Curr Oral Health Rep (2014) 1:286–294
DOI 10.1007/s40496-014-0034-7

EPIDEMIOLOGY (M LAINE, SECTION EDITOR)

Natural History of Periodontitis

Christoph A. Ramseier · Ho-Yan Duong · Eric Schmid

Published online: 23 September 2014

Abstract Prospective studies have been conducted around the world to investigate the natural progression of periodontitis. Eight populations, with at least five years of observation, have been investigated in Norway, Sri Lanka, China, Indonesia, Australia, Germany, New Zealand, and Brazil. The findings from each population were based on the assessments of clinical periodontal parameters, microbiological samplings, socioeconomic status, tobacco use, and daily oral hygiene. Periodontitis was not commonly seen in the younger cohorts. With increasing age, however, the prevalence and severity of periodontitis rose significantly, although there was substantial variance within this general finding among individual cohorts. While the accumulation of plaque and subgingival calculus appears to be primarily accountable for the onset and progression of periodontitis, it is also necessary to consider the individual's susceptibility and their response to the exposure of common risk factors such as smoking, as well as their socioeconomic status, in order to understand the natural progression of the disease.

Keywords Periodontal disease · Periodontitis · Natural periodontal disease progression · Longitudinal studies

Introduction

Periodontitis is an irreversible inflammatory state of the supporting structures of the teeth. After its onset, the disease progresses, with loss of collagen fibers and attachment to the root surface, apical migration of the pocket epithelium, formation of deepened periodontal pockets, and the resorption of alveolar bone. If left untreated, the disease continues with progressive alveolar bone destruction, leading to increased tooth mobility and subsequent tooth loss.

The geographical distribution of periodontal disease is worldwide . According to recent epidemiological studies, the overall prevalence of periodontitis is estimated in the range of 47 % in adults aged 30 years or older [1]. Variations in host response to the presence of supra- and subgingival microbial deposits, however, undoubtedly influence the destructive process of periodontal tissues over time, although the basic pathogenetic nature of periodontal disease has been described as multifactorial [2]. According to Socransky and colleagues (1984), data from longitudinal monitoring of periodontal attachment levels and alveolar bone suggest that periodontitis advances by recurrent acute episodes, so-called bursts of activity [3]. These bursts occur for short periods of time randomly at periodontal sites throughout the dentition. As such, Socransky et al. proposed an extension of the continuous disease model in which bursts of destructive periodontal disease periods occur with a higher frequency during certain periods of an individual's life [3]. Moreover, more recent evidence includes a consensus that the individual's susceptibility or genetic background may contribute to the high variability of periodontal disease progression observed in various populations worldwide [4].

Whether the association between periodontitis and age is a result of the aging process per se, or also represents the destruction taking place in response to an increased length of exposure to etiological factors, can be explored only in longitudinal investigations. Consequently, studies following human cohorts over a number of decades may provide better insight into the characteristics of disease progression.

Clinical Data Collection

In addition to collecting demographic and socioeconomic data as well as dental findings, all cohort studies to determine the natural progression of periodontitis included the accurate

C. A. Ramseier (✉) · H.-Y. Duong · E. Schmid
Department of Periodontology, School of Dental Medicine, University of Bern, Freiburgstrasse 7, 3012 Bern, Switzerland
e-mail: christoph.ramseier@zmk.unibe.ch

measurement of periodontal probing depths and clinical attachment loss. To validate these parameters, inter-examiner and intra-examiner reproducibility was tested in individual studies.

For example, in two cohorts in Norway and Sri Lanka, this validation was conducted regularly in both the baseline examination and the follow-up examinations. In 1969, a total of 29 subjects in the Norwegian cohort were selected at random and re-examined the next day. In Sri Lanka, the reproducibility of data collection was validated in 1970 on a total of 35 subjects [5••]. In 1978 in Indonesia, every tenth subject in the baseline examination was re-examined. For the follow-up examinations in 1994 and 2002, a total of 18 subjects of this cohort were selected at random for a second examination [6]. To validate reproducibility in a cohort in China, the same subject was re-examined by the same clinician [7•]. Another method was selected for the New Zealand cohort. To save time, not all of the measurements of selected subjects were repeated. However, the second examination was conducted at 672 selected periodontal sites [8].

The Cohorts

Around 40 years ago, the two cohorts selected in Norway and Sri Lanka could not have been more different with regard to oral hygiene habits and basic dental care [5••]. However, since an accurate assessment of the natural progression of periodontitis could not be determined solely on the basis of these two extremes, no efforts have been spared in recent decades to improve the available global data. Further large longitudinal studies have been conducted with cohorts in China, Indonesia, Australia, Germany, New Zealand, and Brazil with the goal of documenting the progression of periodontitis (Fig. 1). The cohorts of the selected longitudinal studies are described in more detail below.

