, separately, compared to the nonoccurrence of gingival recession. The probability of occurrence of recession was expressed as a relative risk ratio (RRR), which is equivalent to the odds ratio statistics in the ordinary logistic regression analysis. In each analysis, a model was first fitted in which all potential risk indicators were entered, and those that did not contribute significantly to the model were excluded.

In order to model the association between gingival recession and age, mean recession was used. The analysis suggested a non-linear relationship between gingival recession and age, and therefore we used a

[†] Stata 7.0 for Windows, Stata Corporation, College Station, TX, USA

piecewise linear regression analysis for complex surveys to study this relationship.¹⁷ Knots were used at the age points 25 years and 50 years, yielding three linear splines. The model adjusted for race, cigarette smoking, socio-economic status, and calculus. Gender did not have a significant effect, and was therefore removed from the model.

RESULTS

In all, 83.4%, 51.6% and 22.0% of the individuals, and 43.5%, 17.0% and 5.8% of teeth per individual showed gingival recession ≥ 1 mm, ≥ 3 mm and ≥ 5 mm, respectively. The prevalence, extent, and severity of recession were correlated with age (Table 2). Slight recession was prevalent, but recession thresholds ≥ 3 mm and ≥ 5 mm affected only a small percentage of teeth in subjects younger than 40 years of age. On the other hand, moderate recession was ubiquitous in the older age groups. Among subjects aged 40 years or older, $\geq 79\%$ of the subjects, and $\geq 32\%$ of teeth per subject had recession ≥ 3 mm.

In subjects 14-29 years old, the mandibular central and lateral incisors showed the highest prevalence of gingival recession ≥ 1 mm, with 32.8% and 24.5% of these teeth affected, respectively. Other teeth showing high prevalence of gingival recession were the mandibular second premolars (17.1%), the maxillary first molars (16.2%) and the maxillary first premolars (13.8%) (Fig. 1).

Maxillary first molars and mandibular second premolars had the highest frequency of recession ≥ 3 mm in individuals 30-49 years old (25.7% and 24.8%, respectively) and 50+ years old (58.8% and 62.0%, respectively) (Fig. 2).

In subjects younger than 30 years of age, there were no significant differences in the prevalence or extent of recession between males and females, or between whites and non-whites. However, in the age groups 30 years and older, males consistently showed higher prevalence and extent of gingival recession than females irrespective of the threshold used (Table 3). The percentages of subjects with gingival recession were notably higher in non-whites than in whites, whereas the percentages of teeth affected per subjects were comparable in the 2 race groups.

The percentage of teeth with recession ≥ 3 mm was significantly higher in the low than in the high socio-economic status groups, irrespective of age (<30 years: $p < 0.05$; ≥ 30 years: $p < 0.01$), whereas the prevalence of recession was comparable in the three socio-economic status groups (Table 4). In the 30 years and older group, subjects who have had

irregular dental care had significantly higher percentage of teeth with recession ≥ 3 mm than in subjects with regular care (29.1% vs. 18.3%, $p < 0.01$).

Young subjects who were moderate or heavy cigarette smokers, and those in the ≥ 30 years group who were heavy smokers had a significantly higher prevalence of recession ($p < 0.01$), and had higher percentages of teeth affected ($p < 0.01$) than subjects who did not smoke (Fig. 3). Furthermore, in both age groups individuals with a higher percentage of teeth with supragingival calculus had a significantly higher prevalence ($p < 0.01$) and percentage of teeth ($p < 0.01$) showing recession (Fig. 4).

A multivariable model showed that, in the <30 years old group, subjects with $> 15\%$ of teeth with calculus, or who were moderate/heavy smokers had increased risk for localized (RRR=2.3, $p < 0.05$; RRR=2.0, $p < 0.05$) and generalized (RRR=3.8, $p < 0.01$; RRR=3.8, $p < 0.01$) recession (Table 5). In this model, gender, dental care, race, and socio-economic status were not significant risk indicators of recession.

In the 30+ years old group, a multivariable analytical model showed that non-Whites were associated with a significantly higher risk for localized recession than whites (RRR=2.8, $p < 0.01$) (Table 6). In addition, a significantly higher risk for generalized recession was found in heavy smokers (RRR=3.0, $p < 0.01$) and in subjects with 25%-50% or $> 50\%$ teeth with calculus (RRR=2.2, $p < 0.01$; RRR=6.4, $p < 0.01$). Gender, dental care and socio-economic status were not associated with a significant increase in the risk for recession in this age group after adjusting for other factors.

Regression analysis adjusted for race, socio-economic status, cigarette smoking status, and percentage of teeth with supragingival dental calculus, showed a significant increase in the severity of gingival recession with age (Fig. 5). The rate of recession was similar in the age intervals 14-25 years ($\beta = 0.05$, $p < 0.01$) and 50-70 years ($\beta = 0.05$, $p < 0.01$), and significantly higher ($p < 0.01$) in the age group 25-50 years ($\beta = 0.10$, $p < 0.01$) than in the younger (14-25 years) and older (50-70 years) age cohorts.

DISCUSSION

This is the first population study of the epidemiology and risk factors of gingival recession in a representative Brazilian population. The results show that gingival recession was prevalent in this urban population. The prevalence of recession ≥ 3

mm and ≥ 5 mm, respectively, was 6% and 0% at 14-19 years of age, it increased to 94% and 65% in the 70+ years group, and the percentages of teeth affected were 0.5% and 0% in the young age group, and 63% and 34% in the older age group. Aging, cigarette smoking, and presence of supragingival calculus were important risk indicators of localized and generalized gingival recession. In addition, males, poor socio-economic status, and irregular dental care were also associated with a significantly higher level of recession.

A national survey of dental health performed in Brazil in 1986 used the CPITN and did not assess gingival recession.²¹ The NHANES III survey estimated that recession ≥ 3 mm affected 22.5% subjects, and 6.5% teeth in the United States adults 30 years and older.¹² In contrast, in the present population the prevalence and extent of recession ≥ 3 mm were 73.1% subjects and 26.8% teeth, respectively, or approximately 3-4 fold higher than the American population. In both populations, the teeth mostly affected with gingival recession ≥ 3 mm were the mandibular central incisors and the maxillary first molars. Many surveys have used partial recording strategies to assess gingival recession and other periodontal variables. Partial recording methods may cause varying degrees of measurement bias.²² In contrast, we used a full-mouth examination, and assessed 6 sites per tooth. Hence, some of the differences between the results of this study and the NHANES III study may be partially attributed to methodological differences. However, based on published estimates of the measurement bias due to partial recordings reported by Kingman and Albandar,²² it is reasonable to infer that a significant part of the difference in results between the 2 surveys is a genuine difference, and that this Brazilian population has a higher level of recession than the American population. This is also consistent with our previous findings of a high level of periodontal disease in this population.²³

The finding in this study that males had more gingival recession than females is consistent with similar results in other populations.^{9,11,12,24} Recession was not significantly different between whites and non-whites, which is somewhat in contrast to the results of NHANES III study in the American population which showed significantly higher prevalence and extent of recession in blacks than in whites.¹² This discrepancy in the findings may be partly due to differences in study design between the two surveys.

Aging was strongly associated with a higher level of recession in this material.

Moreover, our analysis revealed that the relationship between age and mean gingival recession was non-linear. The 14-25 and 50-70 years old groups had similar rates of recession, which were somewhat lower than that for the 26-49 years old group. This nonlinear pattern of recession with age may be attributed to the low rate of periodontal disease progression in the young group, and to the high frequency of tooth loss in the older age groups. In this population, non-edentulous subjects 50 years of age and older had on average 12.9 missing teeth. Notably, the nonlinear relationship of gingival recession with age in this population was consistent with a nonlinear relationship between the rate of alveolar bone loss and age described by Albandar et al.^{25,26} in a group of Norwegians followed over 2 and 6 years interval.

Cigarette smoking was strongly associated with the occurrence of localized and generalized gingival recession in young individuals. In older adults, however, smoking was associated with generalized recession only, and the association with localized recession was not statistically significant. Although there is ample evidence in the literature of a strong association between cigarette smoking and attachment loss,^{27,28} there has been some inconsistency with regards to the reported pattern of relationship between smoking and gingival recession. Some studies have used cross-sectional and case-control study design and reported a positive relationship between smoking and recession.^{7,8,10,11} However, a six-months follow-up study in a group of young subjects failed to show that smokers had increased risk for recession.²⁹

Presence of supragingival calculus was strongly associated with gingival recession in this study. Compared to subjects with low levels of calculus, and adjusting for other variables, 14-29 years old individuals with $>15\%$ sites with calculus had 2.3 and 3.8 times higher risk for having localized and generalized recession ≥ 1 mm, respectively. In addition, subjects 30 years of age or older, and with 25%-50%, and $>50\%$ sites with calculus, respectively, had 2.2 and 6.4 times higher risk for having generalized recession ≥ 3 mm. This is in agreement with the findings of other studies^{9,24,30} showing positive correlations between recession and dental calculus.

