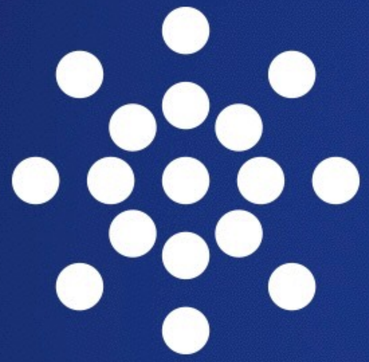


EFP

New Classification

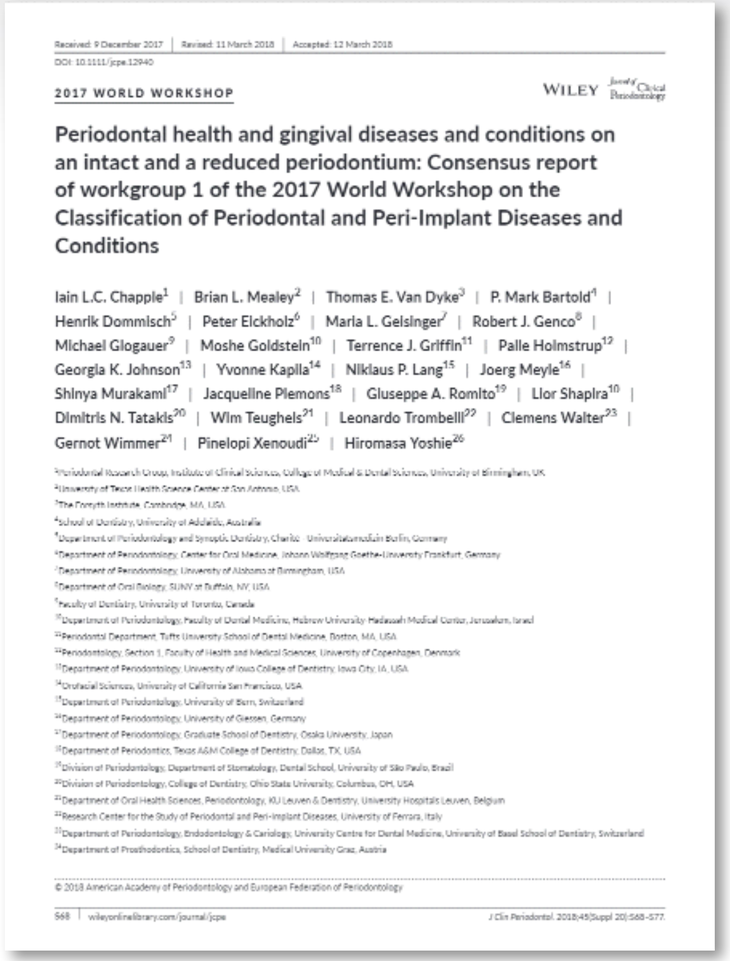
of periodontal and peri-implant diseases

EFP European Federation of Periodontology

Periodontal health and gingivitis

Iain Chapple

World Workshop: Workgroup 1



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2017 WORLD WORKSHOP

WILEY Journal of Clinical Periodontology

Periodontal health and gingival diseases and conditions on an intact and a reduced periodontium: Consensus report of workgroup 1 of the 2017 World Workshop on the Classification of Periodontal and Peri-Implant Diseases and Conditions

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Periodontal health, gingival diseases and conditions

Chairs: Iain Chapple
Brian Mealey

Four expert position papers

Periodontal health
Niklaus P. Lang and P. Mark Bartold

Dental plaque-induced gingival conditions
Shinya Murakami, Iain Chapple, Brian Mealey, and Angelo Mariotti

Non-plaque-induced gingival diseases
Palle Holmstrup, Jacqueline Plemons, and Joerg Meyle

Plaque-induced gingivitis: Case definition and diagnostic considerations
Leonardo Trombelli, Roberto Farina, Cléverson Silva, and Dimitris Tatakis

Expert paper 1: Periodontal health

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2017 WORLD WORKSHOP

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Periodontal health

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The proceedings of the workshop were jointly and simultaneously published in the *Journal of Periodontology* and *Journal of Clinical Periodontology*.

Abstract

Objectives: To date there is a paucity of documentation regarding definitions of periodontal health. This review considers the histological and clinical determinants of periodontal health for both intact and reduced periodontium and seeks to propose appropriate definitions according to treatment outcomes.

Importance: Defining periodontal health is can serve as a vital common reference point for assessing disease and determining meaningful treatment outcomes.

Findings: The multifactorial nature of periodontitis is accepted, and it is recognized that restoration of periodontal health will be defined by an individual's response to treatment, taking into account allostatic conditions.

Conclusions: It is proposed that there are 4 levels of periodontal health, depending on the state of the periodontium (structurally and clinically sound or reduced) and the relative treatment outcomes: (1) pristine periodontal health, with a structurally sound and uninflamed periodontium; (2) well-maintained clinical periodontal health, with a structurally and clinically sound (intact) periodontium; (3) periodontal disease stability, with a reduced periodontium, and (4) periodontal disease remission/control, with a reduced periodontium.

KEYWORDS
Clinical health, gingiva, periodontal remission, periodontal stability, pristine health

INTRODUCTION

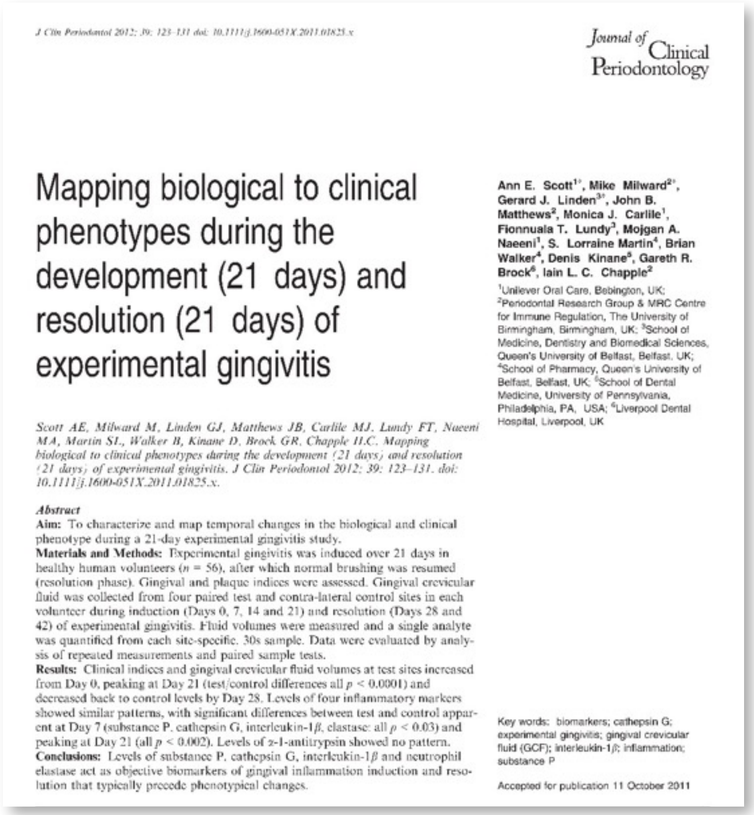
"Health is a state of complete physical, mental and social well-being and not merely the absence of disease or infirmity."[1] In accordance with this definition by the World Health Organization, periodontal health should be defined as a state free from inflammatory periodontal disease that allows an individual to function normally and not suffer any consequences (mental or physical) as a result of past disease. However, while this definition is holistic and patient-outcome based, it seems an impractical and limiting definition for the purposes of clinical management of periodontal diseases. Therefore, a more practical definition of periodontal health would be a state free from inflammatory periodontal disease. This, in turn, means that absence of inflammation associated with gingivitis or periodontitis, as assessed clinically, is a prerequisite for defining periodontal health.

It is a matter of debate if altered morphological conditions resulting from previous exposure to disease processes (eg. gingival recession, loss of attachment, and bone loss) may be redefined as novel healthy conditions in the absence of clinical signs and symptoms of inflammation.

Interestingly, there are almost no studies or reports attempting to define periodontal health.[2] Defining periodontal health is very important if we are to have a common reference point for assessing periodontal disease and determining meaningful treatment outcomes. Health can be evaluated at both the histological and clinical levels and should be considered in the context of a preventive starting point and a therapeutic end point. Thus, periodontal health can exist before disease commences but, conversely, periodontal health can be restored to an anatomically reduced periodontium. In this review, the clinical criteria for distinguishing pristine health from health on a reduced periodontium are presented and discussed.

- Is there a holistic definition of periodontal health?
- Does "pristine periodontal health" exist?
- If pristine health exists, is that "normality" (95% population)?
- Is there a biological level of inflammation consistent with clinical gingival health & homeostasis? Clinical vs pristine health.
- Can periodontitis enter into "remission"?

Biological phenotype *versus* clinical phenotype



J Clin Periodontol 2012; 39: 123–131 doi: 10.1111/j.1600-051X.2011.01825.x

Journal of Clinical Periodontology

Mapping biological to clinical phenotypes during the development (21 days) and resolution (21 days) of experimental gingivitis

Ann E. Scott[1,*], Mike Milward[2,*], Gerard J. Linden[3,†], John B. Matthews[2], Monica J. Carlile[1], Fionnuala T. Lundy[3], Mojgan A. Naeeni[1], S. Lorraine Martin[4], Brian Walker[4], Denis Kinane[5], Gareth R. Brock[6], Iain L. C. Chapple[2]

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Scott AE, Milward M, Linden GJ, Matthews JB, Carlile MJ, Lundy FT, Naeeni MA, Martin SL, Walker B, Kinane D, Brock GR, Chapple ILC. Mapping biological to clinical phenotypes during the development (21 days) and resolution (21 days) of experimental gingivitis. J Clin Periodontol 2012; 39: 123–131. doi: 10.1111/j.1600-051X.2011.01825.x.

Abstract

Aim: To characterize and map temporal changes in the biological and clinical phenotype during a 21-day experimental gingivitis study.

Materials and Methods: Experimental gingivitis was induced over 21 days in healthy human volunteers ($n = 56$), after which normal brushing was resumed (resolution phase). Gingival and plaque indices were assessed. Gingival crevicular fluid was collected from four paired test and contra-lateral control sites in each volunteer during induction (Days 0, 7, 14 and 21) and resolution (Days 28 and 42) of experimental gingivitis. Fluid volumes were measured and a single analyte was quantified from each site-specific, 30s sample. Data were evaluated by analysis of repeated measurements and paired sample tests.

Results: Clinical indices and gingival crevicular fluid volumes at test sites increased from Day 0, peaking at Day 21 (test/control differences all $p < 0.0001$) and decreased back to control levels by Day 28. Levels of four inflammatory markers showed similar patterns, with significant differences between test and control apparent at Day 7 (substance P, cathepsin G, interleukin-1β, elastase: all $p < 0.03$) and peaking at Day 21 (all $p < 0.002$). Levels of α-1-antitrypsin showed no pattern.