Norway

The Norwegian cohort was first studied in 1969 by Harald Löe and colleagues [5••]. All subjects were born and grew up in Oslo [9]. At the start of the study in 1969, 565 subjects were recruited, all of them male. The subjects included several age groups. The oldest group included subjects born between 1934 and 1939. The subjects born in 1940, 1942, 1944, 1946, and 1948 were added to the cohort through cooperation with the Central Bureau of Statistics. The youngest subjects were added by the municipal school board from three high schools in Oslo. None of the subjects were students of medicine or dentistry. All subjects were from the middle class and were well-educated. After World War I, the city of Oslo introduced a comprehensive and continuously adapted and improved oral health/disease prevention program. Beginning in 1936, all children were examined on the basis of an annual recall. After 1946, every child who needed it was given comprehensive dental care, with periodontal, restorative, endodontic, oral surgery, or orthodontic treatment. Additional programs were added over the years to include the population aged 3 to 23 years [9]. Until 1995, this was the only documented cohort to have been integrated into a systematic dental care program for individuals up to the age of 60. According to surveys, all subjects went to the dentist at least once a year, had a toothbrush, and brushed their teeth daily [10].

Attachment Loss

For this study, the periodontal development of every individual was examined and documented at regular intervals over a period of several decades (Table 1). The examinations took place in 1969, 1971, 1973, 1975, 1981, 1988, and 1995. The ages of the subjects with good dental care ranged from 16 to 60. It was observed that 5 % of 16-year-old Norwegians already had initial periodontal attachment loss of at least 2 mm at one or more sites. The frequency of these sites increased with age. At age 32, every subject in this age group already had initial attachment loss of 2 mm or more at at least one site. At age 40, the buccal sites in particular had an attachment loss in the form of gingival recession of at least 2 mm. At age 50, the majority of the sites had a periodontal lesion in the form of pocket formation or a combination of pocket formation and gingival recession. At age 60, the situation was even more advanced. The 60-year-olds had attachment loss at 50 % of all possible sites. It was also found that the molars and premolars were the most affected, regardless of age [11].

In summary, although the subjects practiced good oral hygiene and had regular dental checkups, the incidence of periodontal destruction still increased with age. The highest rate of destruction was in individuals between the ages of 50 and 60. At age 40, the predominant periodontal lesion was in the form of gingival recession. This changed between age 50 and 60 to primarily lesions with periodontal pockets [11].

Tooth Loss

At the baseline examination in 1969, 17-year-old Norwegians had an average of 27.4 of 28 teeth examined. The cohort of 30-year-olds presented with an average of 27.2 teeth. Hardly any teeth were lost between the ages of 17 and 30. Additionally, in this cohort, a number of teeth had been extracted before age 17 compared with the period between ages 17 and 40. Judging from the type of teeth, they were probably extracted for orthodontic reasons [12].