This population had a high level of periodontal disease,²³ and supragingival calculus was common. The observed positive association with dental calculus suggests that gingival recession in this population was related to chronic inflammatory periodontal

disease rather than a mechanical trauma from oral hygiene. On the other hand, populations with high standards of oral hygiene and low burden of periodontitis may reveal different associations, including an association with mechanical trauma.^{1,2,5,6}

The cross-sectional design of the present study does not permit an unequivocal inference about the causal relationship between the studied risk indicators and gingival recession. However, it may be concluded that the observed high level of recession in this Brazilian population is related to periodontal disease, and is significantly associated with a high level of supragingival dental calculus and cigarette smoking.

ACKNOWLEDGMENT

Funding for this project was provided by CAPES Foundation – Ministry of Education – Brazil, Grant number 1614/99-1.

REFERENCES

1. Kassab MM, Cohen RE. The etiology and prevalence of gingival recession. *J Am Dent Assoc* 2003;134:220-225.
2. Smith RG. Gingival recession. Reappraisal of an enigmatic condition and a new index for monitoring. *J Clin Periodontol* 1997;24:201-205.
3. Lawrence HP, Hunt RJ, Beck JD. Three-year root caries incidence and risk modeling in older adults in North Carolina. *J Public Health Dent* 1995;55:69-78.
4. Al-Wahadni A, Linden GJ. Dentine hypersensitivity in Jordanian dental attenders. A case control study. *J Clin Periodontol* 2002;29:688-693.
5. Litonjua LA, Andreana S, Bush PJ, Cohen RE. Toothbrushing and gingival recession. *Int Dent J* 2003;53:67-72.
6. Loe H, Ånerud Å, Boysen H. The natural history of periodontal disease in man: prevalence, severity and extent of gingival recession. *J Periodontol* 1992;63:489-495.
7. Albandar JM, Streckfus CF, Adesanya MR, Winn DM. Cigar, pipe, and cigarette smoking as risk factors for periodontal disease and tooth loss. *J Periodontol* 2000;71:1874-1881.
8. Calsina G, Ramon JM, Echeverria JJ. Effects of smoking on periodontal tissues. *J Clin Periodontol* 2002;29:771-776.
9. Josphura KJ, Kent RL, DePaola PF. Gingival recession: intra-oral distribution and associated factors. *J Periodontol* 1994;65:864-871.
10. Gunsolley JC, Quinn SM, Tew J, Gooss CM, Brooks CN, Schenkein HA. The effect of smoking on individuals with minimal periodontal destruction. *J Periodontol* 1998;69:165-170.
11. Martinez-Canut P, Lorca A, Magan R. Smoking and periodontal disease severity. *J Clin Periodontol* 1995;22:743-749.
12. Albandar JM, Kingman A. Gingival recession, gingival bleeding, and dental calculus in adults 30 years of age and older in the United States, 1988-1994. *J Periodontol* 1999;70:30-43.
13. Gjermo P, Rosing CK, Susin C, Oppermann R. Periodontal diseases in Central and South America. *Periodontol* 2000 2002;29:70-78.
14. Levy P, Lemeshow S. Sampling of populations. Methods and applications. 3rd ed. New York: John Wiley & Sons, Inc; 1999:70-79.
15. Rio Grande do Sul State Agency for Metropolitan Affairs. Environmental cartographic data of the Metropolitan Area of Porto Alegre (in Portuguese). Porto Alegre:Rio Grande do Sul State Agency for Metropolitan Affairs; 1997.
16. Brazilian Institute of Geography and Statistics. Demographics Census 1991 (in Portuguese). Rio de Janeiro:Brazilian Institute of Geography and Statistics; 1991.
17. Korn E, Graubard B. Analysis of health surveys. New York: John Wiley & Sons, Inc; 1999:92-93,159-165,193-196.
18. Brazilian Institute of Geography and Statistics. Population Census (in Portuguese). Rio de Janeiro:Brazilian Institute of Geography and Statistics; 1996.
19. Shrout P, Fleiss J. Intraclass correlations: uses in assessing rater reliability. *Psychol Bull* 1979;86:420-428.
20. Hubert L. Kappa revisited. *Psychol Bull* 1977;84:289-297.
21. Ministry of Health. Oral health survey: Brazil, urban area, 1986 (in Portuguese):Ministry of Health; 1988
22. Kingman A, Albandar JM. Methodological aspects of epidemiological studies of periodontal diseases. *Periodontol* 2000 2002;29:11-30.
23. Susin C, Dalla Vecchia CF, Oppermann RV, Haugejorden O, Albandar JM. Periodontal attachment loss in an urban population of Brazilian adults, and the effect of demographic, behavioral and environmental risk indicators. *J Periodontol* 2004;75:1033-1041.
24. Mumghamba EGS, Markkanen HA, Honkala E. Risk factors for periodontal diseases in Ilala, Tanzania. *J Clin Periodontol* 1995;22:347-354.
25. Albandar JM, Rise J, Gjermo P, Johansen JR. Radiographic quantification of alveolar

- bone level changes. A 2-year longitudinal study in man. *J Clin Periodontol* 1986;13:195-200.
26. Albandar JM. A 6-year study on the pattern of periodontal disease progression. *J Clin Periodontol* 1990;17:467-471.
27. Bergstrom J, Eliasson S, Dock J. A 10-year prospective study of tobacco smoking and periodontal health. *J Periodontol* 2000;71:1338-1347.
28. Hyman JJ, Reid BC. Epidemiologic risk factors for periodontal attachment loss among adults in the United States. *J Clin Periodontol* 2003;30:230-237.
29. Muller HP, Stadermann S, Heinecke A. Gingival recession in smokers and non-smokers with minimal periodontal disease. *J Clin Periodontol* 2002;29:129-136.
30. Hosanguan C, Ungchusak C, Leelasithorn S, Prasertsom P. The extent and correlates of gingival recession in non-institutionalised Thai elderly. *J Int Acad Periodontol* 2002;4:143-148.
31. Brazilian Institute of Geography and Statistics. Demographics Census 2000: Characteristics of the Population and Households- General Results (in Portuguese). Rio de Janeiro:Brazilian Institute of Geography and Statistics; 2001.

Correspondence:

Jasim M. Albandar, DDS, DMD, PhD
Department of Periodontology
Temple University School of Dentistry
3223 North Broad Street
Philadelphia, PA 19140, USA
Fax: 1-215-707-7616
email: albandar@temple.edu

Accepted for publication February 11, 2004.

Table 1. Number and percentage* of subjects in the study sample by gender and age group, and the corresponding number and estimated percentage of subjects they represent in the target population.

Age (years)	Dentate sample*				Whole sample*				Target population†	
	Male		Female		Male		Female		Males N=1,241,926	Females N=1,375,868
	N	%	N	%	N	%	N	%	%	%
14-19	133	9.1	130	8.9	133	8.4	130	8.2	6,5	6,5
20-29	158	10.8	191	13.0	158	10.0	191	12.0	11,4	11,7
30-39	137	9.4	158	10.8	137	8.6	160	10.1	11,2	12,1
40-49	108	7.4	146	10.0	109	6.9	151	9.5	8,4	9,3
50-59	84	5.7	91	6.2	91	5.7	109	6.9	5,1	5,8
60-69	40	2.7	45	3.1	58	3.7	69	4.4	3,1	4,1
≥ 70	19	1.3	25	1.7	33	2.1	57	3.6	1,8	3,1
Total	679	46.3	786	53.7	719	45.3	867	54.7	47,4	52,6

* Percentages are not adjusted for sampling bias

† 1996 Population census^{18, 31}

Table 2. Percentage of subjects and the percentage of teeth per subject with gingival recession, by age.

	Gingival recession	Age (years)														Total	
		14-19		20-29		30-39		40-49		50-59		60-69		70+			
		%	S.E.	%	S.E.	%	S.E.	%	S.E.	%	S.E.	%	S.E.	%	S.E.	%	S.E.
Subjects	≥1 mm	29.5	6.2	76.5	2.5	95.7	1.3	99.0	0.5	100.0	0.0	100.0	0.0	100.0	0.0	83.4	1.2
	≥2 mm	12.2	2.9	51.6	2.9	84.9	0.8	96.1	1.0	98.8	0.8	100.0	0.0	100.0	0.0	71.4	1.3
	≥3 mm	5.9	1.6	24.0	2.4	54.0	2.0	79.3	2.5	93.5	2.5	91.5	3.1	94.1	3.9	51.6	1.3
	≥5 mm	0.0	0.0	2.8	1.0	12.8	1.7	45.3	3.1	46.5	3.3	64.8	3.7	64.8	7.2	22.0	1.3
Teeth	≥1 mm	2.9	0.6	18.1	1.5	44.3	1.6	69.9	2.3	77.0	1.4	86.4	2.9	92.1	1.5	43.5	1.1
	≥2 mm	1.1	0.3	8.9	0.9	26.9	1.2	53.4	3.1	62.8	2.0	70.8	3.0	81.1	4.5	31.0	1.2
	≥3 mm	0.5	0.2	2.3	0.3	10.0	0.6	31.7	3.2	39.5	2.3	47.2	1.7	62.5	4.9	17.0	0.9
	≥5 mm	0.0	0.0	0.1	0.0	1.1	0.2	11.6	2.5	15.3	1.0	18.3	1.7	33.5	5.4	5.8	0.5

Table 3. Percentage of subjects, and the percentage of teeth per subject with gingival recession in individuals 30 years and older, by threshold of gingival recession, gender, and race.