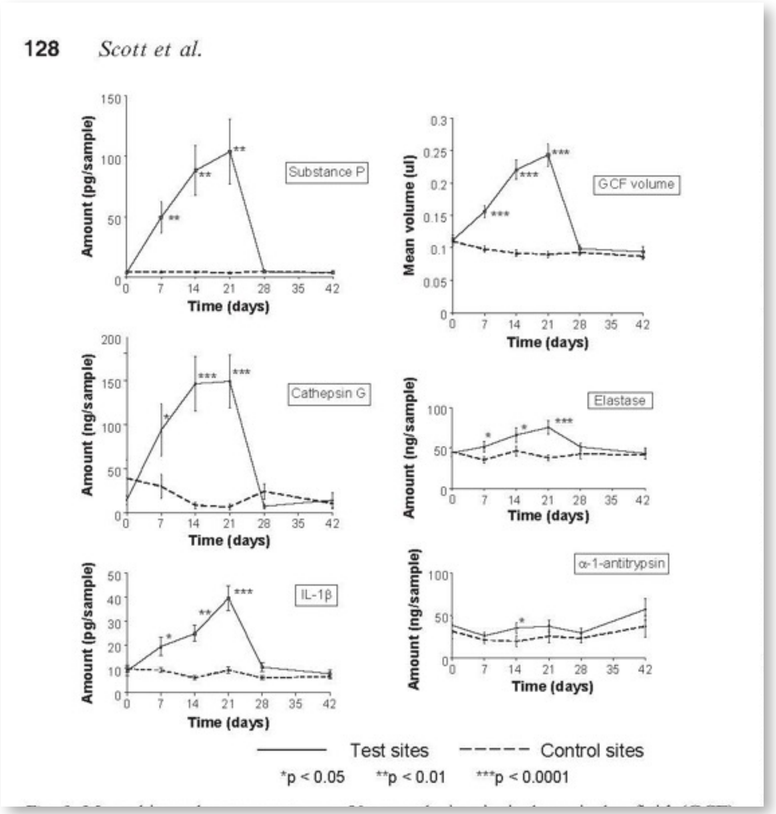
Conclusions: Levels of substance P, cathepsin G, interleukin-1β and neutrophil elastase act as objective biomarkers of gingival inflammation induction and resolution that typically precede phenotypical changes.

Key words: biomarkers; cathepsin G; experimental gingivitis; gingival crevicular fluid (GCF); interleukin-1β; inflammation; substance P

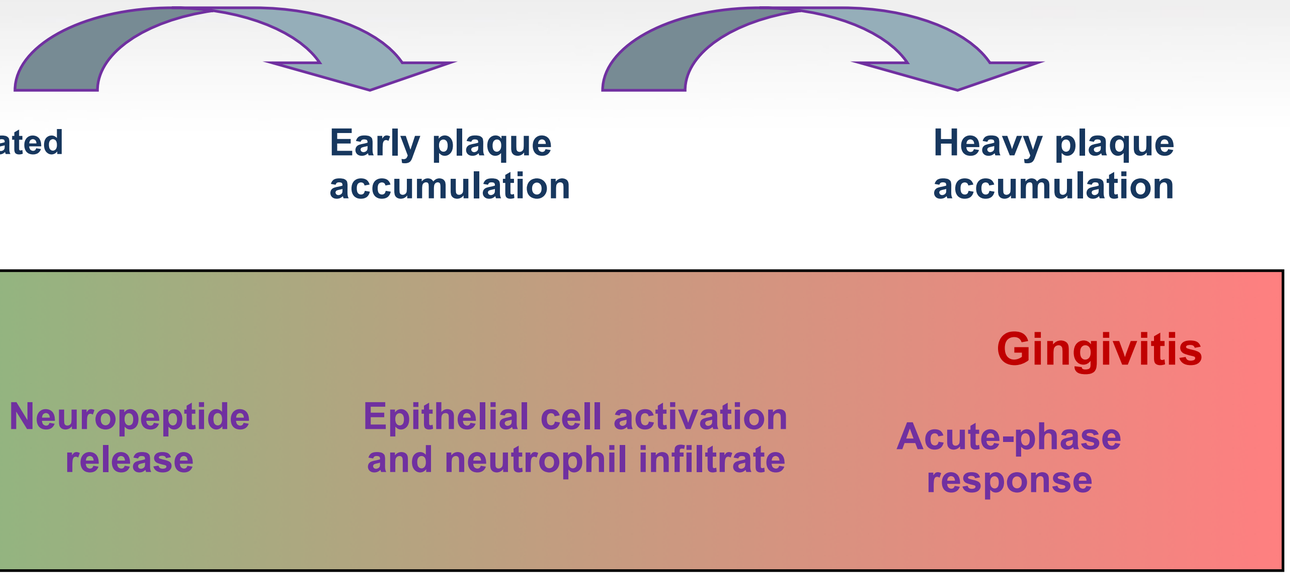
Accepted for publication 11 October 2011

What can we use for perio?

- **A biological phenotype exists in gingivitis.**
- **The biological phenotype can be mapped to clinical changes.**
- **Such a biological phenotype has not yet been defined for periodontitis.**



Biological model for stages from health to gingivitis



We cannot do this yet for periodontitis.
It is too complex: there is no single cause.

Gingival transcriptome data for CP versus AgP

RESEARCH REPORTS
Clinical

M. Kebschull[1†], R.T. Demmer[2], B. Grün[3], P. Guarnieri[4,5], P. Pavlidis[6], and P.N. Papapanou[1*]

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J Dent Res 93(5):459-468, 2014

Gingival Tissue Transcriptomes Identify Distinct Periodontitis Phenotypes

ABSTRACT

The currently recognized principal forms of periodontitis—chronic and aggressive—lack an unequivocal, pathobiology-based foundation. We explored whether gingival tissue transcriptomes can serve as the basis for an alternative classification of periodontitis. We used cross-sectional whole-genome gene expression data from 241 gingival tissue biopsies obtained from sites with periodontal pathology in 120 systemically healthy nonsmokers with periodontitis, with available data on clinical periodontal status, subgingival microbial profiles, and serum IgG antibodies to periodontal microbiota. Adjusted model-based clustering of transcriptomic data using finite mixtures generated two distinct clusters of patients that did not align with the current classification of chronic and aggressive periodontitis. Differential expression profiles primarily related to cell proliferation in cluster 1 and to lymphocyte activation and unfolded protein responses in cluster 2. Patients in the two clusters did not differ with respect to age but presented with distinct phenotypes (statistically significantly different whole-mouth clinical measures of extent/severity, subgingival microbial burden by several species, and selected serum antibody responses). Patients in cluster 2 showed more extensive/severe disease and were more often male. The findings suggest that distinct gene expression signatures in pathologic gingival tissues translate into phenotypic differences and can provide a basis for a novel classification.

KEY WORDS: gene expression, genomics, cluster analysis, classification, diagnosis, periodontal diseases.

DOI: 10.1177/0022034514527288

INTRODUCTION

There are few readily discernible phenotypic differences between the two currently recognized principal forms of periodontitis: chronic (CP) and aggressive (AgP) ("International Workshop for the Classification of Periodontal Diseases and Conditions," 1999). Early onset of the disease and rapid progression rate that leads to extensive and severe tissue destruction are more frequently associated with AgP than CP, without being pathognomonic for either form. The resulting diagnostic dilemma based on clinically identifiable traits (Armitage and Cullinan, 2010) is further sustained by the presence of common microbiological, immunologic, and histopathologic features of the two entities (Armitage, 2010; Ford *et al.*, 2010; Smith *et al.*, 2010).

To further explore potential differences in the pathophysiology of the two forms, we recently carried out a "class validation" analysis by studying whole-genome transcriptomic profiles in CP and AgP gingival lesions (Kebschull *et al.*, 2013). Although our data demonstrated differential enrichment in a number of gene ontology pathways, the highly variable performance of the supervised machine learning algorithms used, as well as the need to account for the expression of thousands of genes to effectively distinguish between CP and AgP, further support the notion that the current classification has an imperfect biological foundation.

In this study, we hypothesized that gene expression profiles in gingival tissues obtained from patients with periodontitis can form the basis for an alternative classification of the disease. To address this hypothesis, we employed the same data set as in our previous work (Kebschull *et al.*, 2013) in a "class discovery" analytical approach and used clustering, an unsupervised learning method, to identify *de novo* groups of periodontitis patients with common gene expression features. Subsequently, we examined the periodontitis-related phenotypes of the emerging clusters of patients, with respect to clinical characteristics, subgingival bacterial profiles, and serum IgG antibody levels to periodontal microbiota.

Adjusted model-based clustering of transcriptomic data using finite mixtures generated two distinct clusters of patients that did not align with the current classification of chronic and aggressive periodontitis.

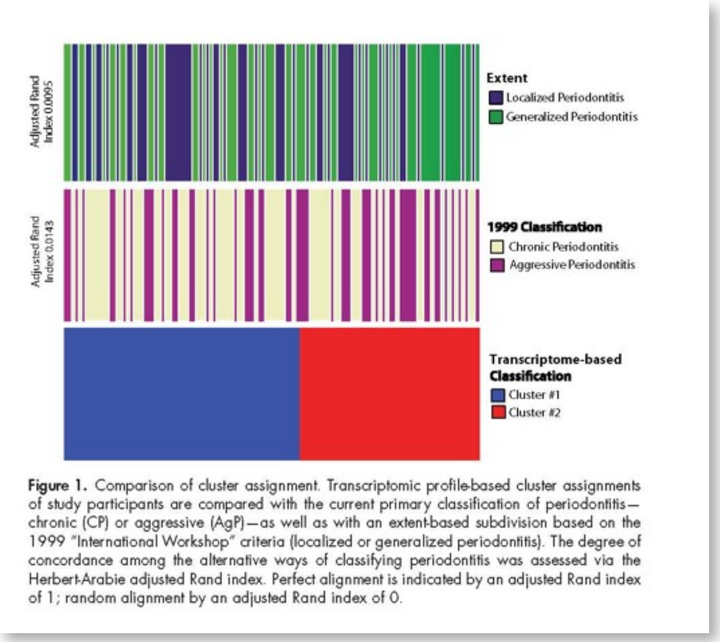
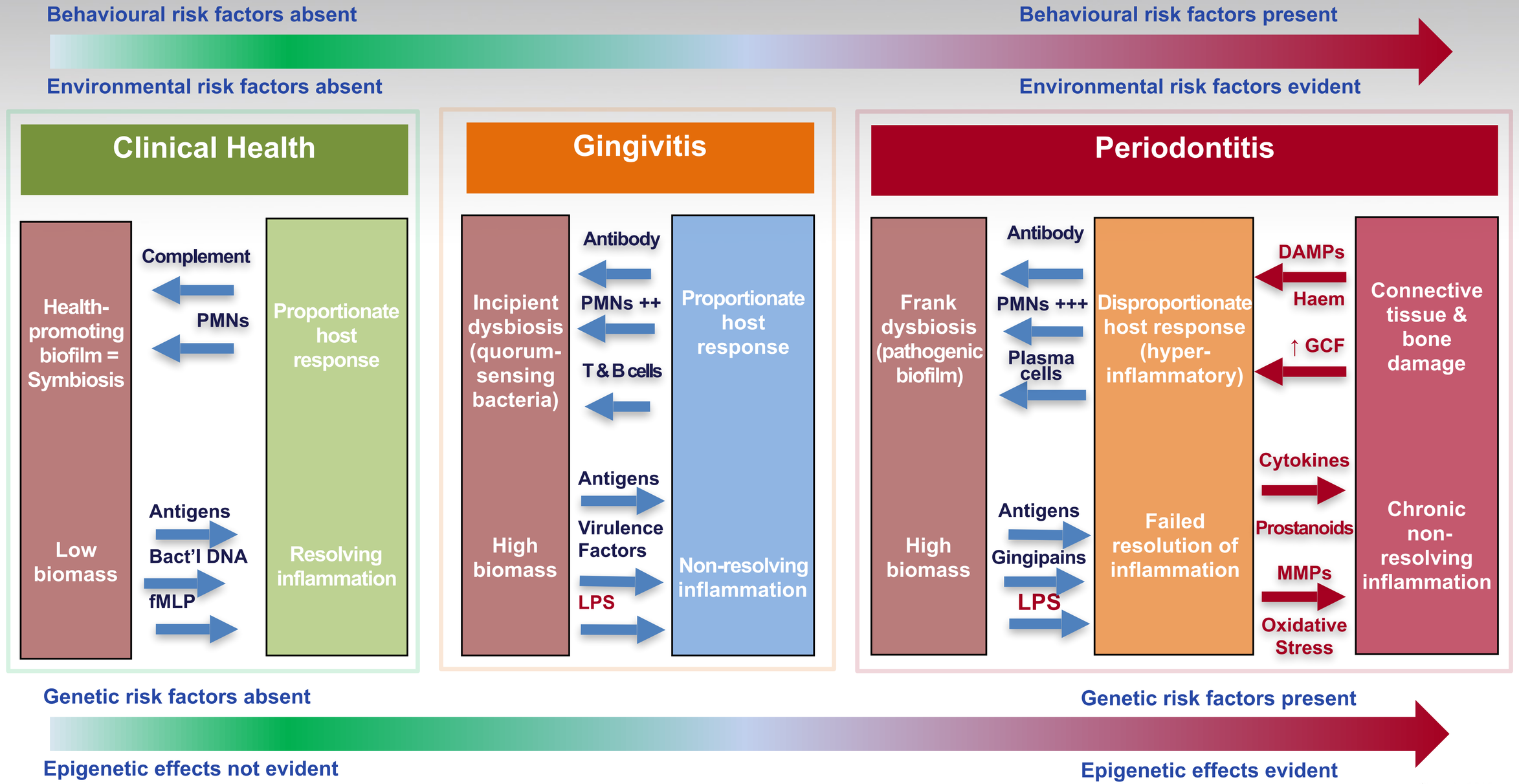


Figure 1. Comparison of cluster assignment. Transcriptomic profile-based cluster assignments of study participants are compared with the current primary classification of periodontitis—chronic (CP) or aggressive (AgP)—as well as with an extent-based subdivision based on the 1999 "International Workshop" criteria (localized or generalized periodontitis). The degree of concordance among the alternative ways of classifying periodontitis was assessed via the Herbert-Arabie adjusted Rand index. Perfect alignment is indicated by an adjusted Rand index of 1; random alignment by an adjusted Rand index of 0.



