Gingivitis as a risk factor in periodontal disease


Abstract
Background: Dental plaque has been proven to initiate and promote gingival inflammation. Histologically, various stages of gingivitis may be characterized prior to progression of a lesion to periodontitis. Clinically, gingivitis is well recognized.

Material & Methods: Longitudinal studies on a patient cohort of 365 middle class Norwegian males have been performed over a 26-year period to reveal the natural history of initial periodontitis in dental-minded subjects between 16 and 34 years of age at the beginning of the study.

Results: Sites with consistent bleeding (GI = 2) had 70% more attachment loss than sites that were consistently non-inflamed (GI = 0). Teeth with sites that were consistently non-inflamed had a 50-year survival rate of 99.5%, while teeth with consistently inflamed gingivae yielded a 50-year survival rate of 63.4%.

Conclusion: Based on this longitudinal study on the natural history of periodontitis in a dentally well-maintained male population it can be concluded that persistent gingivitis represents a risk factor for periodontal attachment loss and for tooth loss.

Conflict of interest and source of funding statement
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We are here in Stockholm this week to celebrate the 100th Anniversary of the Swedish Dental Association and to congratulate our Swedish colleagues on their many important contributions to the science and practice of dental medicine.

Let us be reminded, however, that during the first 50 years of the Association’s history, insufficient knowledge existed regarding the nature and causes of periodontal disease, and there was little agreement on their clinical management both in Sweden and worldwide.

It was from this state of ignorance and professional inaction that the Scandinavian school, led by Dr. Jens Wærhaug during the early 1950s, initiated what would amount to a revolution in periodontal research and practice.

Dental Plaque

First microscopic and electron microscopic research revealed the intimate relationship between dental plaque and the gingival and periodontal tissues.

Then, epidemiological studies in many countries pointed to a close association between tooth deposits and periodontal diseases.

Finally, in 1965, the experimental gingivitis studies demonstrated that the accumulation of plaque on healthy gingiva produced gingivitis (Löe et al. 1965) (Fig. 1a–d), and that after reinstitution of oral hygiene measures for 7 days, the gingiva reverted back to normal (Fig. 2). These studies also described the sequential development of gingival plaque from a simple monolayer of Gram-positive coccoïd bacteria colonizing the enamel surface and the marginal gingiva to a complex microbial plaque dominated by Gram-negative anaerobic cocci, filaments and spirochetes (Theilade et al. 1966). These and other studies came to be the ultimate documentation that bacterial plaque was something very different from food debris, something much more colourful than Materia Alba, and much more interesting than just “Schmutz” (Fig. 3a and b). Consequently, the research to understand more fully the interaction between the infectious agents and the lost tissues in the transition from health to disease, and in the progression from gingivitis to periodontitis was intensified.

Supragingival plaque related to healthy gingival tissues has a bacterial com-
These and others have proven their effects on signalling gene expression, mobilizing of inflammatory cells in mediating the release of enzymes like collagenases, other proteolytic enzymes, including the metallo-proteinases, which are responsible for the degradation, and the extracellular matrix of the connective tissue. Also, these in turn give rise to potent products, such as prostaglandins, particularly PGE2 and others that are involved in several inflammatory responses or enzymes, or indirectly, as a result of activation of the patient’s cellular and inflammatory systems – the so-called host responses, which may serve to both damage and protect the periodontal tissues.

Although no one has directly observed the earliest stages of gingivitis development, it is surmised that these include processes germane to acute inflammation:

- transudation of serum through the vascular endothelium,
- extravasation of leucocytes and their outward emigration,
- widening of the intercellular spaces of the surface epithelia and
- gradual release of immuno-inflammatory mediators with a variety of potentials, such as cytokines [interleukin (IL)-1β, IL-6, IL-8 and tumour necrosis factor-α].

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processes during the progression of the
disease, of which bone resorption might
be the most important.

Also during the early stages of the
process, various bacterial antigens will
elicit the production of antibodies, mobilize the complement system and
release other immunoglobulins, along
with the emergence of phagocytic cells
such as plasma cells and activation of
other cell types such as lymphocytes
and macrophages (Fig. 5a and b). As
these complex processes pervade the
marginal gingiva, the developing gin-
gival lesion matures into chronic gin-
givitis, with massive accumulations of
cells and fluid overtaking the connective
tissues stroma.