	Gingival recession	Gender				p	Race				p
		Males		Females			Whites		Non-whites		
		%	S.E.	%	S.E.		%	S.E.	%	S.E.	
Prevalence	≥1 mm	98.9	0.6	97.1	1.0	0.14	97.7	0.7	98.8	0.7	0.35
	≥2 mm	94.5	1.3	90.8	1.2	0.11	91.6	0.9	96.1	0.9	0.03
	≥3 mm	77.3	2.4	69.3	1.1	0.01	70.5	1.0	83.4	3.2	0.001
	≥5 mm	41.5	2.1	28.8	1.7	<0.001	33.1	2.3	41.9	4.7	0.17
Extent	≥1 mm	66.3	1.7	60.1	1.4	0.03	63.2	0.9	62.4	2.7	0.78
	≥2 mm	51.4	2.0	42.8	1.5	0.007	47.1	1.1	46.0	3.3	0.74
	≥3 mm	31.1	1.6	22.9	1.5	0.002	26.6	1.1	27.4	3.5	0.83
	≥5 mm	12.1	1.2	7.0	0.9	0.004	9.1	0.8	10.8	1.8	0.40

Table 4. Percentage of subjects, and the percentage of teeth per subject with gingival recession, by threshold of gingival recession, age group, and socio-economic status.

Age	Gingival recession	High		Low		p*	Middle		p*	
		%	S.E.	%	SE		%	S.E.		
14-29 years	Prevalence	≥1 mm	57.5	3.5	67.7	5.2	0.10	53.3	3.3	0.49
		≥2 mm	32.8	4.0	48.5	4.0	0.03	29.0	2.4	0.52
		≥3 mm	15.4	2.2	24.9	4.7	0.12	10.2	2.4	0.18
		≥5 mm	0.5	0.5	3.8	1.5	0.07	0.5	0.4	0.99
	Extent	≥1 mm	10.1	1.2	17.6	2.1	0.01	9.2	1.1	0.65
		≥2 mm	4.8	0.5	8.9	1.5	0.06	4.0	0.8	0.47
		≥3 mm	1.2	0.1	2.6	0.5	0.01	0.9	0.4	0.50
		≥5 mm	0.0	0.0	0.2	0.1	0.12	0.0	0.0	0.64
≥30 years	Prevalence	≥1 mm	97.0	1.2	99.2	0.4	0.11	97.1	1.2	0.95
		≥2 mm	89.1	1.5	93.5	1.4	0.07	94.9	1.7	0.03
		≥3 mm	68.2	2.7	75.6	3.1	0.07	74.5	2.6	0.19
		≥5 mm	28.4	3.2	38.2	3.7	0.11	36.8	2.0	0.10
	Extent	≥1 mm	55.4	1.7	67.8	1.8	0.0001	64.2	2.8	0.05
		≥2 mm	40.5	1.9	50.8	2.1	0.002	47.7	3.0	0.12
		≥3 mm	20.6	1.4	30.8	2.3	0.005	27.3	2.6	0.10
		≥5 mm	6.0	0.8	11.7	1.5	0.01	9.6	1.6	0.11

*p compared to high socio-economic status

Table 5. Assessment of the risk for having localized or generalized gingival recession ≥ 1 mm in subjects 14-29 years old.

Variables		Localized			Generalized		
		RRR*	C.I.†		RRR*	C.I.†	
Cigarette smoking	Non-smokers	1.0			1.0		
	Light smokers	0.7	0.3	1.5	0.8	0.3	1.6
	Moderate/ heavy smokers	2.0‡	1.2	3.4	3.8§	2.0	7.0
Supragingival calculus	<5%	1.0			1.0		
	5% - 15%	1.3	0.5	3.3	1.0	0.3	2.9
	>15%	2.3‡	1.3	4.2	3.8§	1.7	8.4

*RRR: Relative risk ratio adjusted for age

†C.I.: 95% Confidence interval

‡ p< 0.05; § p< 0.01

Table 6. Assessment of the risk for having localized or generalized gingival recession ≥ 3 mm in subjects 30+ years old.

Variables		Localized			Generalized		
		RRR*	C.I.†		RRR*	C.I.†	
Race	White	1.0			1.0		
	Non-white	2.8§	1.7	4.6	1.2	0.7	2.3
Cigarette smoking	Non-smokers	1.0			1.0		
	Light smokers	1.2	0.6	2.6	1.1	0.4	2.9
	Moderate smokers	1.3	0.7	2.4	1.7	0.9	3.2
	Heavy smokers	1.4	0.7	2.8	3.0§	1.4	6.3
Supragingival calculus	<25%	1.0			1.0		
	25% - 50%	1.4	0.8	2.2	2.2§	1.3	3.8
	>50%	1.6	0.9	2.7	6.4§	3.7	11.0

*RRR: Relative risk ratio is also adjusted for age

†C.I.: 95% Confidence interval

‡ p< 0.05; § p< 0.01

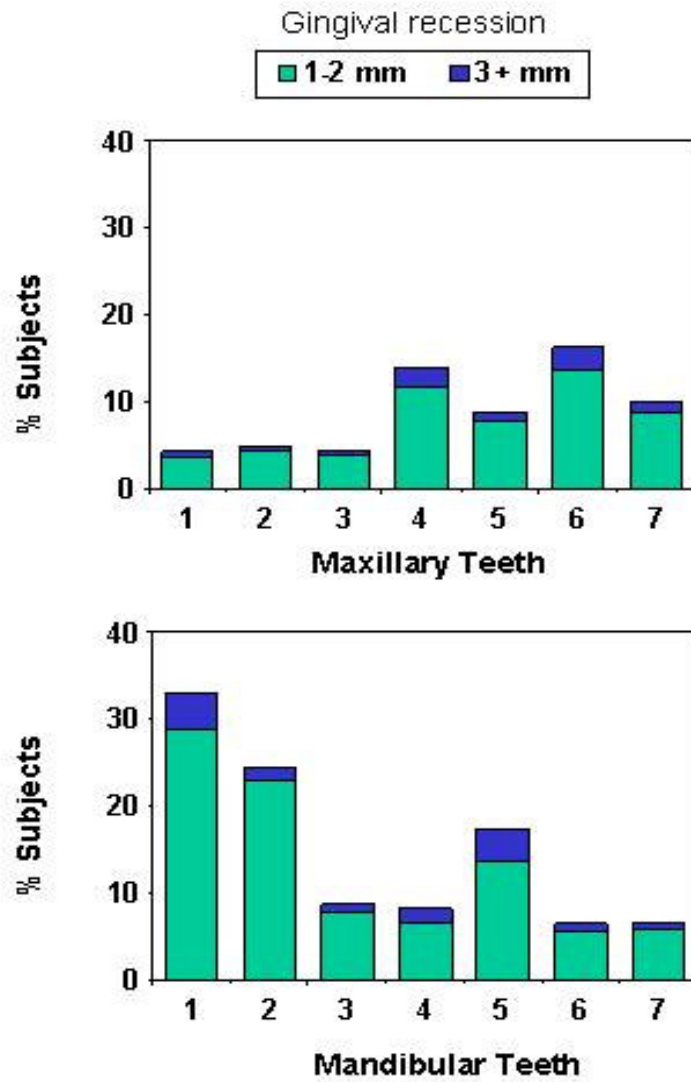


Figure 1.

Percentage of subjects <30 years old with gingival recession, by tooth type and age group. Central incisor: 1, lateral incisor: 2, canine: 3, first premolar: 4, second premolar: 5, first molar: 6, second molar: 7