Chapple 2015

WHO definition of health

“Health is a state of complete physical, mental and social well-being and not merely an absence of disease or infirmity.”

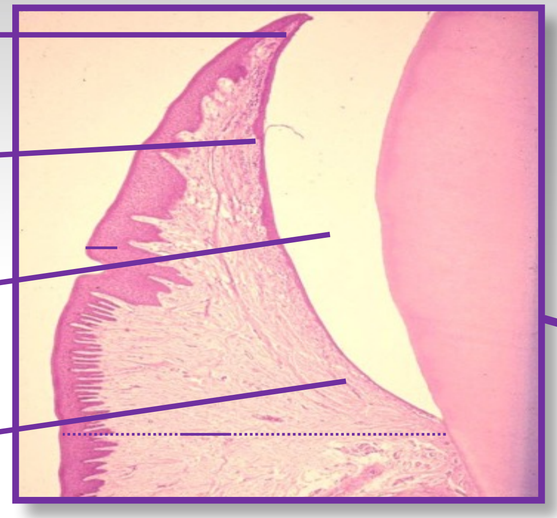
WHO 1948: 19456 No 2:1

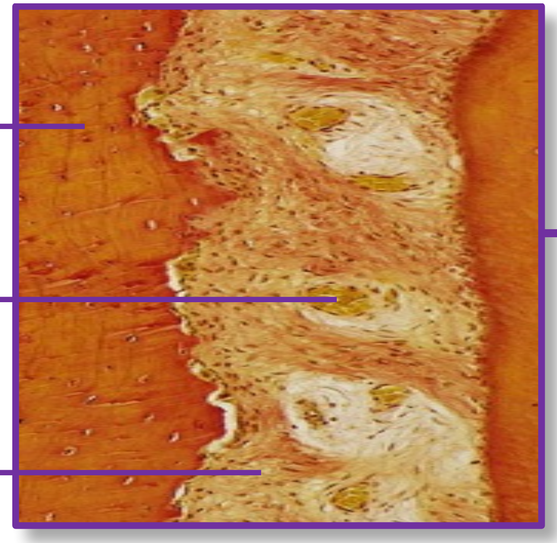
Thus, periodontal health should be defined as a state free from inflammatory periodontal disease that allows an individual to function normally and avoid physical or mental consequences due to current or past disease.

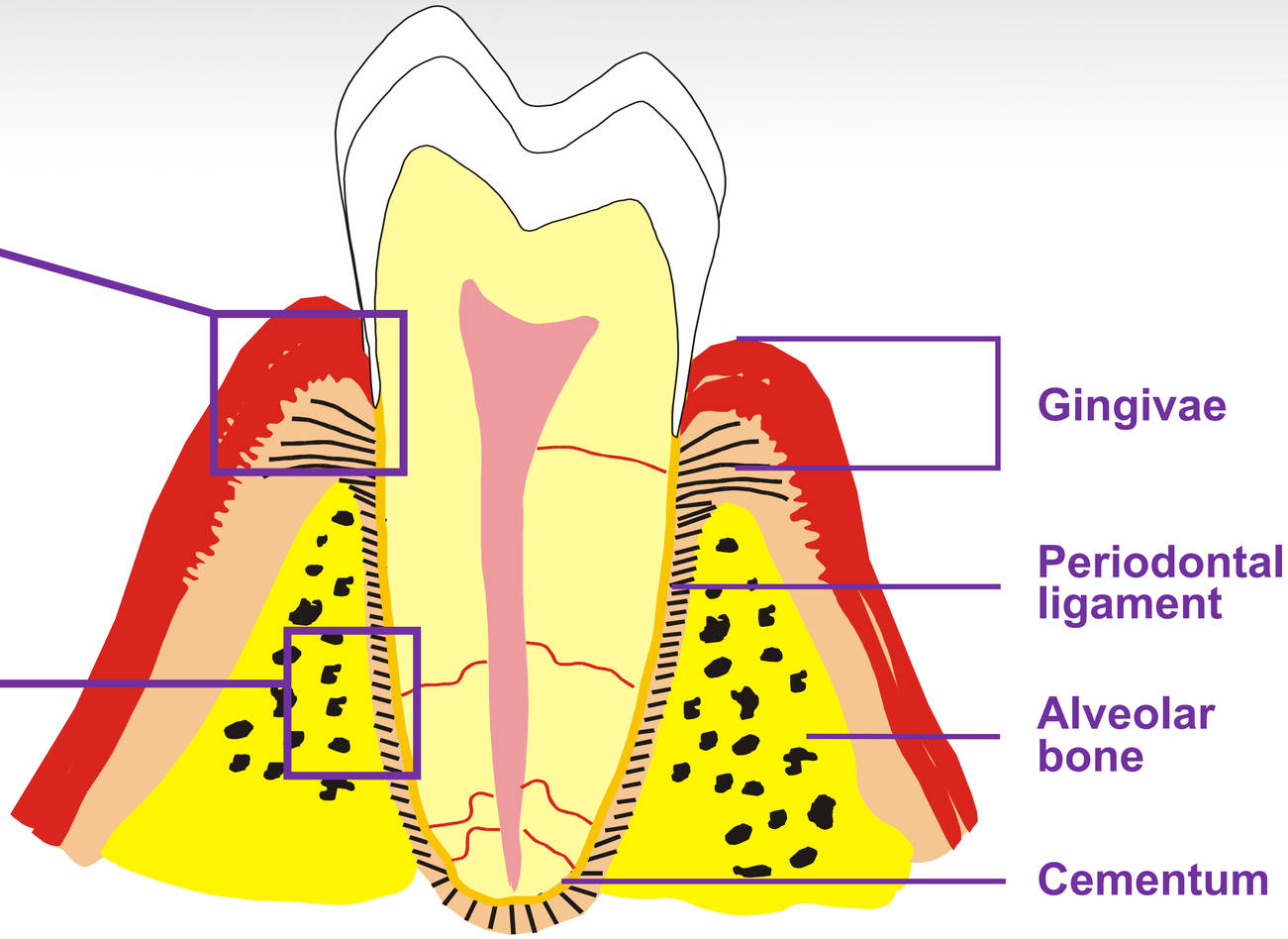
This is a complex definition: how do we create an objective set of measures for this?

Pristine health:
Does it exist? Is it natural?