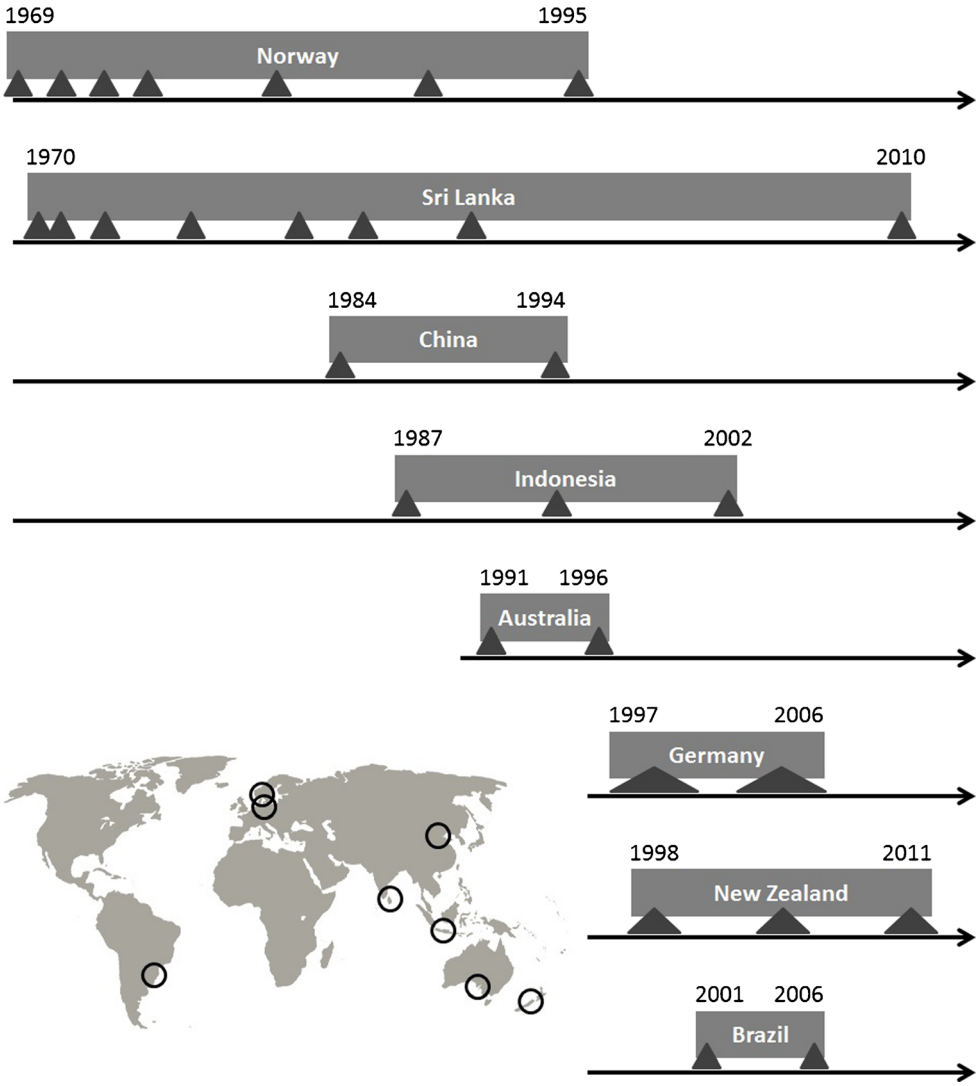

Fig. 1 Multiple longitudinal cohort studies around the globe to investigate the natural progression of periodontitis (1969–2011) (*triangles* indicate each examination per cohort)

Cigarette Smoking

A total of 119 non-smokers and 17 smokers were compared. In the baseline examination, smokers and non-smokers had similar attachment losses of 0.14 mm. With increasing age, especially around age 50, the rate of attachment loss increased significantly in smokers (2.31 mm) compared to non-smokers (1.57 mm).

Linear regression analysis further revealed that aging and moderate smoking correlated independently and significantly with attachment loss. Consequently, lifelong moderate smoking may be considered an independent risk factor for periodontal disease progression [13].

Sri Lanka

The longitudinal cohort in Sri Lanka was recruited in 1970, one year after the Norwegian cohort [5••]. The all-male subjects from Sri Lanka lived and worked on the Dunsinane, Harrow, and Sheen tea estates located in the central highlands of Sri Lanka, about 50 miles from Kandy.

The cohort of the tea plantation laborers in Sri Lanka originally consisted of 480 subjects, who were between the ages of 15 and 30 at the baseline examination. Their overall medical condition was generally satisfactory. This applied to their nutritional condition as well. Chewing betel nuts was a widespread habit.

The workers had never in their lives been included in a dental prevention program. Oral hygiene at home – for example, toothbrush and toothpaste – was largely unknown. For dental emergencies, subjects were treated by a general practitioner in a hospital.

A total of six follow-up examinations were conducted through 1985 [14••]. As in the Norwegian cohort, the periodontal development of each subject in Sri Lanka was also documented at every examination. In addition to demographic

Table 1 Longitudinal studies on the natural progression of periodontitis

Country (location)	Ethnicity	Oral care (yes/no)	Population at baseline	Years of examination	Total observation period (no. of years)	Number of examinations	Mean clinical attachment loss per year (mm)	Reference
Norway (Oslo)	Caucasian	Yes	565	1969, 1971, 1973, 1975, 1981, 1988, and 1995	26	7	0.04–0.10 0.05 (age 16–59) 0.09 (age >20) 0.07–0.10 (age 20–30) 0.05 (age >30)	[9, 11, 31–33]
Sri Lanka (Kandy, Dunsinane, Sheen and Harrow tea estates)	Tamils	No	480	1970, 1971, 1973, 1977, 1982, 1985, and 1990	20	7	0.1–1.0 (RP) 0.05–0.5 (MP) 0.05–0.09 (NP)	[5••, 10, 12, 14••, 15–18, 34, 35]
China (Yanqing, Gushen)	Asian	No	587	1984 and 1994	10	2	0.15–0.19	[7•, 21, 23, 24, 36–40]
Indonesia (Java, Malabar, Purbasari tea estates)	Asian	No	255	1987, 1994, and 2002	15	3	0.05 (years 1–7) 0.15 (years ≥8)	[25–27, 41]
Australia (Adelaide or Mount Gambier, South Australia)	All	Yes	801	1991 and 1996	5	2	NA	[8]
Germany (West Pomerania, northeast region in Germany)	Caucasian	Yes	2,558	1997–2001 and 2002–2006	5	2	0.02	[22]
New Zealand	Caucasian	Yes	1,037	1998–1999 2004–2005 2010–2011	12	3	NA	[28]
Brazil (Porto Alegre)	Hispanic	Yes	1,465	2001 and 2006	5	2	0.3	[29]