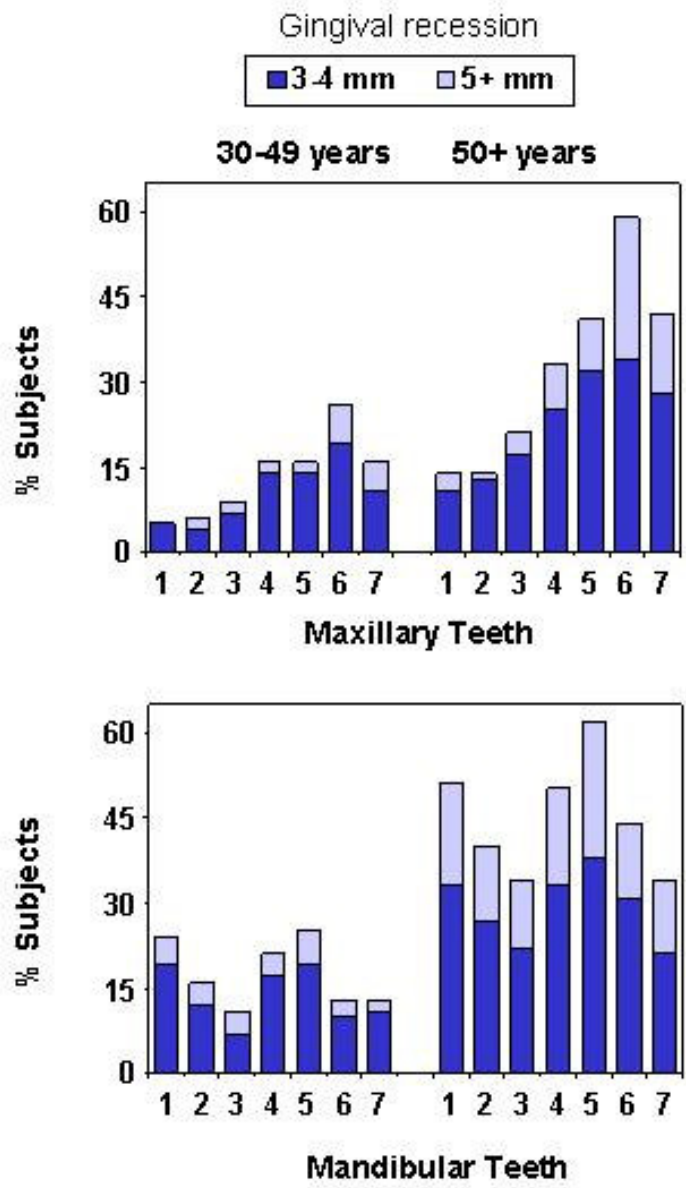


Figure 2.

Percentage of subjects ≥ 30 years old with gingival recession, by tooth type and age group. Central incisor: 1, lateral incisor: 2, canine: 3, first premolar: 4, second premolar: 5, first molar: 6, second molar: 7

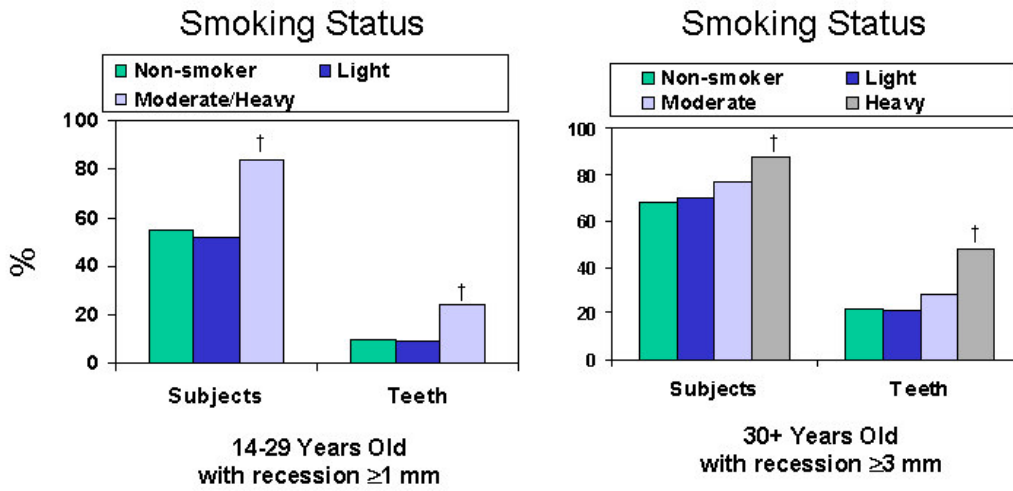


Figure 3. Percentage of subjects, and percentage of teeth per subject with gingival recession by cigarette smoking status. (†: $p < 0.01$, compared to non-smokers)

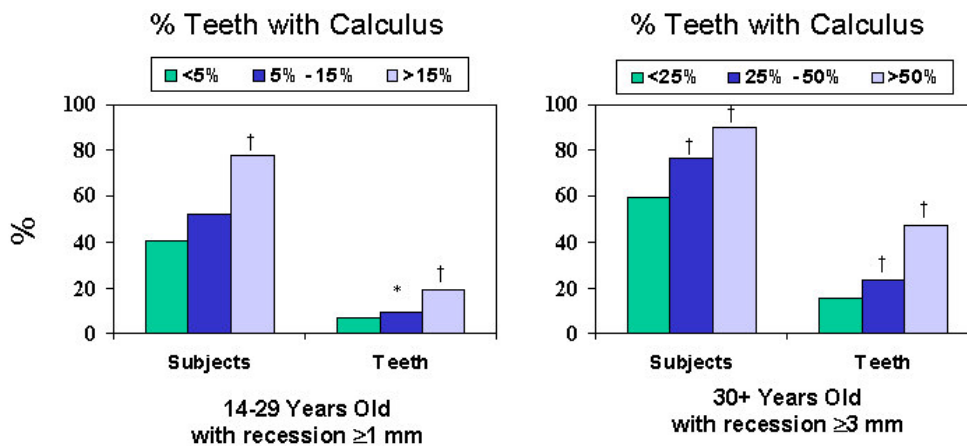


Figure 4. Percentage of subjects, and percentage of teeth per subject with gingival recession by percentage of teeth with supragingival dental calculus. (*: $p < 0.05$, †: $p < 0.01$, compared to the lowest group)

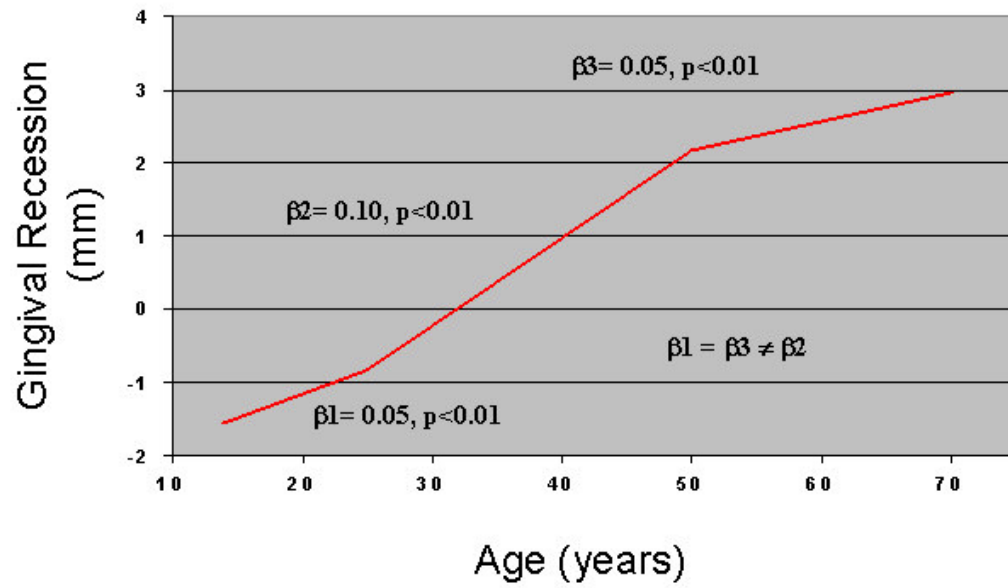


Figure 5. The relationship of mean gingival recession (mm) and age, adjusted for race, cigarette smoking status, and percentage teeth with supragingival dental calculus.

Paper V

Early-Onset Aggressive Periodontitis in an Urban Population in South Brazil

Cristiano Susin^{*†} and Jasim M. Albandar^{*}

Background: There are limited data about the epidemiology and risk factors for early-onset aggressive periodontitis (AgP) in Latin American and Brazilian populations. The aim of this study was to assess the prevalence of AgP and the risk associated with demographic variables, smoking behavior, and other periodontal variables in a young urban population in South Brazil.

Methods: A representative sample of 612 subjects aged 14–29 years were sampled using a multistage probability method. A full-mouth clinical examination of 6 sites per tooth, and an interview were performed in a mobile examination center. Subjects in the age groups 14-19 and 20-29 years were classified with AgP if they had 4 or more teeth with attachment loss ≥ 4 mm or ≥ 5 mm, respectively.

Results: The prevalence of AgP was 5.5% subjects. The disease occurred equally among males and females, but was twice as prevalent among non-whites than whites. In the age groups 20-24 and 25-29 years, the AgP subjects had a significantly higher prevalence of tooth loss (90.2% vs. 40.4% and 86.1% vs. 43.4%, $p < 0.01$) and mean number of missing teeth (2.6 vs. 0.9 and 3.4 vs. 1.5, $p < 0.05$) than subjects without attachment loss. The AgP subjects also had significantly higher percentages of sites with dental plaque ($p < 0.0001$), gingival bleeding ($p < 0.05$), and supragingival calculus ($p < 0.0001$) than normal subjects. The risk for AgP was higher in the 25-29 than 14-19 years old groups (OR=6.2), in the low than middle or high socioeconomic status (OR=4.5), in moderate or heavy smokers than nonsmokers (OR=3.1), and in subjects with $\geq 10\%$ versus $< 10\%$ sites with supragingival calculus (OR=3.6).