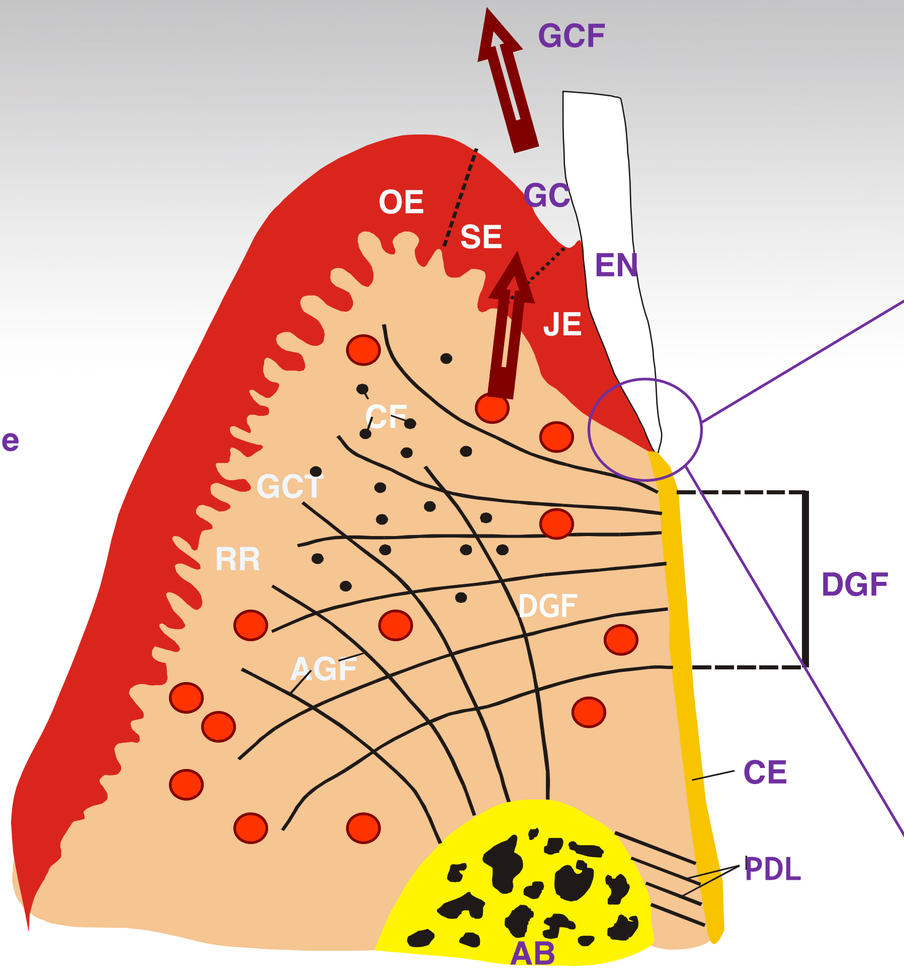
FREE GINGIVAE

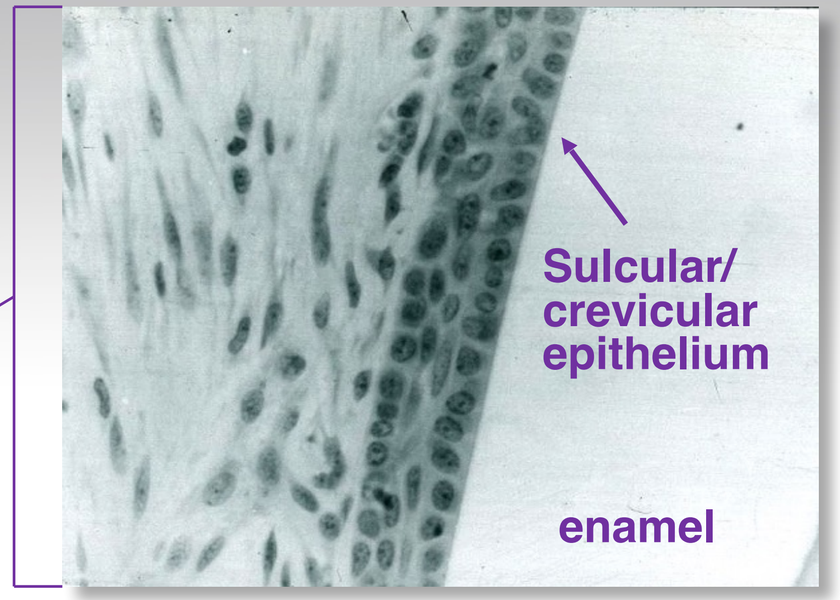


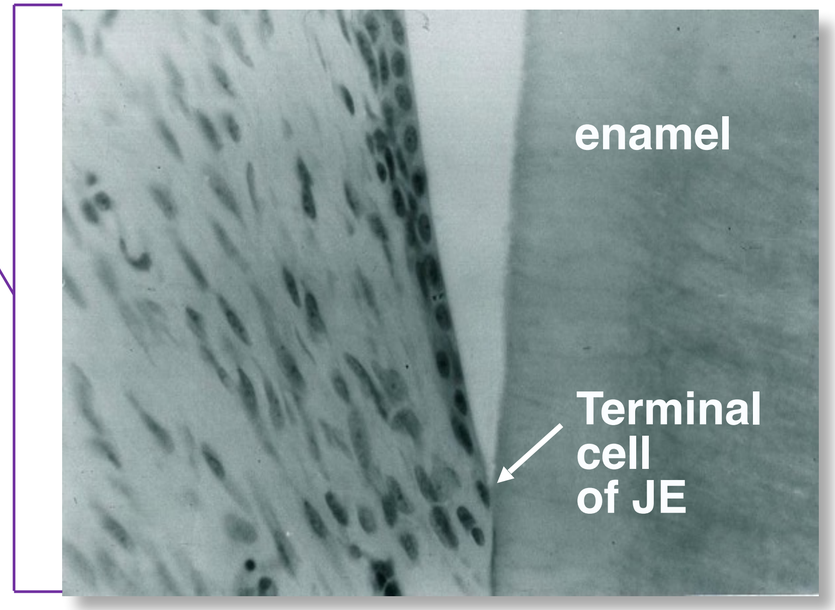


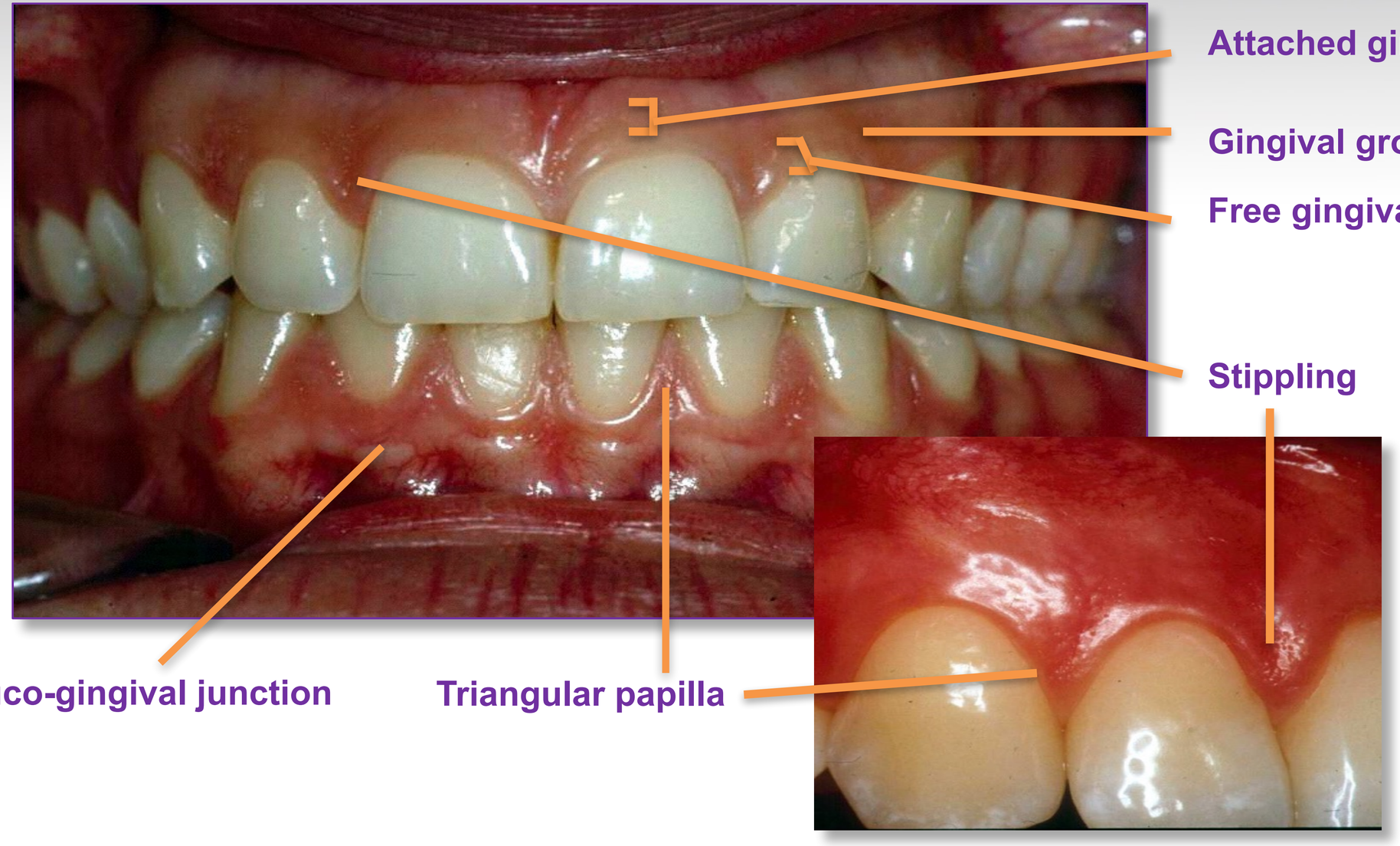


AB Alveolar bone
AGF Alveologingival fibres
CE Cementum
CF Circular fibres
DGF Dentogingival fibres
EN Enamel
EOE External oral epithelium
GCT Gingival connective tissue
GS Gingival sulcus
JE Junctional epithelium
SE Sulcular epithelium
RR Rete ridges
PL Principal fibres of periodontal ligament








Attached gingiva
Gingival groove
Free gingiva
Stippling
Muco-gingival junction
Triangular papilla

Changes in histology arise after only four days of plaque exposure

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2017 WORLD WORKSHOP

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Periodontal health

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The proceedings of the workshop were jointly and simultaneously published in the *Journal of Periodontology* and *Journal of Clinical Periodontology*.

Abstract

Objectives: To date there is a paucity of documentation regarding definitions of periodontal health. This review considers the histological and clinical determinants of periodontal health for both intact and reduced periodontium and seeks to propose appropriate definitions according to treatment outcomes.

Importance: Defining periodontal health is can serve as a vital common reference point for assessing disease and determining meaningful treatment outcomes.

Findings: The multifactorial nature of periodontitis is accepted, and it is recognized that restoration of periodontal health will be defined by an individual's response to treatment, taking into account allostatic conditions.

Conclusions: It is proposed that there are 4 levels of periodontal health, depending on the state of the periodontium (structurally and clinically sound or reduced) and the relative treatment outcomes: (1) pristine periodontal health, with a structurally sound and uninflamed periodontium; (2) well-maintained clinical periodontal health, with a structurally and clinically sound (intact) periodontium; (3) periodontal disease stability, with a reduced periodontium, and (4) periodontal disease remission/control, with a reduced periodontium.

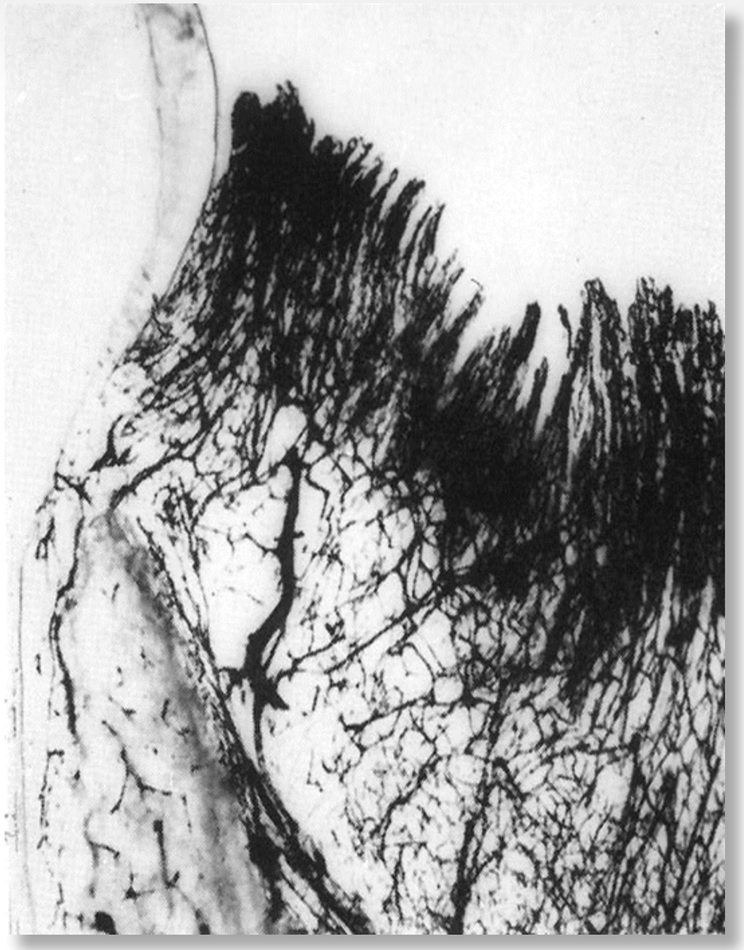
KEYWORDS
Clinical health, gingiva, periodontal remission, periodontal stability, pristine health