RP rapidly progressive periodontal disease, *MP* moderately progressive periodontal disease, *NP* non-progressive periodontal disease

data, the subjects were asked about oral hygiene at home and smoking or betel nut chewing.

Attachment Loss

The effect of age on attachment loss was detected early (Table 1). Attachment loss was greater on buccal surfaces than on mesial surfaces with increasing age. In all subjects, the greatest percentage of attachment loss was found at the first molars of the maxilla, the first premolars of both jaws, and the first incisors of the mandible. The mean rate of attachment loss in this young cohort was less than 0.06 mm per year [15]. These results allow us to conclude that periodontal destruction progresses steadily and continuously [12].

In the 1986 study, the cohort from Sri Lanka was divided into three subgroups based on the course of periodontal disease. The division was determined according to the amount of tooth loss and interproximal attachment loss over a period of 15 years [12]. The three subgroups of periodontal disease progression were 1) rapidly progressive, 2) moderately progressive, and 3) non-progressive . Eight percent of the subjects were assigned to the rapidly progressive subgroup, 81 % to the moderately progressive subgroup, and 11 % to the non-progressive subgroup.

These three subgroups were also compared within different age cohorts. At age 35, the average attachment loss was around 9 mm in the rapidly progressive subgroup, around 4 mm in the moderately progressive subgroup, and less than 1 mm in the non-progressive subgroup. The annual rate of destruction varied between 0.1 and 1.0 mm in the rapidly progressive subgroup, between 0.05 and 0.5 mm in the moderately progressive subgroup, and finally, between 0.05 and 0.09 mm in the non-progressive subgroup.

Tooth Loss

In the baseline examination in Sri Lanka in 1970, the tea plantation workers had a total of 415 missing teeth. The mean number of teeth per individual was 27.05 (27.7–25.8), meaning that the average tooth loss was 0.95 per person. The loss of molars, especially of the mandible, was more conspicuous than that of other types of teeth. The mean number of teeth for the 15-year-olds was 27.0, and for the cohorts aged 30+ it was 26.7 [16].

In the rapidly progressive subgroup, tooth loss began at age 20 and increased over the next 25 years. At age 35, 12 teeth had been lost; at age 40, 20 teeth; and at age 45, all teeth were missing. In the moderately progressive subgroup, tooth loss did not begin until age 30, and increased over the decades. It should be noted, however, that because of the nearly caries-free status of this population, tooth loss for all subjects was attributed to periodontitis.

In 2005, tooth loss in the Sri Lanka cohort was analyzed in correlation with chewing betel nuts and other risk factors, and it was concluded that betel nut consumption, a history of loss of teeth, and the progression of attachment loss were significant predictors for tooth loss. [17].

Microbiological Examinations

The prevalence of periodontopathogenic bacteria – namely *Aggregatibacter actinomycetemcomitans*, *Porphyromonas gingivalis*, and *Prevotella intermedia* – in the Sri Lanka cohort was comprehensively described in 1995 [18]. With the use of microbiological culture techniques, *P. intermedia*, *P. gingivalis*, and *A. actinomycetemcomitans* were identified in 76 %, 40 %, and 15 % of the subjects, respectively. The prevalence of *P. intermedia*, however, was significantly higher in teeth with moderate or advanced periodontitis than in healthy teeth or teeth with gingivitis. *A. actinomycetemcomitans* was not found in healthy periodontal sites, but it was equally common in periodontal sites with gingivitis and moderate or advanced periodontitis [18].

Subgingival microbiological samples of 32 adults were assessed for characterization in an examination in 2010. Samples were taken from both shallow pockets (≤3 mm) and deep pockets (≥6 mm). Using a polymerase chain reaction-based strategy, the investigators constructed a clone library of 16S ribosomal RNA genes for each site. They found that the subgingival microbiota was composed of Firmicutes (69.8 %), Proteobacteria (16.3 %), and Fusobacteria (8.0 %). There was a significant difference in the composition of the microbiota between shallow and deep pocket sites [19].