Conclusion: Socioeconomic status, smoking, and dental calculus were significant risk indicators of AgP in this population. Suitable periodontal prevention programs implementing these risk indicators may prevent or reduce the prevalence of AgP in this and similar populations. *J Periodontol 2005;76:*

KEY WORDS

Periodontal disease/epidemiology, early-onset periodontitis, periodontal attachment loss, risk factors, smoking, dental calculus.

Aggressive forms of periodontitis in young subjects are characterized by involvement of multiple teeth, a rapidly progressive disease, and may also show characteristic radiographic features.¹⁻⁴ If left untreated the disease may lead to a pronounced tooth loss.⁵

The prevalence of early-onset aggressive periodontitis (AgP) has been reported to vary greatly in different populations.³ Among young subjects in the USA the prevalence of AgP ranged from 0.06% in whites, to 2.6% in African-Americans.⁶ Generally, low prevalence rates (~0.1-0.5%) have been reported among Caucasians in developed countries, whereas much higher frequencies are seen in developing countries.³

Gjermeo et al.⁷ using radiographic examination found a prevalence of AgP of 2.6% in students of low socio-economic status in Brazil. More recently, Tinoco et al.⁸ screened populations in 3 Brazilian cities and reported a prevalence of only 0.3%. Other studies investigated the periodontal health in young subjects in Brazil using the Community Periodontal Index of Treatment Needs (CPITN) methodology,⁹⁻¹¹ and did not assess the

* Periodontal Diagnostics Research Laboratory, Department of Periodontology, Temple University School of Dentistry, Philadelphia, USA

† Faculty of Dentistry, University of Bergen, Bergen, Norway

prevalence of AgP.

It has been suggested that specific genetic^{12,13} and microbiologic profiles^{8,13-15} may be associated with AgP. Demographic characteristics and socio-economic status have also been associated with destructive periodontal disease in young individuals. Few studies have assessed the effect of local plaque-retaining factors in the occurrence of periodontal attachment loss in young age cohorts. It has been shown that dental calculus, caries lesions that are near the gingival margin, and proximal dental fillings, including defective as well as the seemingly non-defective restorations, were associated with a statistically significant increase in risk for alveolar bone loss.¹⁶⁻¹⁸ Comprehensive oral hygiene programs, although effective in controlling the formation of dental plaque and gingival inflammation in adolescents,¹⁹ may not be effective in controlling the initiation and progression of AgP.²⁰

Data on the epidemiology and risk factors for AgP in Latin American and Brazilian populations are scarce.²¹ The aim of the present study was to assess the prevalence of AgP in a young urban population in South Brazil, and to assess the association of demographic variables, smoking behavior, and other periodontal variables with the occurrence of AgP in this population.

MATERIAL AND METHODS

Study design

This cross-sectional survey examined a group of young subjects 14-29 years old who were a subset derived from a larger sample representative of the population of Porto Alegre in the Brazilian state of Rio Grande do Sul.²² This state is located in the southern part of Brazil, neighboring Argentina and Uruguay. The present survey covered 14 major municipalities from the Porto Alegre metropolitan area, with a total population of approximately 3 million.

A representative sample of the target population was derived using a multistage probability sampling method using information provided by Rio Grande do Sul State Government Agency for Metropolitan Affairs (METROPLAN) and the Brazilian Institute of Geography and Statistics (IBGE). Primary sampling units (PSU) were selected randomly from geographic areas that had been stratified by income level. The PSUs were selected with a probability proportional to size and using a sampling frame of these PSUs. Area sectors were then selected randomly within each geographic area, and the number of sectors selected was

proportional to the number of sectors in each area. Households were sampled consecutively within the selected sectors.

Consenting household members who were 14 to 29 years old were included in this study. Exclusion criteria were presence of diseases and conditions that may pose health risks to the participant or examiner, or that may interfere with the clinical examination. Hence, subjects were excluded if they were diagnosed with psychiatric problems, or intoxicated with alcohol or drugs. Individuals requiring a prophylactic regimen of antibiotics were provided with the appropriate medicine before the clinical examination.

Study sample

The study sample included 612 individuals aged 14 – 29 years, and comprised 291 (47.5%) males and 321 (52.5%) females, 507 (82.8%) whites and 105 (17.2%) non-whites. None of the study subjects were completely edentulous.

Examinations

The study subjects had clinical examinations performed in a mobile examination center consisting of a trailer equipped with a complete dental unit, comprising a dental chair, light, and basic amenities. Four periodontists performed the clinical examinations, and the data entry was performed by two dental assistants. Eligible study subjects who consented to participation were interviewed to gather demographic, socio-economic status, oral health and other health-related data using a structured written questionnaire.

All permanent fully erupted teeth, excluding third molars, were examined with a manual periodontal probe[‡] color coded at 1,2,3,5,7,8,9 and 10 mm. Six sites per tooth were assessed in the mesiobuccal, midbuccal, distobuccal, distolingual, midlingual, and mesiolingual sites.

Teeth of each quadrant were dried with a blast of air, and presence of visible dental plaque and supragingival calculus was recorded. Thereafter, gingival bleeding was assessed. The periodontal probe was inserted 1-2 mm into the gingival sulcus starting at one interproximal area and moving to the other. Presence of gingival bleeding was scored after the sites of a single quadrant were probed.

Probing depth was defined as the distance from the free gingival margin to the

[‡] PCP10-SE, Hu-Friedy Mfg. Co. Inc., Chicago, USA

bottom of the pocket/sulcus. Gingival recession was defined as the distance from the cemento-enamel junction (CEJ) to the free gingival margin, and this assessment was assigned a negative sign if the gingival margin was located coronal to the CEJ. Periodontal attachment loss was defined as the distance from the CEJ to the bottom of the pocket/sulcus, and was calculated as the sum of the probing depth and gingival recession measurements. Measurements were made in mm and were rounded to the lower whole mm.

Two classification criteria of AgP were used, depending on the age of the subject. In the 14-19 years group, subjects with 4 or more teeth with attachment loss ≥ 4 mm were defined as having AgP. In the 20-29 years group, cases were defined as those with 4 or more teeth with attachment loss ≥ 5 mm. Since attachment loss at the midbuccal surface of teeth can also be caused by factors not related to periodontal inflammation, attachment loss measurements at midbuccal sites were excluded when classifying the subjects by AgP status.

Ethical considerations

The study protocol was reviewed and approved by the following committees: Research Ethics Committee, Federal University of Rio Grande do Sul, Porto Alegre, Brazil; the National Commission on Ethics in Research, Ministry of Health, Brasília, Brazil; Ethics in Medical Research Committee, University of Bergen, Bergen, Norway. Subjects who agreed to participate signed an informed consent form. At the conclusion of the study the participants were provided with a written report detailing their oral status and any diagnosed mucosal lesions. Patients with diagnosed pathological conditions were advised to seek specialist consultation and treatment.

Measurement reproducibility

The examiners were trained and calibrated in performing the clinical measurements before and during the field examinations. The examination team followed a quality control protocol which was aimed at reducing systematic and random measurement errors and to quantify remaining error. The protocol involved standard examination environment and methodology, standard equipment, and detailed written instructions for performing the clinical procedures.

Assessment of measurement reproducibility used replicate periodontal measurements

performed during the fieldwork. One examiner with the most clinical experience served as the "gold standard" examiner. A total of 57 subjects, divided into four groups ranging from 8 to 20 subjects, were used for the reproducibility assessment. In one of the groups, the replicate measurements consisted of repeated measurements by the gold standard examiner. In each of the remaining 3 groups, the replicate measurements were made by one examiner and the gold standard examiner. Measurement reproducibility at the subject level was assessed by the intraclass correlation coefficient²³ and weighted kappa.²⁴ At the site level, reproducibility was assessed by the weighted kappa. The intraclass correlation coefficients for mean attachment loss ranged between 0.95 and 0.99, and for percentage teeth with attachment loss ≥ 5 mm and ≥ 7 mm ranged between 0.80 and 0.98. The weighted kappas (± 1 mm) of the prevalence of attachment loss ≥ 5 mm were between 0.69 and 1.00, and at site level ranged between 0.65 and 0.87. The intraclass correlation coefficients at the site level ranged between 0.73 and 0.98 for supragingival calculus, and between 0.64 and 0.82 for dental plaque.

Data Analysis

In this study, potential risk indicators were studied by comparing subjects with AgP to those without periodontitis (normal). Individuals who had teeth with only attachment loss ≤ 2 mm, or had only one site with attachment loss 3 mm were considered without periodontitis for the purpose of this study.

Race was scored as White or Non-white. Socio-economic status was scored by combining information about family economy using a standard Brazilian economy classification (CCEB),²⁵ and the level of education of the individual. High socio-economic status was defined as having ≥ 9 years of education and being in the upper two tertiles of the CCEB economy classification, or having 5-8 years of education and being in the highest tertile of the CCEB classification. Low socio-economic status was defined as having 1-4 years of education, and being in the lowest two tertiles of the CCEB classification, or having 5-8 years of education and being in the lowest tertile of the CCEB classification. Individuals who had higher economy and education than the low socioeconomic group, but less than the high group were classified as having middle socioeconomic status. The percentage of sites per person with visible dental plaque, gingival

bleeding, or supragingival calculus were calculated by dividing the number of sites with each of these variables, by the total number of sites within the subject.