INTRODUCTION

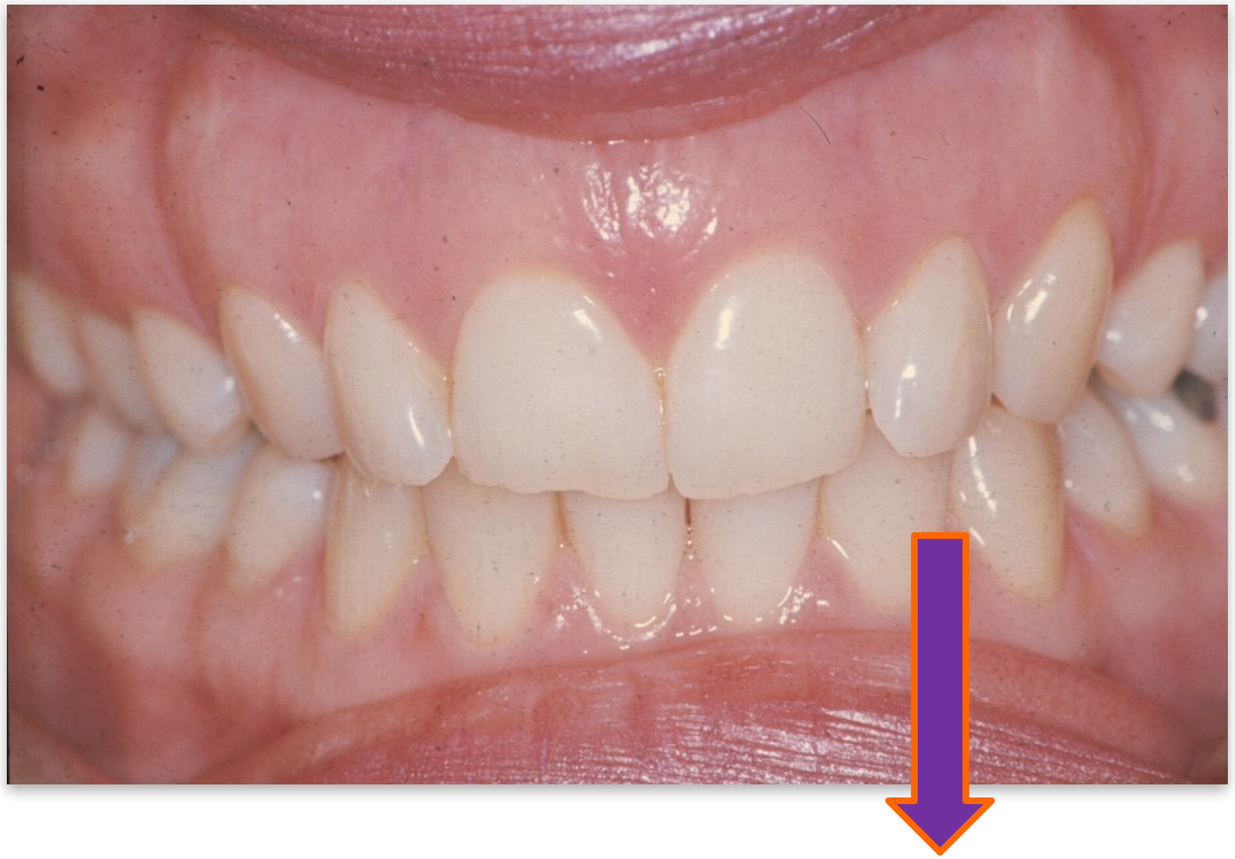
"Health is a state of complete physical, mental and social well-being and not merely the absence of disease or infirmity."[1] In accordance with this definition by the World Health Organization, periodontal health should be defined as a state free from inflammatory periodontal disease that allows an individual to function normally and not suffer any consequences (mental or physical) as a result of past disease. However, while this definition is holistic and patient-outcome based, it seems an impractical and limiting definition for the purposes of clinical management of periodontal diseases. Therefore, a more practical definition of periodontal health would be a state free from inflammatory periodontal disease. This, in turn, means that absence of inflammation associated with gingivitis or periodontitis, as assessed clinically, is a prerequisite for defining periodontal health.

It is a matter of debate if altered morphological conditions resulting from previous exposure to disease processes (eg. gingival recession, loss of attachment, and bone loss) may be redefined as novel healthy conditions in the absence of clinical signs and symptoms of inflammation.

Interestingly, there are almost no studies or reports attempting to define periodontal health.[2] Defining periodontal health is very important if we are to have a common reference point for assessing periodontal disease and determining meaningful treatment outcomes. Health can be evaluated at both the histological and clinical levels and should be considered in the context of a preventive starting point and a therapeutic end point. Thus, periodontal health can exist before disease commences but, conversely, periodontal health can be restored to an anatomically reduced periodontium. In this review, the clinical criteria for distinguishing pristine health from health on a reduced periodontium are presented and discussed.



What does clinical health look like?



Case *versus* site

Is clinical inflammation compatible with health?

Can there be a *site* of inflammation that is also a *case* of clinical health?

Periodontal health – conclusions

- Periodontal health is defined as:
  “An absence of clinically detectable inflammation.”
- “There is a biological level of immune surveillance consistent with clinical gingival health & homeostasis.”
- “Clinical health can be restored following treatment of gingivitis and periodontitis.”
- “A case of clinical health represents a different situation to a ‘site’ of clinical health.”

How do we classify clinical gingival health?

- **Clinical gingival health on an intact periodontium.**

  (An *intact* periodontium refers to an absence of detectable attachment and/or bone loss).

- **Clinical gingival health on a reduced periodontium:**

  - **Stable periodontitis patient (successful treatment);**
  - **Non-periodontitis patient (e.g. recession, crown-lengthening surgery).**

Expert paper 2: Plaque-induced gingival conditions

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2017 WORLD WORKSHOP

WILEY Journal of Clinical Periodontology

Dental plaque–induced gingival conditions

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The proceedings of the workshop were jointly and simultaneously published in the *Journal of Periodontology* and *Journal of Clinical Periodontology*.

Abstract

Objective: This review proposes revisions to the current classification system for gingival diseases and provides a rationale for how it differs from the 1999 classification system.

Importance: Gingival inflammation in response to bacterial plaque accumulation (microbial biofilms) is considered the key risk factor for the onset of periodontitis. Thus, control of gingival inflammation is essential for the primary prevention of periodontitis.

Findings: The clinical characteristics common to dental plaque–induced inflammatory gingival conditions include: a) clinical signs and symptoms of inflammation that are confined to the gingiva; b) reversibility of the inflammation by removing or disrupting the biofilm; c) the presence of a high bacterial plaque burden to initiate the inflammation; d) systemic modifying factors (e.g., hormones, systemic disorders, drugs) which can alter the severity of the plaque-induced inflammation and; e) stable (i.e., non-changing) attachment levels on a periodontium which may or may not have experienced a loss of attachment or alveolar bone. The simplified taxonomy of gingival conditions includes: 1) introduction of the term "incipient gingivitis;" 2) a description of the extent and severity of gingival inflammation; 3) a description of the extent and severity of gingival enlargement and; 4) a reduction of categories in the dental plaque–induced gingival disease taxonomy.

Conclusions: Dental plaque–induced gingival inflammation is modified by various systemic and oral factors. The appropriate intervention is crucial for the prevention of periodontitis.

KEYWORDS
diagnosis, evidence-based dentistry, gingivitis

Plaque-induced gingivitis may exhibit various patterns of observable signs and symptoms of inflammation that are localized to the gingiva and initiated by the accumulation of a microbial biofilm on teeth. Even when dental plaque biofilm levels are minimized, an inflammatory infiltrate is present within gingival tissues as part of a physiologic immune surveillance.[1] However, the initiation of gingivitis occurs if dental plaque accumulates over days or weeks without disruption or removal, due to a loss of symbiosis between the biofilm and the host's immune-inflammatory response, and development of an incipient dysbiosis (Figure 1). Various systemic factors, including endocrinopathies, hematologic conditions, diet, and drugs, can modify the immune-inflammatory response.[2,3]

Gingivitis associated with plaque and/or endogenous hormonal fluctuations, drugs, systemic diseases, and malnutrition, exhibit several essential characteristics. The universal features of these gingival conditions include: clinical signs and symptoms of inflammation that are confined to the free and attached gingiva and do not extend beyond the mucogingival junction; reversibility of the inflammation by disrupting/

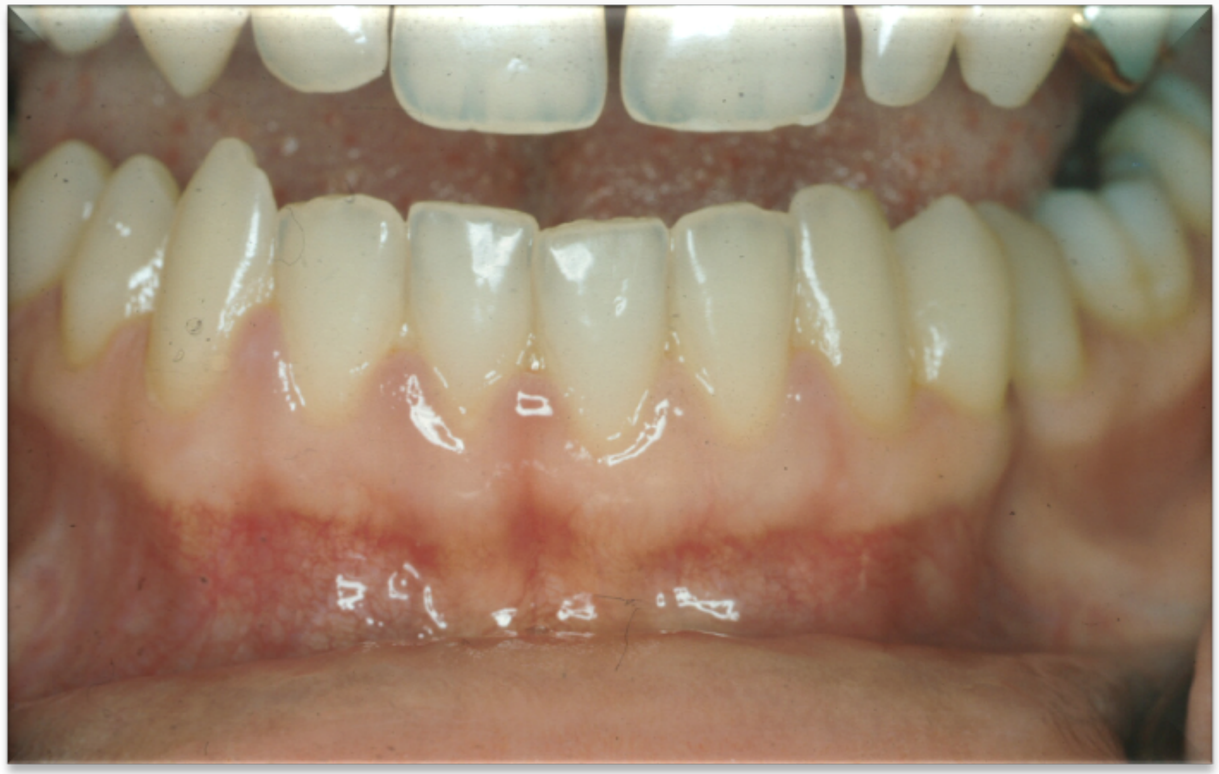
Critical questions:

- Define plaque-induced gingivitis on an intact & a reduced periodontium.
- Define a reduced periodontium (previous periodontitis vs non-periodontitis patient).
- What are the *predisposing* factors – local risk factors?
- What are the *modifying* factors – systemic risk factors?
- Can we reduce the number of categories?

Plaque-induced gingival conditions

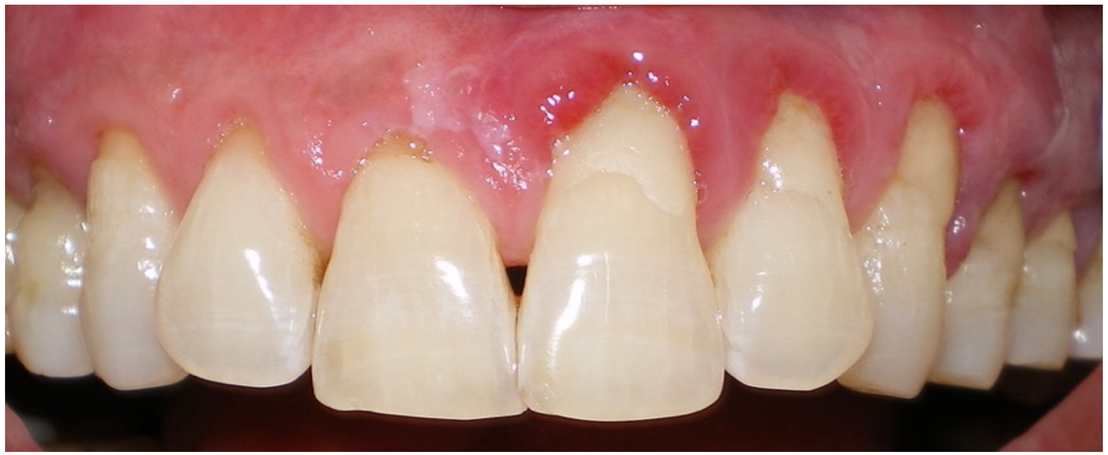
Is this a *case* of clinical health (with sites of gingival inflammation)?

Or is it a case of gingivitis?