Clinical Gingivitis

The earliest clinical sign of gingival
inflammation is the transudation of gin-
gival fluid. This thin and almost a cel-
lar transudate is gradually superseded
by a fluid consisting of serum plus
leucocytes.

The redness of the gingival margin
arises partly from the aggregation and
enlargement of blood vessels in the
immediate subepithelial connective tis-
tue and the loss of keratinization of the
facial aspects of gingiva (Fig. 6a and b).

Swelling and loss of texture of the
free gingiva reflect the loss of fibrous
connective tissue and the semi liquidity
of the interfibrillar substance.

Individually and collectively, the
clinical symptoms of chronic gingivitis
are rather vague, and usually painless.
These features leave most patients una-
ware of the disease and are generally
underestimated by the dental practi-
tioners. Chronic gingivitis rarely shows
spontaneous bleeding. The fact that the
gingival tissues can be provoked to
bleed just by touching the gingival
margin with a blunt instrument (Fig. 7)
as during tooth brushing or in assessing
the gingival index (GI) suggests that
the epithelial changes and the vascular
transfigurations are quite conspicuous.

Progression to Periodontitis

Cross-sectional epidemiological studies
from many countries have indicated
that gingivitis is common both in primary
and in secondary dentitions of children,
and affects most adults. National prob-
ability surveys in some industrialized
countries have found that gingivitis
occurs in the majority of teenagers and
adults, but that only a few, perhaps
<10% of the total gingival sites, "bleed
on probing" (GI = 2). Similar data based
on probability samples are not available
for developing regions, but it has been
suggested that gingivitis might be highly
prevalent, extensive and severe.

The distribution of gingivitis in any
population is important since current
theory holds that the gingival lesion is
the precursor of periodontitis. Clearly,
not all gingivitis lesions progress to
periodontitis. Actually, the proportion
of gingival lesions converting to perio-
dontitis, and the factor causing this
conversion have not been well under-
stood. Most seem to think, however, that
the established periodontitis lesion was
preceded by gingivitis.

Fig. 3. Experimental gingivitis in humans (Löe et al. 1965). Graphs demonstrating the cause-
and-effect relationship between dental plaque and the development of gingivitis (b). As the
Plaque indices increase (a), the gingival indices follow, yielding the host response to the
bacterial challenge (b). Following re-institution of oral hygiene (a), gingival health is, again,
reached within a week to 10 days (b).

Fig. 4. Plaque begins to form in the gingival sulcus and other protected niches of the tooth.
(a) Destruction of collagen will occur as a direct result of microbial action through their
release of toxins, lipopolysaccharides or enzymes. (b) Bacterial plaque forms as a biofilm in
the gingival sulcus shortly after the cleansing of the tooth surface. Three-day-old plaque.
While traditional cross-sectional human surveys may describe reasonably well the distribution of disease and the associated factors, cross-sectional studies are unable to characterize the continuous process and the temporal and spatial relationships between the various factors influencing the initiation and the progression of the disease. Also, the determination of the cause of a disease or the definition of the true risk factors of a disease requires a longitudinal design of a study.

Longitudinal Studies

The material presented in this review is obtained from longitudinal studies of periodontal disease in humans (Heitz-Mayfield et al. 2003, Schätzle et al. 2003a, b, 2004).

The cohort was established in Oslo Norway in 1969, and consisted of 565 randomized healthy, well-educated males between 16 and 34 years old. All participants were born in Oslo, and all had been enrolled in the City Dental Program during childhood, and reported to have subsequently seen their private dentist on a regular basis, as well as performing daily oral hygiene care.

Subsequent clinical examination of the participants occurred in 1971, 1973, 1975, 1981, 1988 and 1995. As in most longitudinal investigations of this size and length, a certain member of the participants dropped out, and could not be followedup. Others would miss one or more examinations, but might show up for the last survey; 223 men showed up for the last examination covering the entire age range from 16 to 60 years.

The following clinical indices were collected during the 26-year observation period: plaque index, GI, calculus index, caries index, gingival recession and loss of attachment. (Pocket depths were derived from calculating LA and GRL.)