The study subjects were classified according to the self-reported pattern of dental visits during the last 5 years. Individuals who had visited a dentist on a regular basis for maintenance care were classified as having regular dental visits. Subjects who during the last 5 years had visited a dentist only for emergency dental treatment, or had not visited a dentist were classified in the irregular dental visits group. The questionnaire also assessed the declared frequency of use of interdental oral hygiene devices. Regular interdental hygiene was defined as regular use of toothpick, dental floss, or other similar tools at least once a day. Irregular interdental hygiene was defined as use of interdental oral hygiene devices more than once a week, but less often than once a day. Non-users were subjects who did not perform interdental hygiene, or who used the device less often than once a week. Most participants in this study claimed using a toothbrush regularly at least once a day, and this information was therefore not used in the present analysis.

Individuals were classified by their smoking habit into 3 groups: non-smokers (<1 pack of cigarettes in a lifetime), light (1 – 912 packs) and moderate/heavy smokers (>912 packs). Individuals were classified according to the presence of supragingival calculus into two groups, <10% and ≥10% sites with calculus.

Data analysis was performed by STATA software[§] and using survey commands that take into account survey design including stratification, clustering, and weighting and robust variance estimation. A weight variable was used to adjust for the probability of selection and deviations in the sample distributions from the target population distribution by age, gender and education.^{26,27} The chosen level of statistical significance was 5%, and the 95% confidence intervals (CI) were calculated.

Univariable and multivariable analyses were used to compare the percentage of sites with dental plaque, gingival bleeding and supragingival calculus in normal and AgP subjects. Pairwise comparisons of unadjusted estimates were carried out using the Wald test.²⁷ Further comparisons were done using linear regression analysis, adjusting for age, socio-economic status and smoking. The variables

dental plaque, gingival bleeding and supragingival calculus were not normally distributed, and to achieve normality these variables were transformed using a square root arc-sine function.

Logistic regression analysis was used to model the relationship between AgP and various risk indicators. Preliminary analyses were performed using univariable models. Next, a multivariable model was performed and only exposures showing in the univariable analyses associations with $p \leq 0.25$ were included.²⁸ Confounding and interaction effects were assessed. The multivariable analyses were performed in two stages. Demographic, socio-economic, and behavioral variables were entered first in the model, and supragingival calculus was entered next. Gender, race, frequency of interdental hygiene, pattern of dental visits and place of residency (urban vs. suburban) did not show significant associations with AgP, and these variables were therefore removed from the final model.

RESULTS

Overall, 28 subjects (5.5%) were diagnosed with AgP. These subjects had, on average, 47.6%, 28.5% and 13.6% of their teeth showing attachment loss of ≥4 mm, ≥5 mm, and ≥6 mm, respectively. The prevalence of AgP increased significantly with age (Table 1). The percentage of teeth affected with attachment loss showed a similar trend of increase with age (Fig. 1).

AgP showed similar prevalence rates in males and females, and was twice as prevalent among non-whites than in white, although the difference was not statistically significant (Table 1). AgP was significantly more prevalent in the low than the middle/high socio-economic status groups ($p < 0.05$), and among heavy smokers than non-smokers ($p < 0.01$).

The AgP subjects had a significantly higher prevalence of tooth loss (75.5% vs. 31.7%, $p < 0.001$) and a higher mean number of missing teeth (2.8 vs. 0.8, $p < 0.01$) than normal subjects. Further analysis by age showed a marked difference in tooth loss between AgP and normal groups in the 20-24 and 25-29 years ($p < 0.05$), but not in the younger age group (Table 2).

Subjects with AgP had significantly higher percentages of sites with dental plaque ($p < 0.0001$), and supragingival calculus ($p < 0.0001$) than normal subjects (Table 3). We used multivariable analysis to investigate whether these differences remained after

[§] Stata 7.0, Stata Corporation, College Station, TX, USA

adjusting for age, socio-economic status, and smoking behavior. The results showed that the AgP group had significantly higher adjusted estimates of dental plaque, gingival bleeding and supragingival calculus, separately, than normal subjects (Table 3).

The risk for AgP was significantly higher in subjects aged 25-29 years than 14-19 years (OR=6.2), having low than middle or high socio-economic status (OR=4.5), moderate or heavy smokers than nonsmokers (OR=3.1), and in subjects with $\geq 10\%$ versus $< 10\%$ sites with supragingival calculus (OR=3.6) (Table 4).

DISCUSSION

The results showed a relatively high prevalence (5.5%) of AgP in this Brazilian population, with 2.5%, 4.3%, and 9.9% of 14-19, 20-24, and 25-29 years olds, respectively diagnosed with the disease. Subjects with AgP showed attachment loss on multiple teeth, and had high percentages of sites with dental plaque, gingival bleeding and supragingival calculus. In addition, in the age group 20-29 years, AgP subjects had more than twice the number of missing teeth, compared to age-matched subjects without periodontitis.

Different studies have used different examination and screening methods and inconsistent classification criteria of AgP.³ Tinoco et al.⁸ used a 2-stage strategy whereby they first used interdental wooden sticks for the initial screening of subjects, and subjects having probing depth ≥ 5 mm at the proximal surfaces of first molars were identified. The latter group then underwent a comprehensive clinical and radiographic examination of all teeth. Using this method, Tinoco et al.⁸ reported that the prevalence of "localized juvenile periodontitis" among 12 to 19 years olds from low socio-economic status populations in 3 cities in Brazil ranged between 0.1% and 1.1%, and the mean prevalence for all 3 populations was 0.3%. However, in the present study we used a full-mouth clinical examination method which included attachment loss measurements at 6 sites of all permanent teeth. The corresponding prevalence of AgP in the age group 14-19 years in the present population was 2.5% subjects.

Gjermeo et al.⁷ used a radiographic screening method using 2 bitewing radiographs and reported that the prevalence of AgP in 15 years old students of a low socio-economic area in Belo Horizonte, Brazil was 2.6%. Using a similar radiographic screening method, Albandar et al.²⁹ reported that 1.3% had AgP, among a population of 13 years old schoolchildren of high

socio-economic status in São Paulo, Brazil. It should be noted, however, that the latter 2 studies used convenience study samples. Other studies in young Brazilian population have used the CPITN methodology.^{9-11,30,31}

Only a few large-scale surveys of AgP have been conducted globally. A national survey in the USA reported that the prevalence of AgP was 0.4% in 13-15 years old, and 0.8% in 16-17 years old students.⁶ Representative samples of schoolchildren aged 14-19 years in Amsterdam, Netherlands,³² Oslo, Norway³³ and Santiago, Chile³⁴ showed low prevalences of AgP ranging between 0% and 0.3%.

It should be noted that classification criteria have a significant impact on the reported prevalence of disease.^{1,3} In addition, the use of partial recording protocols in some studies may underestimate the true prevalence of AgP, particularly in populations with low occurrence of disease.³⁵ Subjects with aggressive periodontitis show rapid progression of attachment loss over time.³⁶ We, therefore, used a more stringent attachment loss threshold in the older age group to increase the specificity of the diagnostic criteria of AgP.

Tooth loss is a frequent occurrence among individuals with AgP⁵ and it might have an impact on the prevalence of the disease. In the present study, individuals with aggressive periodontitis had a higher occurrence of tooth loss than non-cases. In the whole sample, 36 subjects had more than 4 missing teeth, and 4 of these were diagnosed with aggressive periodontitis. Most subjects showing significant tooth loss were in the 25-29 years age group, nevertheless this age group also showed the highest prevalence of aggressive periodontitis. No reliable data were available on the reasons for extraction of teeth in these subjects.

More recently the International Workshop on Classification of Periodontal Diseases recommended the new disease classifications: chronic periodontitis, aggressive periodontitis, and periodontitis as a manifestation of systemic diseases in young subjects.⁴ It was suggested that age should not be used as a criterion in the classification of these diseases. However, data supporting this view are lacking. A position paper by the American Academy of Periodontology acknowledged that aggressive periodontitis may occur mainly in children and adolescents.²

There is a considerable deficiency in present knowledge of the pathogenesis and risk factors of AgP in young subjects. There is

evidence that AgP commences during adolescence or early adulthood.^{6,37,38} In addition, the present study showed a trend towards an increase in the prevalence of AgP in the age groups 20-24 and 25-29 compared to 14-19 years. In the multivariable model, the correlation of increase prevalence with age remained statistically significant after controlling for a number of other pertinent exposures.

In this population, gender did not play a significant role as a risk indicator for AgP. There has been some inconsistency regarding the role of gender as a risk indicator for AgP, with some studies showing significant, though contradictory effects,³⁸⁻⁴⁰ whereas others not showing significant effects.^{6, 37} The percentage of subjects with AgP was twice as high among non-whites than whites. However, the difference was not statistically significant, and the effect did not hold in the multivariable analytical model. In the U.S. population, Albandar et al.⁶ and Loe and Brown³⁸ used national data and reported significantly higher prevalence of AgP among African-Americans and Hispanics than in whites. The latter studies standardized the data by a few demographic variables, but did not investigate the effect of race using elaborate multivariable analytical models.