Predisposing and modifying factors

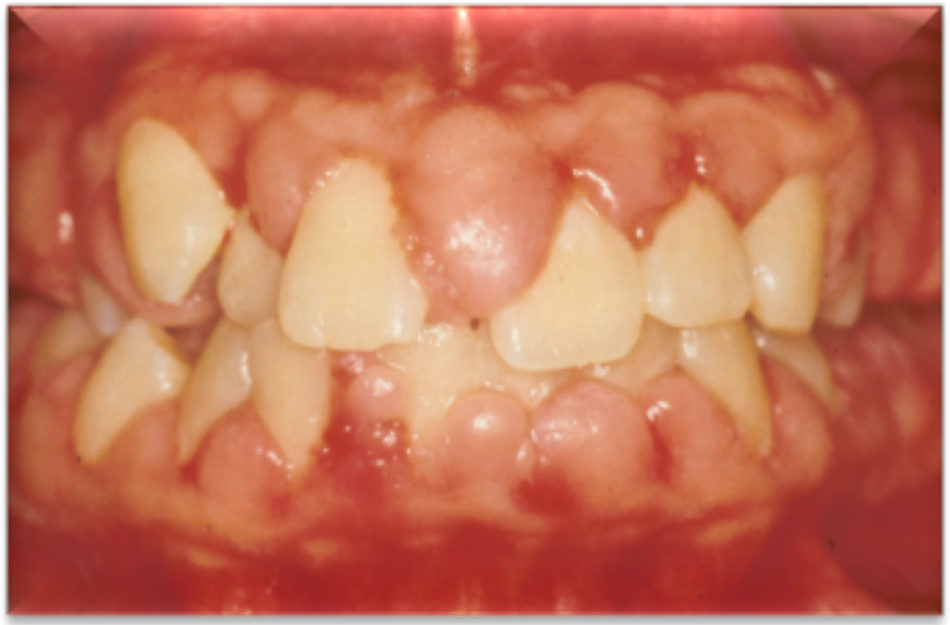
Local risk factors (*predisposing* factors)



- Dental plaque-biofilm retention factors:
  - Tooth anatomy
  - Restoration margins, etc.
- Oral dryness:
  - ↓ saliva flow
  - ↓ saliva quality
- e.g. Sjögren's, medications, mouth-breathing

Predisposing and modifying factors

Systemic risk factors (*modifying* factors)



- Smoking
- Metabolic factors (hyperglycemia)
- Nutritional factors (Vitamin C)
- Pharmacological agents
- ↑ sex steroids (puberty, pregnancy)
- Haematological conditions

Expert paper 3:
Non-plaque-induced gingival diseases

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2017 WORLD WORKSHOP

WILEY Journal of Clinical Periodontology

Non–plaque-induced gingival diseases

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The proceedings of the workshop were jointly and simultaneously published in the *Journal of Periodontology* and *Journal of Clinical Periodontology*.

Abstract

While plaque-induced gingivitis is one of the most common human inflammatory diseases, several non-plaque-induced gingival diseases are less common but often of major significance for patients. The non-plaque-induced gingival lesions are often manifestations of systemic conditions, but they may also represent pathologic changes limited to gingival tissues. A classification is proposed, based on the etiology of the lesions and includes: Genetic/Developmental disorders; Specific infections; Inflammatory and immune conditions and lesions; Reactive processes; Neoplasms; Endocrine, Nutritional and metabolic diseases; Traumatic lesions; and Gingival pigmentation.

KEYWORDS
classification, diagnosis oral, epulis, gingiva, gingival diseases, immunological, inflammation, mouth mucosa, oral manifestations, oral medicine, periodontal disease, rare diseases

Human gingiva as well as other oral tissues may exhibit several non-plaque-induced pathologic lesions, which may in some instances be manifestations of a systemic condition or a medical disorder. They may also represent pathologic changes limited to gingival tissues. Although these lesions are not directly caused by plaque, their clinical course may be impacted by plaque accumulation and subsequent gingival inflammation. Dentists are the key healthcare providers in establishing diagnoses and formulating treatment plans for patients affected by such lesions. Specialists in periodontology should be familiar with and be able to diagnose, treat, or refer for treatment any such lesion.

A review of non-plaque-induced gingival lesions was presented at the 1999 International Workshop for a Classification of Periodontal Diseases and Conditions,[1] and the present review aims to add available additional literature as well as diseases and conditions which were not included in the former review. Several of the diseases and their treatment have been reviewed recently.[2–4] The purpose of the current review is not to repeat the details of such texts, but to present a contemporary classification of the most relevant non-plaque-induced gingival diseases and conditions (Table 1) and to discuss briefly the more common of these. The major difference between the present classification proposal and that of the 1999 workshop is creation of a more comprehensive nomenclature and inclusion of ICD-10 diagnostic codes. Because some of the conditions seldom manifest in the oral cavity and some even more seldom present gingival manifestations, detailed appraisal is included within Table 2.

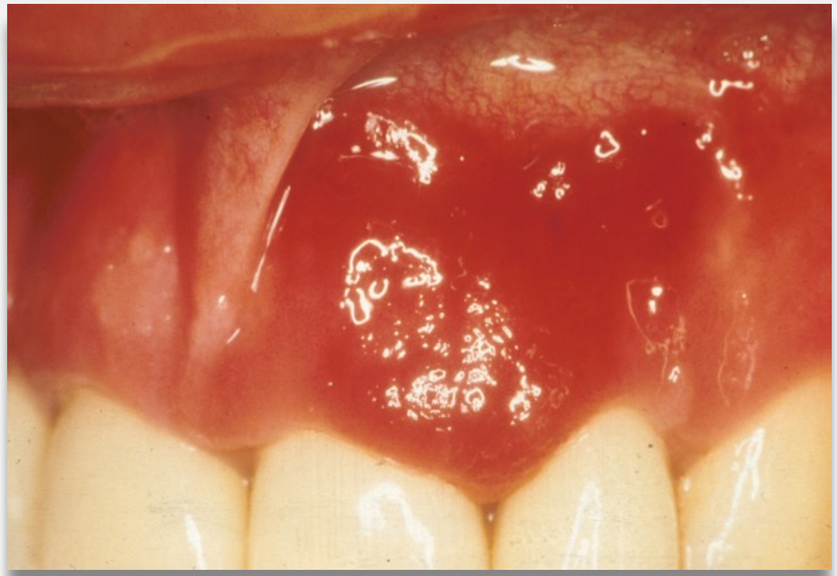
DESCRIPTION OF SELECTED DISEASE ENTITIES:

1 | GENETIC/DEVELOPMENTAL ABNORMALITIES

1.1 | Hereditary gingival fibromatosis (HGF)

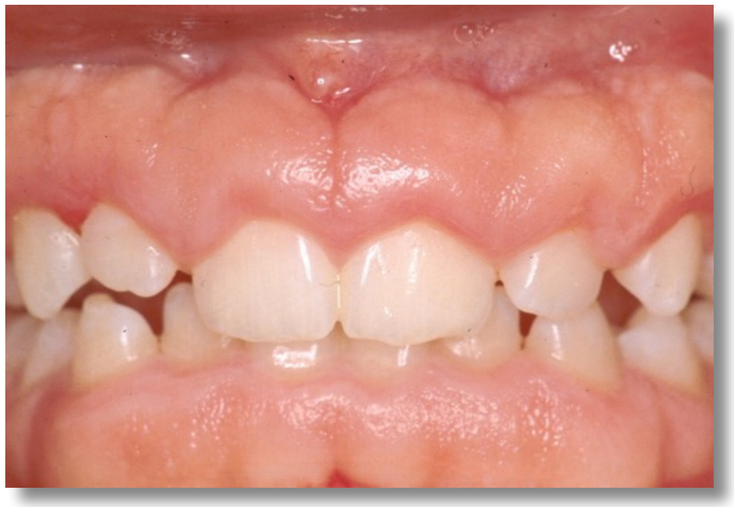
Clinically, gingival fibromatosis may present gingival overgrowth in various degrees. Compared to drug-related gingival overgrowth, hereditary gingival fibromatosis is a rare disease which may occur as

1. Genetic/developmental disorders.
2. Specific infections.
3. Inflammatory and immune conditions.
4. Reactive processes (epulides).
5. Neoplasms.
6. Endocrine, nutritional, and metabolic disorders.
7. Traumatic lesions.
8. Gingival pigmentation.