These studies have shown that in well-educated middle-class men who had received state-of-the-art professional and personal dental care during childhood, adolescence and adulthood, the dentition is maintained in health and function throughout life. The 16-year-old boys had 27.4 teeth (excluding wisdom teeth), and as the men approached 60 years, they still had 27.1 teeth. They have lost 0.3 teeth in 45 years of adult

Fig. 5. (a) Photomicrograph of a gingivitis lesion. The inflammatory infiltrate is clearly visible: extravasations of leucocytes and their outward emigration, widening of the intercellular spaces of the surface epithelia and a gradual release of immuno-inflammatory mediators with a variety of potentials. (b) Higher magnification of (a): Bacterial plaque is located adjacent to the non-keratinized crevice epithelium. Emergence of phagocytic cells such as plasma cells and activation of other cell types such as lymphocytes and macrophages.

Fig. 6. Aggregation and enlargement of blood vessels in the immediate subepithelial connective tissue. (a) Vital microscopy of a clinically healthy gingival margin. Presence of numerous fine capillary networks. (b) Vital microscopy of a gingival margin presenting with gingivitis. Capillary loops are enlarged. Clinically, these changes are recognized as redness and swelling of the gingival margin.

Fig. 7. In health, the insertion of a blunt instrument or probe into the gingival crevice will maintain its integrity. However, bleeding may be induced as a result of developing gingivitis.
life, and the mortality rate was 0.01 teeth/year (Schätzle et al. 2004).

Although none of the participants at any one survey exhibited a dentition completely free of plaque, it is noteworthy that approximately 60–70% of the total tooth surfaces were either plaque free or scored PI $I = 1$ (invisible plaque), and that throughout the age range (16–60 years) only 20–30% of the surfaces scored PI $I = 2$ (visible plaque).

Gingival indices also varied very little during adult life and ranged between GI $= 0.1$ and 0.9. In other words, the gingival health of this group of people must be characterized as good to excellent, and the frequency of GI $= 2$ scores (bleeding on probing) occurred only in 10–20% of sites in all men 16–60 years of age (Schätzle et al. 2003a).

Attachment loss occurred in a few sites already at 16 years, but mainly as gingival recession on buccal surfaces (Heitz-Mayfield et al. 2003). Deepened pockets were scarce below 40 years of age, and rarely exceeded 3.4 mm in depth. The mean individual loss of attachment (LA) and the frequency of sites with attachment loss increased somewhat during the 40s and 50s, and reached a maximum (LA $= 2.44$ mm) as the men approached 60 years. However, $< 0.5\%$ of the sites had lost $> 4$ mm.

To analyse the role of gingival inflammation in the pathogenesis of the periodontal lesion, three gingival index severity groups were established across all age groups according to their degree of clinical inflammation scores over the 26-year observation period (Schätzle et al. 2003a).

The results show that gingival units that consistently scored GI $= 0$ experienced a mean cumulative LA over the 60 years life span of $< 2$ mm (range 0.08–1.94). This attachment loss essentially occurred in buccal sites of young men below 40 years, displaying gingival recession. Sites with slight inflammation (GI $= 1$) showed a cumulative attachment loss of $> 2$ mm (range 0.17–2.42), and in sites that consistently bled on probing (GI $= 2$), the mean LA was $> 3$ mm (range 0.04–3.53) (Schätzle et al. 2003a) (Fig. 8).

From the age of 40 and 50 years, pocket formation increased in extent and depth in sites exhibiting gingival inflammation. As the men approached 60 years of age, gingival sites that, throughout the observation period consistently bled on probing, had 70% more attachment loss than sites that consistently were non-inflamed, yielding an odds ratio of 3.22 for inflamed sites converting to attachment loss. Also, subgingival calculus formation increased the odds ratio for converting to attachment loss to 4.22, for the GI $= 2$ group, compared with sites of the GI $= 0$ cohort (Schätzle et al. 2003a).

From this part of the analysis, it was concluded that it has now convincingly been demonstrated that the development of periodontitis only occurs in areas of long-standing gingivitis.

In order to further elucidate the transition from gingivitis to periodontitis, the incidence of the initial loss of attachment over the 60-year life span was next assessed (Heitz-Mayfield et al. 2003). The loss of the first 2 mm or more of periodontal attachment as measured apically from the cemento-enamel junction was termed as initial loss of attachment (ILA). The incidence of ILA was established for all age levels, all teeth and groups of teeth as well as for the type of lesion expressed (gingival recession, pockets alone or in combination with recession). Again, and not surprisingly, at age 20 years and before ILA occurred in a few buccal sites in a few individual and almost all were gingival recessions.