China

In 1984, a total of 587 subjects, aged 20 to 80, from the village of Gushen in the Yanqing region of China were examined [20]. This village, with a population of around 250,000, was part of an agricultural community. Oral hygiene was largely neglected in this cohort, and dental care was provided only as emergency treatment in a hospital by non-dental personnel. Follow-up examinations of 440 subjects were conducted in 1994. In addition to documenting demographic data and dental findings, a periodontal examination was conducted of all subjects.

The variables of gender, percentage of sites with attachment loss of 4 mm or greater, and the presence of loose teeth were identified as positive predictors for the natural progression of periodontitis [21]. All subjects had a mean attachment loss of 2 mm or more over a period of 10 years. Higher rates of attachment loss progression were found for lower incisors and upper molars than for other teeth. The differences in the mean rates of attachment loss among the age groups were not significant. The results were similar to those reported for the

cohorts in more developed countries [9, 22]. On the basis of these results, it was concluded that the variability of periodontal destruction in humans was determined primarily by intrinsic factors [7•].

Up to 96 % of all subjects presented with tooth loss during the observation period of 10 years, and the mean tooth loss was 1.0 to 7.2 teeth. Using logistic regression analysis, additional predictors for tooth loss were described as age, high number of teeth with carious lesions, presence of periodontal disease at baseline examination, presence of teeth with attachment loss of 7 mm or more, and presence of loose teeth. At the subject level, both caries and periodontitis were equally important predictors for the incidence of tooth loss over the 10-year period. At the tooth level, however, caries was the major cause of tooth loss [23].

Microbiological Examinations

Eighteen microbial species associated with periodontitis were analysed in the China cohort. "Checkerboard" DNA–DNA hybridization technology was used to detect these species in the microbiological samples. In terms of prevalence, investigators found equally high rates in this population on both the subject and tooth site level. The distribution, however, was rather unequal. The bacteria preferred to concentrate on limited tooth sites. *Porphyromonas gingivalis*, *Treponema denticola*, and *Bacteroides forsythus* were frequently found in deep pockets or progression sites. Contrary to expectations, *A. actinomycetemcomitans* occurred the least frequently and always at low levels. With respect to this finding, it was considered that the virulence of *A. actinomycetemcomitans* may have different impacts among diverse populations [24].

Indonesia

The subjects of this prospective longitudinal study in Indonesia lived in West Java on the Malabar and Purbasari tea plantations [25]. The village selected for the study had around 2,000 inhabitants at the start of the study. The first examination took place in 1987 and included all inhabitants aged 15 to 25. The investigators documented clinical and microbiological data.

The cohort in Java was comparable to the tea plantation laborers in Sri Lanka in many respects. Of the 225 subjects who were included at the beginning of the study, 204 had completed primary education, but only 51 had continued their education. Thus the level of education was rather low. Health and medical care addressed only basic needs. There was no regular dental care and there were no programs for preventive dental health. For dental emergencies, tooth extractions were performed by a general practitioner.

After the baseline examination in 1987, two follow-up examinations were conducted in 1994 and 2002. At the last examination, 128 subjects remained, 69 male and 89 female. After the clinical parameters were documented, the microbiological parameters were measured and reported in 1998 and 2000 [25, 26]. The microbiological samples were taken from the dorsum of the tongue, the buccal gingiva in the upper jaw, the saliva, and the deepest bleeding pocket without clinical loss of attachment. Using phase contrast microscopy and indirect immunofluorescence, investigators identified *A. actinomycetemcomitans*, *P. gingivalis*, *P. intermedia*, and spirochetes on 53 %, 88 %, 100 %, and 100 % of all surfaces, respectively [26]. The prevalence of periodontopathogenic bacteria was conspicuously high even in periodontal pockets with no periodontitis.

In the 1998 study, it was concluded that the age of the subjects, the amount of subgingival plaque, and the subgingival presence of *A. actinomycetemcomitans* were predictors for progression of periodontal disease, with an annual progression rate of 0.15–0.19 mm [25]. This statement was formulated more precisely in a later report in 2006, where it was concluded that the subject's age, the amount of calculus, and the subgingival presence of *A. actinomycetemcomitans* correlated with the onset of periodontal disease. The natural progression of periodontitis was also associated with the presence of periodontal pockets with a probing depth of 5 mm or more, gingival recession, and male gender [27].