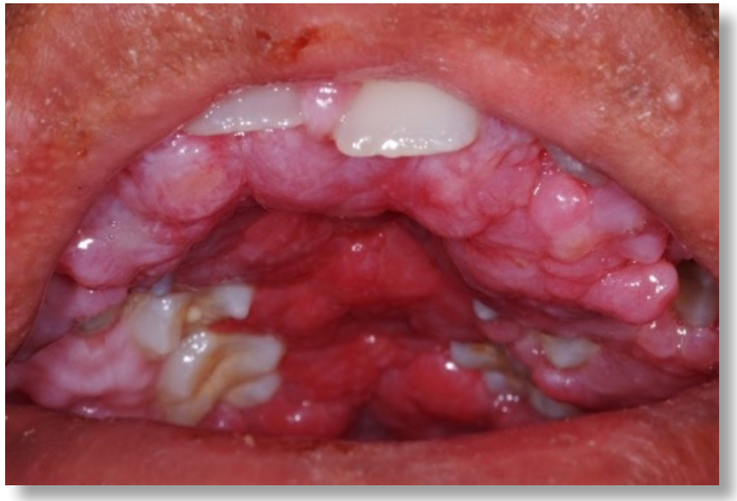


1. Genetic/developmental disorders

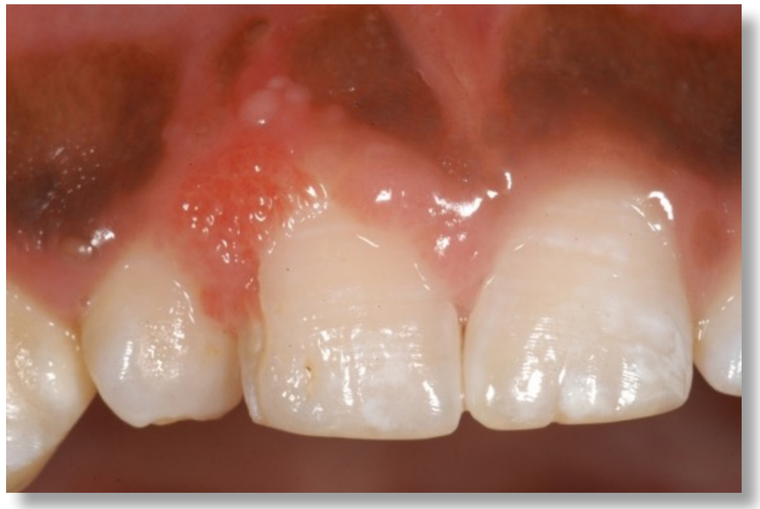
HGF



Juvenile hyaline fibromatosis

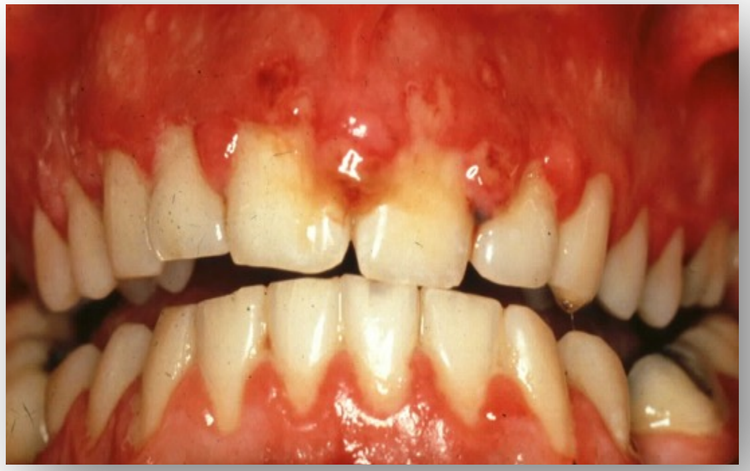


Sebaceous naevus of Jadassohn

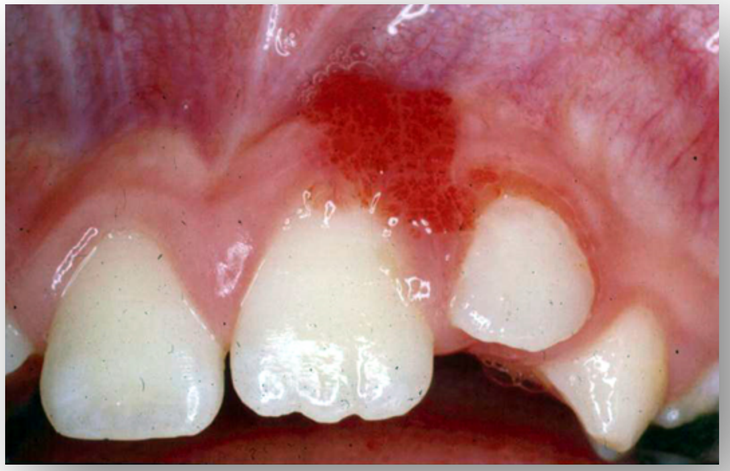


2. Specific infections

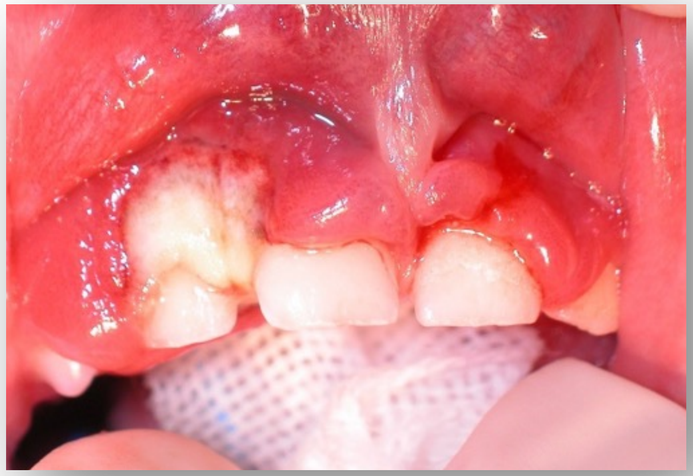
Gingival herpes simplex I



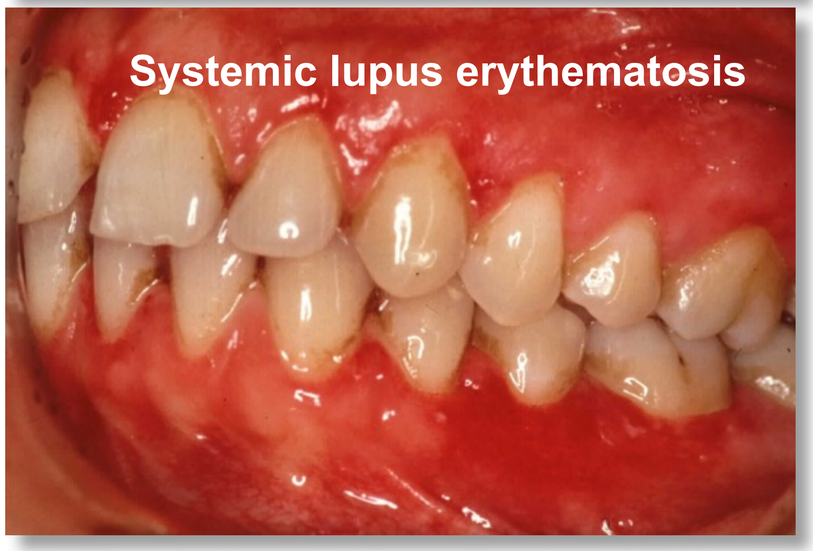
Molluscum contegeosum



Histoplasma capsulatum

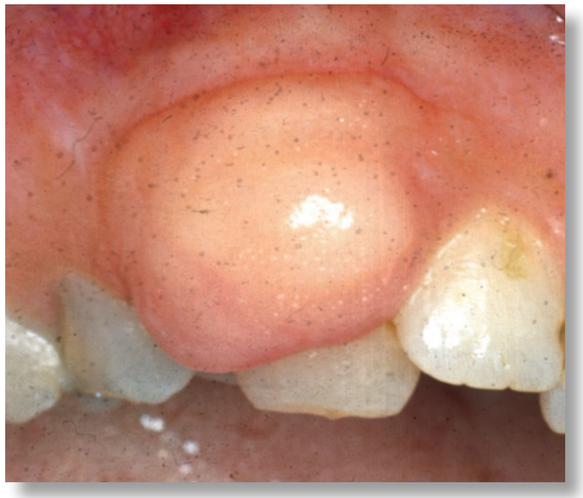


3. Inflammatory and immune conditions

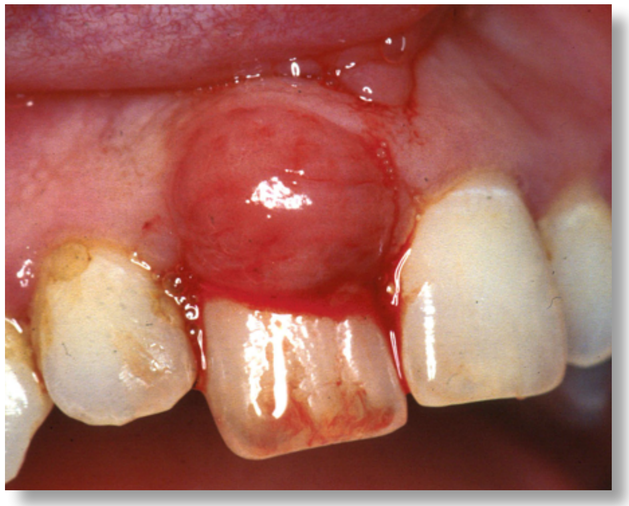


4. Reactive processes (epulides)

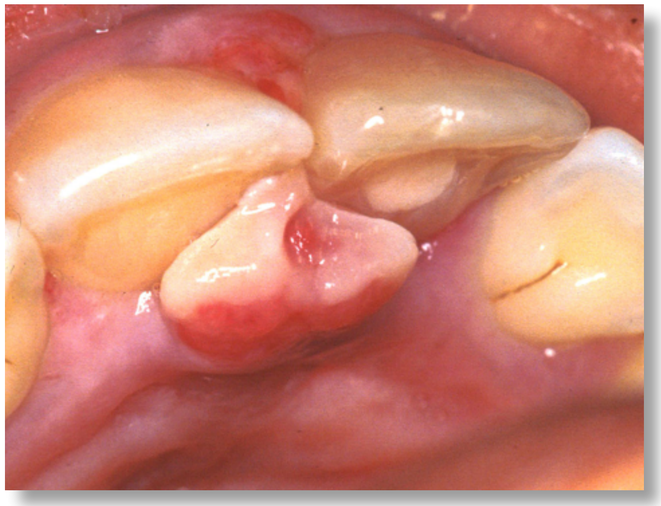
Fibrous epulis



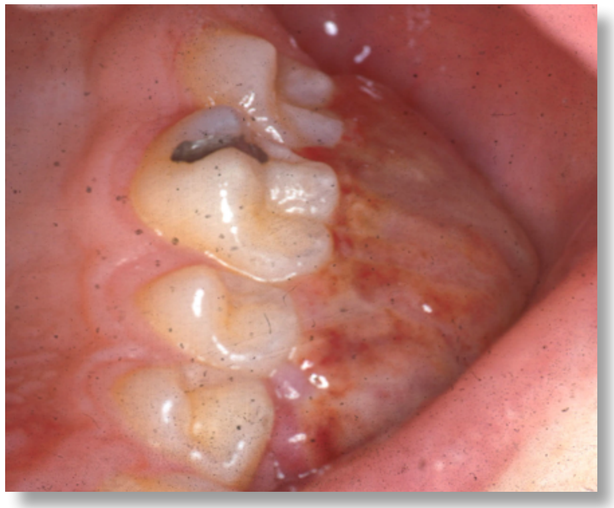
Vascular epulis



Pregnancy epulis

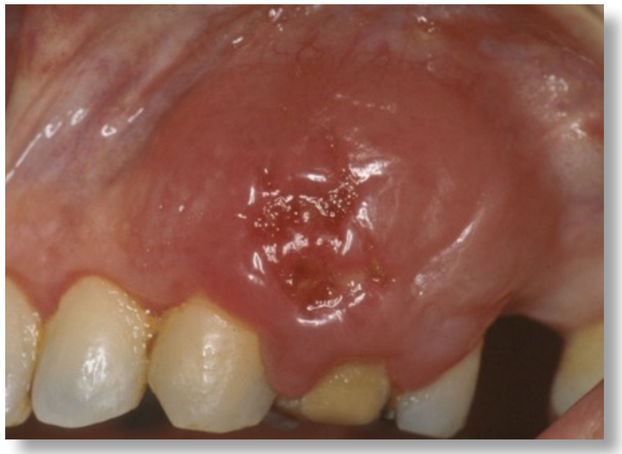


Peripheral giant cell granuloma

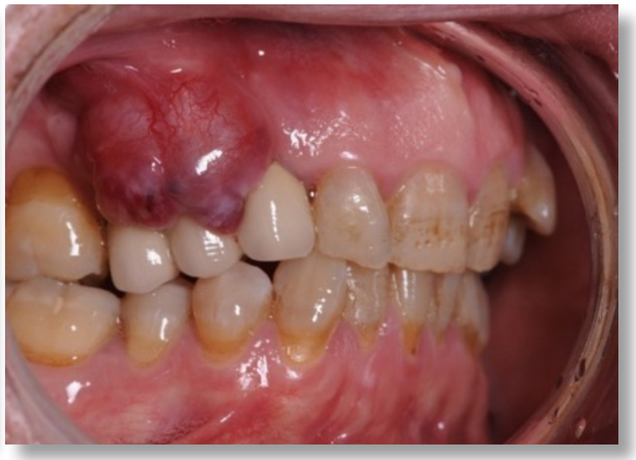


5. Neoplasms

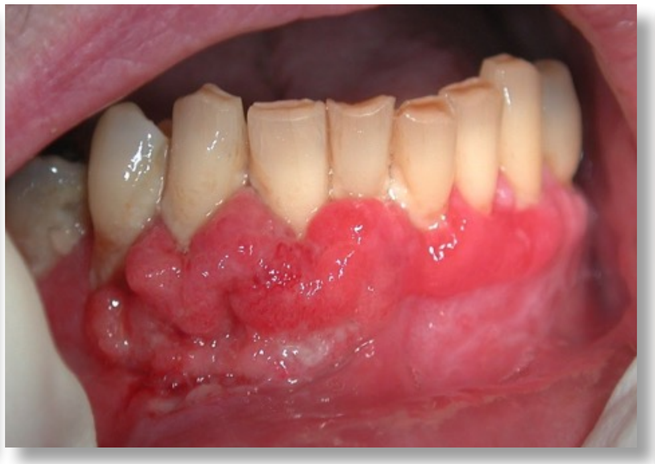
Non-Hodgkins lymphoma



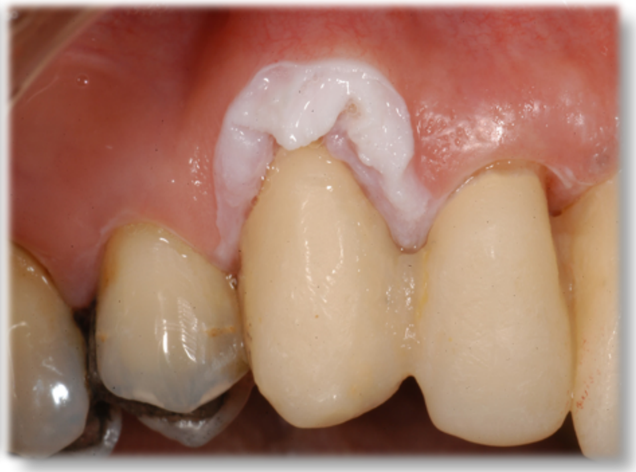
Chondrosarcoma



Squamous cell carcinoma

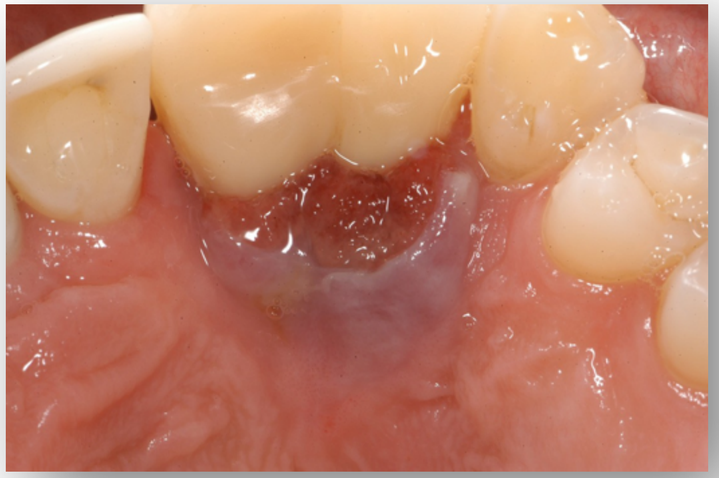


Proliferative verrucous leukoplakia

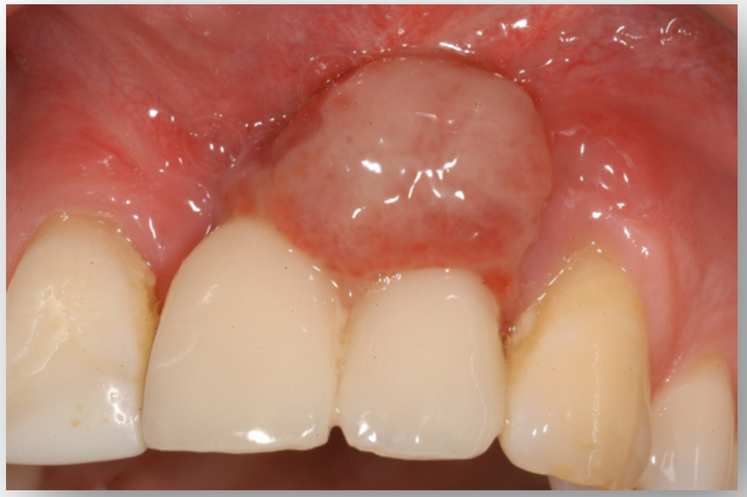


6. Endocrine, nutritional, and metabolic disorders

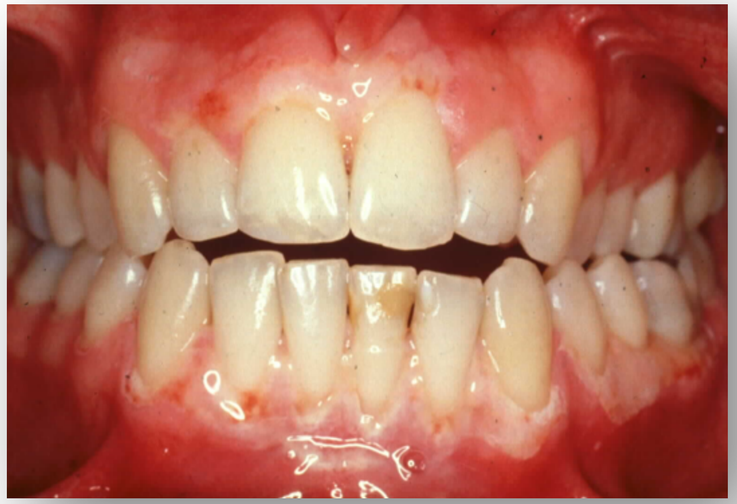
Giant cell tumour of bone



Giant cell tumour of bone

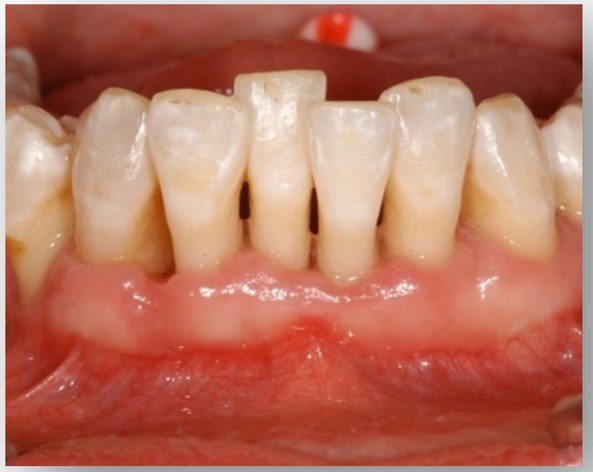


Scurvy – Vitamin C deficiency

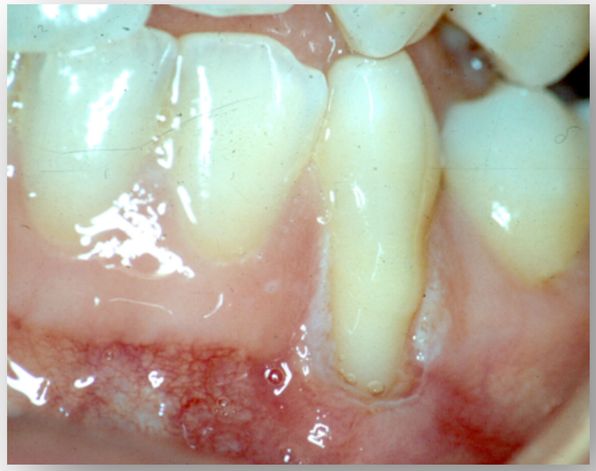


7. Traumatic lesions

Cocaine-induced necrosis

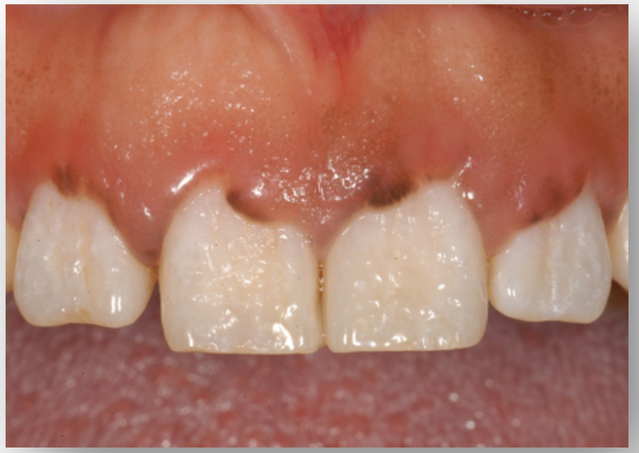


Gingivitis artefacta

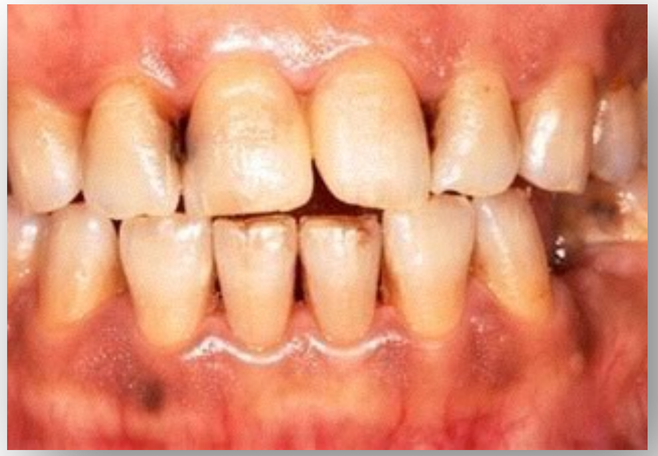


8. Gingival pigmentation

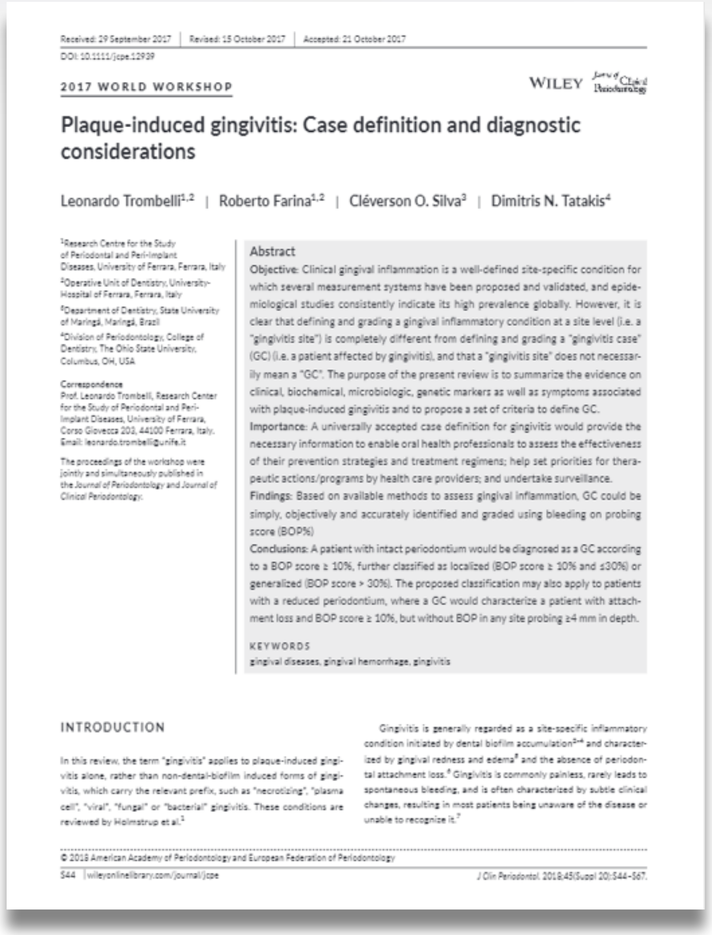
AZT pigmentation



Smoker’s melanosis



Expert paper 4: Plaque-induced gingivitis: Case definition and diagnostic considerations



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2017 WORLD WORKSHOP

WILEY Journal of Clinical Periodontology

Plaque-induced gingivitis: Case definition and diagnostic considerations