Low socio-economic status showed a significant association with AgP in the present population. This is in agreement with other studies.^{33,34,41} Furthermore, epidemiological studies show a higher prevalence of this disease in developing countries³ and among deprived groups within various populations.^{33,38} This underscores the importance of environmental factors in the pathogenesis of AgP.

The effect of cigarette smoking on chronic periodontitis in adults is well documented, and there is overwhelming evidence showing that smoking is an important risk factor for this disease. However, the role of smoking as a risk factor for early-onset aggressive periodontitis is still unclear. To investigate this effect we used a multivariable model that adjusted for several other variables, including supragingival calculus. This model showed that smoking was a significant risk indicator for having AgP in this young population.

Findings of a number of studies suggest a higher level of attachment loss among young subjects who are smokers than nonsmokers.⁴²⁻⁴⁵ Scheinlein et al.³⁷ observed that among individuals with generalized AgP, smokers had a higher severity of attachment loss compared to

nonsmokers. Mullally et al.⁴⁶ in a group of patients with AgP, found that smokers had greater radiographic bone loss than nonsmokers. However, one study did not find a significant association between smoking and attachment loss in young subjects.⁴¹

Baer⁴⁰ had suggested that "juvenile periodontitis" is not associated with significant amounts of plaque and calculus. This was not corroborated in the present study, which found that young subjects with AgP had significantly higher percentages of sites showing dental plaque and supragingival calculus, and a higher extent of gingival inflammation than subjects without periodontitis. Furthermore, a model that adjusted for the variance in age, gender, race, socio-economic status, and smoking behavior, showed that subjects with 10% or more sites with calculus had 3.6 times higher chance of having AgP than those with fewer sites with calculus. Albandar³⁹ found no significant differences between AgP and non-AgP children with respect to gingival inflammation, dental plaque, or calculus deposits on teeth. In a national survey in the United States, higher percentage of sites with gingival bleeding and subgingival calculus were found in individuals with AgP.⁶ Albandar et al.¹⁷ showed that presence of gingival inflammation at baseline was associated with a significant subsequent periodontal breakdown in these subjects. Other follow-up studies also showed that dental plaque,^{42,47,48} supragingival calculus^{47,48} and gingival inflammation^{42,47-49} were associated with future attachment loss.

This study showed a relatively high prevalence of AgP in this Brazilian population of adolescents and young adults. Individuals with AgP had significantly higher levels of dental plaque, gingival bleeding and supragingival calculus. Risk indicators for AgP were socio-economic status, smoking and supragingival calculus. Suitable periodontal prevention programs implementing these risk indicators may prevent or reduce the prevalence of AgP in this and similar populations.

ACKNOWLEDGMENT

Funding for this project was provided by Foundation for Post-Graduate Education (CAPES), Ministry of Education, Brazilia, DF, Brazil. Grant number 1614/99-1.

REFERENCES

1. Albandar JM, Brown LJ, Genco RJ, Loe H. Clinical classification of periodontitis in

- adolescents and young adults. *J Periodontol* 1997;68:545-555.
2. Califano JV. Position paper: periodontal diseases of children and adolescents. *J Periodontol* 2003;74:1696-1704.
 3. Albandar JM, Tinoco EM. Global epidemiology of periodontal diseases in children and young persons. *Periodontol* 2000 2002;29:153-176.
 4. Armitage GC. Development of a classification system for periodontal diseases and conditions. *Ann Periodontol* 1999;4:1-6.
 5. Albandar JM, Brown LJ, Loe H. Dental caries and tooth loss in adolescents with early-onset periodontitis. *J Periodontol* 1996;67:960-967.
 6. Albandar JM, Brown LJ, Loe H. Clinical features of early-onset periodontitis. *J Am Dent Assoc* 1997;128:1393-1399.
 7. Gjermeo 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;11:104-113.
 8. Tinoco EM, Beldi MI, Loureiro CA, et al. Localized juvenile periodontitis and *Actinobacillus actinomycetemcomitans* in a Brazilian population. *Eur J Oral Sci* 1997;105:9-14.
 9. Flores-de-Jacoby L, Bruchmann S, Mengel R, Zafiroopoulos GG. Periodontal conditions in Rio de Janeiro City (Brazil) using the CPITN. *Community Dent Oral Epidemiol* 1991;19:127-128.
 10. Dini EL, Guimaraes LO. Periodontal conditions and treatment needs (CPITN) in a worker population in Araraquara, SP, Brazil. *Int Dent J* 1994;44:309-311.
 11. Dini EL, Foschini AL, Brandao IM. Periodontal conditions in a 7-19-year-old student population in Araraquara, Sao Paulo, Brazil, 1995. *Cad Saude Publica* 1997;13:321-324.
 12. Kinane DF, Hart TC. Genes and gene polymorphisms associated with periodontal disease. *Crit Rev Oral Biol Med* 2003;14:430-449.
 13. Albandar JM, Rams TE. Risk factors for periodontitis in children and young persons. *Periodontol* 2000 2002;29:207-222.
 14. Albandar JM, Brown LJ, Loe H. Putative periodontal pathogens in subgingival plaque of young adults with and without early-onset periodontitis. *J Periodontol* 1997;68:973-981.
 15. Trevilatto PC, Tramontina VA, Machado MA, Goncalves RB, Sallum AW, Line SR. Clinical, genetic and microbiological findings in a Brazilian family with aggressive periodontitis. *J Clin Periodontol* 2002;29:233-239.
 16. Albandar JM, Buischi YA, Axelsson P. Caries lesions and dental restorations as predisposing factors in the progression of periodontal diseases in adolescents. A 3-year longitudinal study. *J Periodontol* 1995;66:249-254.
 17. Albandar JM, Brown LJ, Brunelle JA, Loe H. Gingival state and dental calculus in early-onset periodontitis. *J Periodontol* 1996;67:953-959.
 18. Albandar JM, Kingman A, Brown LJ, Loe H. Gingival inflammation and subgingival calculus as determinants of disease progression in early-onset periodontitis. *J Clin Periodontol* 1998;25:231-237.
 19. Albandar JM, Buischi YA, Mayer MP, Axelsson P. Long-term effect of two preventive programs on the incidence of plaque and gingivitis in adolescents. *J Periodontol* 1994;65:605-610.
 20. Albandar JM, Buischi YA, Oliveira LB, Axelsson P. Lack of effect of oral hygiene training on periodontal disease progression over 3 years in adolescents. *J Periodontol* 1995;66:255-260.
 21. Gjermeo P, Rosing CK, Susin C, Oppermann R. Periodontal diseases in Central and South America. *Periodontol* 2000 2002;29:70-78.
 22. Susin C, Dalla Vecchia CF, Oppermann RV, Haugejorden O, Albandar JM. Periodontal attachment loss in an urban population of Brazilian adults: the effect of demographic, behavioral and environmental risk indicators. *J Periodontol* 2004;75:1033-1041.
 23. Shrout P, Fleiss J. Intraclass correlations: uses in assessing rater reliability. *Psychol Bull* 1979;86:420-428.
 24. Hubert L. Kappa revisited. *Psychol Bull* 1977;84:289-297.
 25. National Association for Opinion and Marketing Research. Economic Classification Criterion - Brazill. Sao Paulo:National Association for Opinion and Marketing Research; 1997.
 26. Brazilian Institute of Geography and Statistics. Population Census (in Portuguese). Rio de Janeiro:Brazilian Institute of Geography and Statistics; 1996.
 27. Korn E, Graubard B. Analysis of health surveys. New York: John Wiley & Sons, Inc; 1999.