Australia

The South Australian Dental Longitudinal Study (SADLS) [8] was a study conducted in southern Australia that included subjects aged 60 and older who lived in Adelaide or Mount Gambier.

The baseline examination was conducted in 1991. Some 801 subjects took part in this initial examination, and 342 (42.7 %) of the subjects were still available for the follow-up examination five years later. After dental findings were documented, all subjects underwent a periodontal examination that included measuring of periodontal pocket depth, gingival recession, and attachment level. Since the total dentition of all subjects was included in this study, an impressive total of 15,522 sites (6,102 in the maxilla and 9,420 in the mandible) were used for the statistical analysis.

A primary objective of this study was to determine the five-year incidence of periodontal attachment loss in older patients. Additionally, further measurements were made to determine parameters such as gingival recession and pocket depth. There was no change in gingival recession or pocket depth at most sites. A threshold of 3 mm was specified for changes in attachment loss. Using this threshold, 2.3 % of the sites located were mesiobuccal, 2.5 % were buccal, and 3.4 % were distolingual. Molars at distolingual sites had the highest rate of progression in this cohort. Interestingly, attachment loss was most frequently in the form of gingival recession. Only 10.1 %

of attachment loss was attributed to an increase in pocket depth. In almost two-thirds of sites that showed attachment loss in the follow-up examination, a corresponding loss of at least 3 mm had been found in the baseline examination. According to the weighted five-year incidence, the overall risk for attachment loss was 43.2 %. However, it was higher for subjects who had already lost 11 teeth at the baseline examination [8].

Germany

Another longitudinal population-based study, the Study of Health in Pomerania (SHIP) [22], was conducted in northeastern Germany. The baseline examinations were performed between 1997 and 2001, and the follow-up examinations were conducted five years later, from 2002 to 2006. The SHIP study included 2,558 adults of both genders aged 20 to 81. In addition to documenting socioeconomic variables and dental findings, a periodontal examination of all subjects was conducted, including measuring periodontal pocket depths and attachment levels. If attachment loss of at least 3 mm was measured at two or more sites, it was documented as disease progression of a periodontal lesion.

The average change in mean attachment loss and mean pocket depth over five years was 0.10 mm and −0.05 mm – a 0.02-mm attachment loss and −0.01-mm reduction of pocket depth per year.

The attachment loss in this cohort was significantly higher in the groups aged 20–29 and 60–69 compared with the group aged 50–59. Interestingly, a total of 6.5 disease progression events per 100 person-years were observed. A statistical analysis of various parameters was made using multivariate models. Progression of disease was found to have a greater association with smokers, subjects with diabetes, medium and low levels of education, and single or divorced individuals.

The increasingly progressive attachment loss in the 20–29 and 60–81 age groups was described by the authors as the effect of negative risk factor distribution in these age groups – marital status (single or divorced) and failure to use interdental oral hygiene aids, for example [22].

New Zealand

The Dunedin Multidisciplinary Health and Development Study (DMHDS) is an ongoing longitudinal study examining a cohort of subjects who were all born at Queen Mary Hospital between 1972 and 1973 [28]. At baseline, the cohort included a total of 1,037 subjects. In addition to documenting general medical status, an intraoral examination was conducted, and systematic periodontal examinations were recorded for 831 subjects in this cohort at age 26, 32, and 38.

In this cohort, the extent of attachment loss increased with increasing age. Between ages 26 and 32, one in nine subjects had at least one site with advanced attachment loss. This figure doubled between the ages of 32 and 38. Based on this observation of disease progression, the cohort was divided into four subgroups; 55.2 % with “very low” progression, 31.5 % with “low” progression, 10.7 % with “moderately increasing” progression, and 2.5 % with “markedly increasing” progression. The subjects who had smoked continuously from age 15 to 38 had very high risk of “moderate” or “striking” progression. The same applied to subjects who were among the top 20 % of consumers of cannabis [28].

Brazil

In 2001, a total of 1,465 subjects in Porto Alegre in southern Brazil underwent a baseline examination [29]. In addition to documenting socioeconomic variables and dental findings, a periodontal examination of all subjects was performed, including measuring periodontal pocket depth and attachment levels. Efficient collection of data in this relatively large cohort was enabled through the use of multistage probability sampling. Five years later, a follow-up examination was conducted in 697 subjects.

In this prospective study, calculation of disease progression was possible by comparing the attachment levels in the two examinations. Disease progression greater than 3 mm at two or more teeth was found in 56 % of the subjects, and attachment loss greater than 3 mm at four or more teeth was found in 36 %. Moreover, attachment loss greater than 3 mm was usually localized, found at 3.8 teeth and 5.7 sites on average. Based on attachment loss, the annual progression of disease was 0.3 mm.