At 40 years of age, still $< 10\%$ of the sites with ILA showed either pocket formation or condensation of attachment loss and pocketing. As the men aged 50, 55, 60 years, however, approximately 30%–50% of the sites had ILA (Heitz-Mayfield et al. 2003).

It appears then that the incidence of attachment loss of $\geq 2$ mm expressed as deepening of the periodontal pocket is absent or extremely low during the early years of life. In fact, gingival recession was the dominant type of initial attachment loss during the first 40 years of life. Pocket formation starts earliest around 50 years of age, and increases in incidence towards the age of 60.

Another analysis of this unique cohort aimed at the assessment of the long-term influence of gingival inflammation on tooth survival (Schätzle et al. 2004).

For tooth-specific calculations, three gingival index groups reflecting their history of inflammation over the 26 years were established. For this reason, the minimal and maximal GI value over all sites was determined:

- Severity I: teeth consistently scoring a minimum of GI $= 0$ and a maximum of GI $\leq 1$ in all sites.
- Severity II: teeth in which all sites always scored a minimum of GI $= 1$ and a maximum of GI $\leq 2$.
- Severity III: teeth that always scored a minimum of GI $= 2$ (bleeding on probing) in all sites and at all observations.

All other teeth not fulfilling these criteria were not considered for further evaluation.

Four hundred and eighty-seven patients with 13,285 teeth in 1969 were followed up for at least two surveys. Of the 487 subjects, 412 (85%) had lost no teeth and the remaining 75 of the re-examined individuals accounted for the 126 teeth lost.

The Kaplan–Meier cumulative survival distribution (Fig. 9) for the three defined GI Severity Groups showed

![Image](7)

Fig. 8. Effect of different gingivitis levels on the loss of attachment. The development of periodontitis only occurs in areas of long-standing gingivitis (from: Schätzle et al. 2003a).
that before a tooth age of approximately 15 years, tooth loss was extremely rare. After a tooth age of 20 years, GI Severity Group III yielded a significantly increased cumulative tooth loss when compared with both GI Severity Groups II and I.

It is remarkable to note that GI Severity Group I, where no bleeding on probing was ever scored throughout the observation period, yielded a cumulative survival of 99.5% at a tooth age of 51 years. This suggests that clinically healthy gingiva is a prognostic indicator of tooth longevity.

The fact that the GI Severity Group II demonstrated a cumulative tooth survival rate of 94% after 51 years of tooth age supports the concept that occasionally, bleeding tooth sites provide a much smaller risk for tooth loss than regularly bleeding gingivae.

The risk of losing a tooth showed a wide range of odds ratios. Teeth always surrounded by healthy or slightly inflamed gingiva had an 8.4 times lower risk of being lost as compared with teeth surrounded by an inflamed gingiva that occasionally bled on probing, and a 45.8 times lower risk than teeth that were always surrounded by an inflamed gingiva that bled on probing. Teeth with slightly inflamed gingiva had a 5.4 times lower risk of tooth loss than those that showed bleeding on probing.

This study has clearly established that gingival inflammation was a risk factor for tooth loss. Teeth consistently surrounded by inflamed gingiva at all surveys during the 26-year observation period had a significantly higher risk of being lost than teeth with no or only slight inflammation.

Clinical Relevance

Scientific rationale for the study: Gingivitis may be perceived as a protective host response against the bacterial challenge. It is unclear whether or not gingivitis may lead to detrimental effects.

Principal findings: Teeth with consistently inflamed dentogingival units showed over a 26-year observation period greater attachment loss and a higher chance for tooth loss than teeth with consistently non-inflamed dentogingival units.

Practical implications: Inflammation of the gingival tissues represents not only the precursor of periodontitis but also a clinically relevant risk factor for disease progression and tooth loss.

Fig. 9. Effect of different gingivitis levels on tooth loss. Survival distribution function for different gingival index Severity Groups. After 50 years of tooth age, the cumulative survival for teeth with healthy gingival tissues is 99.5%. Teeth with always bleeding gingivae demonstrate a cumulative survival of 63.4% after 48 years of tooth age (from: Schätzle et al. 2004).

References


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