28. Hosmer D, Lemeshow S. Applied logistic regression. 2nd ed. New York: Jhon Wiley & Sons; 2000: 260-287.
29. Albandar JM, Buischi YA, Barbosa MF. Destructive forms of periodontal disease in adolescents. A 3-year longitudinal study. *J Periodontol* 1991;62:370-376.
30. Ministério da Saúde. Levantamento epidemiológico em saúde bucal: Brasil, zona urbana, 1986 (in Portuguese):Ministério da Saúde; 1988.
31. Campos Jr A, Passanezi E, Kim SH, Navarro MFL, Souza SLS. Identification of risk groups to periodontal disease in a population from Bauru city, Sao Paulo. *Rev Fac Odontol Bauru* 1994;2:20-28.
32. Van der Velden U, Abbas F, Van Steenberghe TJ, et al. Prevalence of periodontal breakdown in adolescents and presence of *Actinobacillus actinomycetemcomitans* in subjects with attachment loss. *J Periodontol* 1989;60:604-610.
33. Aass AM, Albandar J, Aasenden R, Tollefsen T, Gjermo P. Variation in prevalence of radiographic alveolar bone loss in subgroups of 14-year-old schoolchildren in Oslo. *J Clin Periodontol* 1988;15:130-133.
34. Lopez NJ, Rios V, Pareja MA, Fernandez O. Prevalence of juvenile periodontitis in Chile. *J Clin Periodontol* 1991;18:529-533.
35. Kingman A, Albandar JM. Methodological aspects of epidemiological studies of periodontal diseases. *Periodontol* 2000 2002;29:11-30.
36. Brown LJ, Albandar JM, Brunelle JA, Loe H. Early-onset periodontitis: progression of attachment loss during 6 years. *J Periodontol* 1996;67:968-975.
37. Schenkein HA, Gunsolley JC, Koertge TE, Schenkein JG, Tew JG. Smoking and its effects on early-onset periodontitis. *J Am Dent Assoc* 1995;126:1107-1113.
38. Loe H, Brown LJ. Early onset periodontitis in the United States of America. *J Periodontol* 1991;62:608-616.
39. Albandar JM. Juvenile periodontitis--pattern of progression and relationship to clinical periodontal parameters. *Community Dent Oral Epidemiol* 1993;21:185-189.
40. Baer PN. The case for periodontosis as a clinical entity. *J Periodontol* 1971;42:516-520.
41. Lopez R, Fernandez O, Jara G, Baelum V. Epidemiology of clinical attachment loss in adolescents. *J Periodontol* 2001;72:1666-1674.
42. Hashim R, Thomson WM, Pack AR. Smoking in adolescence as a predictor of early loss of periodontal attachment. *Community Dent Oral Epidemiol* 2001;29:130-135.
43. Kamma JJ, Nakou M, Baehni PC. Clinical and microbiological characteristics of smokers with early onset periodontitis. *J Periodontol Res* 1999;34:25-33.
44. Linden GJ, Mullally BH. Cigarette smoking and periodontal destruction in young adults. *J Periodontol* 1994;65:718-723.
45. Machuca G, Rosales I, Lacalle JR, Machuca C, Bullon P. Effect of cigarette smoking on periodontal status of healthy young adults. *J Periodontol* 2000;71:73-78.
46. Mullally BH, Breen B, Linden GJ. Smoking and patterns of bone loss in early-onset periodontitis. *J Periodontol* 1999;70:394-401.
47. Suda R, Cao C, Hasegawa K, Yang S, Sasa R, Suzuki M. 2-year observation of attachment loss in a rural Chinese population. *J Periodontol* 2000;71:1067-1072.
48. Clerehugh V, Worthington HV, Lennon MA, Chandler R. Site progression of loss of attachment over 5 years in 14- to 19-year-old adolescents. *J Clin Periodontol* 1995;22:15-21.
49. Griffiths GS, Duffy S, Eaton KA, Gilthorpe MS, Johnson NW. Prevalence and extent of lifetime cumulative attachment loss (LCAL) at different thresholds and associations with clinical variables: changes in a population of young male military recruits over 3 years. *J Clin Periodontol* 2001;28:961-969.

Correspondence:

Dr. Jasim M. Albandar, Department of Periodontology, Temple University School of Dentistry, 3223 North Broad St., Philadelphia, PA 19140, USA. Fax: 1-215-707-7616; email: Jasim.Albandar@temple.edu

Accepted for publication July 13, 2004.

Table 1.
Percentage of subjects with early-onset aggressive periodontitis by demographic, socio-economic status and behavioral variables.

Variable	%	S.E.	p
Age			
14 to 19	2.5	1.1	
20 to 24	4.3	2.0	0.47
25 to 29	9.9	2.5	0.03
Gender			
Male	5.7	1.7	
Female	5.3	1.0	0.80
Race			
White	2.4	1.5	
Non-white	6.1	1.3	0.12
Socio-economic status			
Low	9.4	2.6	
Middle/high	2.8	0.7	0.04
Smoking			
Nonsmokers	3.1	0.9	
Light	3.2	2.3	0.98
Heavy	14.1	3.0	0.001

Table 2.
Percentage of subjects and mean tooth loss in young subjects with early-onset aggressive periodontitis and normal subjects (without periodontitis), by age group.

	Age group (years)	Normal subjects		Aggressive periodontitis		p
		Estimate	S.E.	Estimate	S.E.	
% subjects	14 - 19	22.5	2.8	9.7	10.5	0.22
	20 - 24	40.4	6.0	90.2	8.9	0.003
	25 - 29	43.4	10.0	86.1	8.6	0.001
	Total	31.7	1.3	75.5	8.9	0.001
Mean tooth loss	14 - 19	0.5	0.1	0.6	0.6	0.86
	20 - 24	0.9	0.1	2.6	0.3	0.0002
	25 - 29	1.5	0.7	3.4	0.9	0.04
	Total	0.8	0.1	2.8	0.6	0.007

Table 3.
Comparison of the aggressive periodontitis and normal subjects (without periodontitis), by percentage of sites per subject with dental plaque, gingival bleeding, or calculus.

Variable	Normal subjects		Aggressive periodontitis		p
	Mean	S.E.	Mean	S.E.	
Crude estimates					
Dental plaque	50.0	2.0	72.3	3.7	0.0003
Gingival bleeding	25.4	1.7	36.8	4.3	0.06
Supragingival calculus	11.1	0.6	38.1	4.1	0.0001
Adjusted estimates*					
Dental plaque	48.4	1.2	66.1	4.6	0.0001
Gingival bleeding	23.1	0.9	34.9	3.5	0.0001
Supragingival calculus	8.2	0.9	25.3	3.5	0.0001

* Adjusted for age, socio-economic status and smoking

Table 4.
Multivariate analysis of the association of demographic, socio-economic, behavioral and local variables with the occurrence of aggressive periodontitis.

Variable		n	Odds Ratio	95% CI	
Age	14 – 19	217	1.0		
	20 – 24	110	1.2	0.3	4.4
	25 – 29	65	6.2†	2.1	17.8
Socio-economic status	High/middle	298	1.0		
	Low	94	4.5†	1.8	11.2
Smoking	Nonsmokers	266	1.0		
	Light	74	0.6	0.1	2.4
	Moderate/heavy	52	3.1*	1.2	8.3
Supragingival Calculus	<10%	223	1.0		
	≥ 10%	169	3.6*	1.3	10.1

* p< 0.05; †p< 0.01

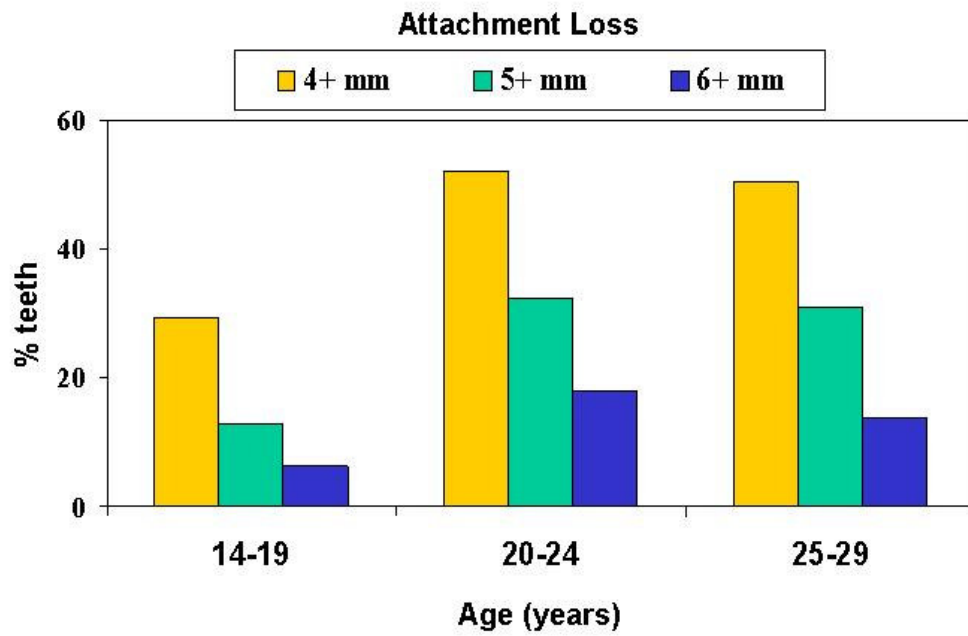


Figure 1.

The relationship between percentage of teeth with attachment loss ≥ 4 mm, ≥ 5 mm, and ≥ 6 mm, and age in subjects with early-onset aggressive periodontitis.

Periodontal Diseases in a Representative Urban Population in Southern Brazil

Concluding remarks:

- Moderate and severe clinical attachment loss is common among adults in this population.
- A high percentage of the population has moderate and severe gingival recession.
- Aggressive periodontitis is prevalent among adolescents and young adults, and is more prevalent than in most other populations.
- Partial recording consistently underestimates the prevalence of attachment loss, and the extent of underestimation is dependent on the type of system used and the characteristics of the population surveyed.
- Important risk indicators for destructive periodontal diseases include older age, lower socio-economic status, presence of dental calculus, and smoking behavior.
- Smoking cessation should be considered for inclusion in all programs designed to prevent or control periodontal diseases.

Cristiano Susin

DDS 1996 Federal University of Rio Grande do Sul
MSc 1999 Lutheran University of Brazil

ISBN 82-8006-018-9