Overall disease progression was higher in men than in women and increased with age. The highest rate of progression was found in those aged 40 to 49, and the rate then dropped in older age groups. In addition, subjects of Latin American heritage and lower socioeconomic status generally suffered greater attachment loss [29].

Summary

The longitudinal studies discussed in this narrative review demonstrate a correlation between the natural progression of periodontitis and the oral hygiene habits of the subjects examined, particularly in the case of the Norway and Sri Lanka studies. However, while the occurrence of gingivitis and periodontitis was described in both cohorts, there were considerable differences in both the severity and progression of the disease. Unlike the subjects in Sri Lanka, the Norwegians had benefited from an excellent preventive dental program, and therefore suffered less from severe forms of periodontitis and subsequent tooth loss.

Periodontitis in subjects younger than 15 years of age was very rare in all cohort studies. With increasing age, however, the prevalence and severity of the disease increased in all populations globally. While gingivitis was detected in adolescents and remained a common occurrence into old age, the prevalence and severity of periodontitis varied among the cohorts and their subgroups around the world. The accumulation of plaque and subgingival calculus as a trigger and sustainer of disease progression was consistently found to play a leading role in all cohorts. Moreover, the different responses of the host to these pathogens were closely correlated with disease progression.

The results of all of the examinations discussed highlight the need for a global oral health program to prevent destructive periodontal disease and the serious consequences of tooth loss. Periodontal prophylaxis and the motivation to reduce risk factors are particularly advisable for individuals who are unfavorably exposed to risk factors. Dental professionals, therefore, should encourage their patients to improve interdental oral hygiene, quit tobacco use, and check diabetes management [30]. Health ministries, dental associations, and the dental industry in general should promote the use of interdental oral hygiene aids and reduction of risk factors. This could result in a significant improvement in public health and oral quality of life for individuals around the world.

Compliance with Ethics Guidelines

Conflict of Interest Ho-Yan Duong, Christoph Ramseier and Eric Schmid declare that they have no conflict of interest.

Human and Animal Rights and Informed Consent This article does not contain any studies with human or animal subjects performed by any of the authors.

References

Papers of particular interest, published recently, have been highlighted as:

- • Of importance
- •• Of major impotance.

1. Eke PI et al. Prevalence of periodontitis in adults in the United States: 2009 and 2010. J Dent Res. 2012;91(10):914–20.
2. Page RC. Current understanding of the aetiology and progression of periodontal disease. Int Dent J. 1986;36(3):153–61.
3. Socransky SS et al. New concepts of destructive periodontal disease. J Clin Periodontol. 1984;11(1):21–32.
4. Taba Jr M, Souza SL, Mariguela VC. Periodontal disease: a genetic perspective. Braz Oral Res. 2012;26 Suppl 1:p. 32–8.
5.•• Löe H et al. The natural history of periodontal disease in man. Study design and baseline data. J Periodontal Res. 1978;13(6):p. 550–62. *This manuscript represents the first description of the study of natural history of periodontitis in man.*
6. Van der Weijden GA et al. Comparison of different approaches to assess bleeding on probing as indicators of gingivitis. J Clin Periodontol. 1994;21(9):589–94.
7.• Baelum V et al. A 10-year study of the progression of destructive periodontal disease in adult and elderly Chinese. J Periodontol. 1997;68(11):p. 1033–42. *This manuscript represents the first study of the natural history of periodontitis in China.*
8. Thomson WM et al. Incidence of periodontal attachment loss over 5 years among older South Australians. J Clin Periodontol. 2004;31(2):119–25.
9. Schätzle M et al. Clinical course of chronic periodontitis. I Role of gingivitis. J Clin Periodontol. 2003;30(10):887–901.
10. Löe H, Anerud A, Boysen H. The natural history of periodontal disease in man: prevalence, severity, and extent of gingival recession. J Periodontol. 1992;63(6):489–95.
11. Heitz-Mayfield LJ et al. Clinical course of chronic periodontitis. II. Incidence, characteristics and time of occurrence of the initial periodontal lesion. J Clin Periodontol. 2003;30(10):902–8.
12. Löe H et al. The natural history of periodontal disease in man. The rate of periodontal destruction before 40 years of age. J Periodontol. 1978;49(12):607–20.
13. Schätzle M et al. Clinical course of chronic periodontitis: effect of lifelong light smoking (20 years) on loss of attachment and teeth. J Investig Clin Dent. 2010;1(1):8–15.
14.•• Löe H et al. Natural history of periodontal disease in man. Rapid, moderate and no loss of attachment in Sri Lankan laborers 14 to 46 years of age. J Clin Periodontol. 1986;13(5):p. 431–45. *This manuscript presents rates for rapid, moderate, and no periodontal disease progression in man for the first time.*
15. Anerud KE et al. Periodontal disease in three young adult populations. J Periodontal Res. 1983;18(6):655–68.
16. Löe H et al. The natural history of periodontal disease in man. Tooth mortality rates before 40 years of age. J Periodontal Res. 1978;13(6):563–72.
17. Neely AL et al. The natural history of periodontal disease in humans: risk factors for tooth loss in caries-free subjects receiving no oral health care. J Clin Periodontol. 2005;32(9):984–93.
18. Preus HR et al. 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(9):674–8.
19. Zhuang LF et al. Subgingival microbiota of Sri Lankan tea labourers naive to oral hygiene measures. J Clin Periodontol. 2014;41(5):433–41.
20. Baelum V et al. Tooth mortality and periodontal conditions in 60–80-year-old Chinese. Scand J Dent Res. 1988;96(2):99–107.
21. Baelum V et al. Predictors of destructive periodontal disease incidence and progression in adult and elderly Chinese. Commun Dent Oral Epidemiol. 1997;25(4):265–72.
22. Gätke D et al. Five-year change of periodontal diseases in the Study of Health in Pomerania (SHIP). J Clin Periodontol. 2012;39(4):357–67.
23. Baelum V et al. Predictors of tooth loss over 10 years in adult and elderly Chinese. Commun Dent Oral Epidemiol. 1997;25(3):204–10.
24. Papapanou PN et al. Subgingival microbiota in adult Chinese: prevalence and relation to periodontal disease progression. J Periodontol. 1997;68(7):651–66.
25. Timmerman MF et al. Untreated periodontal disease in Indonesian adolescents. Clinical and microbiological baseline data. J Clin Periodontol. 1998;25(3):215–24.
26. Timmerman MF et al. Untreated periodontal disease in Indonesian adolescents. Longitudinal clinical data and prospective clinical and microbiological risk assessment. J Clin Periodontol. 2000;27(12):932–42.
27. Van der Velden U et al. Java project on periodontal diseases. The natural development of periodontitis: risk factors, risk predictors and risk determinants. J Clin Periodontol. 2006;33(8):540–8.

28. Thomson WM et al. The natural history of periodontal attachment loss during the third and fourth decades of life. J Clin Periodontol. 2013;40(7):672–80.
29. Haas AN et al. Pattern and rate of progression of periodontal attachment loss in an urban population of South Brazil: a 5-years population-based prospective study. J Clin Periodontol. 2012;39(1):1–9.
30. Ramseier CA. Potential impact of subject-based risk factor control on periodontitis. J Clin Periodontol. 2005;32 Suppl 6:283–90.
31. Schätzle M et al. Clinical course of chronic periodontitis. III. Patterns, variations and risks of attachment loss. J Clin Periodontol. 2003;30(10):909–18.
32. Schätzle M et al. The clinical course of chronic periodontitis. J Clin Periodontol. 2004;31(12):1122–7.
33. Schätzle M et al. The clinical course of chronic periodontitis: V.Predictive factors in periodontal disease. J Clin Periodontol. 2009;36(5):365–71.
34. Anerud A, Loe H, Boysen H. The natural history and clinical course of calculus formation in man. J Clin Periodontol. 1991;18(3):160–70.
35. Neely AL et al. 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(8):1006–15.
36. Baelum V, Fejerskov O, Manji F. Periodontal diseases in adult Kenyans. J Clin Periodontol. 1988;15(7):445–52.
37. Baelum V, Fejerskov O, Manji F. The "natural history" of dental caries and periodontal diseases in developing countries: some consequences for health care planning. Tandlaegebladet. 1991;95(4): 139–48.
38. Baelum V et al. Six-year progression of destructive periodontal disease in 2 subgroups of elderly Chinese. J Periodontol. 1993;64(9):891–9.
39. Dahlen GG et al. Periodontopathogens in elderly Chinese with different periodontal disease experience. J Clin Periodontol. 1995;22(3):188–200.
40. Baelum V et al. Profiles of destructive periodontal disease in different populations. J Periodontal Res. 1996;31(1):17–26.
41. Timmerman MF et al. How do data from deepest pocket per quadrant relate to full-mouth scores? Progression of untreated periodontal disease in young Indonesians. J Clin Periodontol. 2002;29(3):219–23.