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The proceedings of the workshop were jointly and simultaneously published in the *Journal of Periodontology* and *Journal of Clinical Periodontology*.

Abstract

Objective: Clinical gingival inflammation is a well-defined site-specific condition for which several measurement systems have been proposed and validated, and epidemiological studies consistently indicate its high prevalence globally. However, it is clear that defining and grading a gingival inflammatory condition at a site level (i.e. a "gingivitis site") is completely different from defining and grading a "gingivitis case" (GC) (i.e. a patient affected by gingivitis), and that a "gingivitis site" does not necessarily mean a "GC". The purpose of the present review is to summarize the evidence on clinical, biochemical, microbiologic, genetic markers as well as symptoms associated with plaque-induced gingivitis and to propose a set of criteria to define GC.

Importance: A universally accepted case definition for gingivitis would provide the necessary information to enable oral health professionals to assess the effectiveness of their prevention strategies and treatment regimens; help set priorities for therapeutic actions/programs by health care providers; and undertake surveillance.

Findings: Based on available methods to assess gingival inflammation, GC could be simply, objectively and accurately identified and graded using bleeding on probing score (BOP%)

Conclusions: A patient with intact periodontium would be diagnosed as a GC according to a BOP score ≥ 10%, further classified as localized (BOP score ≥ 10% and ≤30%) or generalized (BOP score > 30%). The proposed classification may also apply to patients with a reduced periodontium, where a GC would characterize a patient with attachment loss and BOP score ≥ 10%, but without BOP in any site probing ≥4 mm in depth.

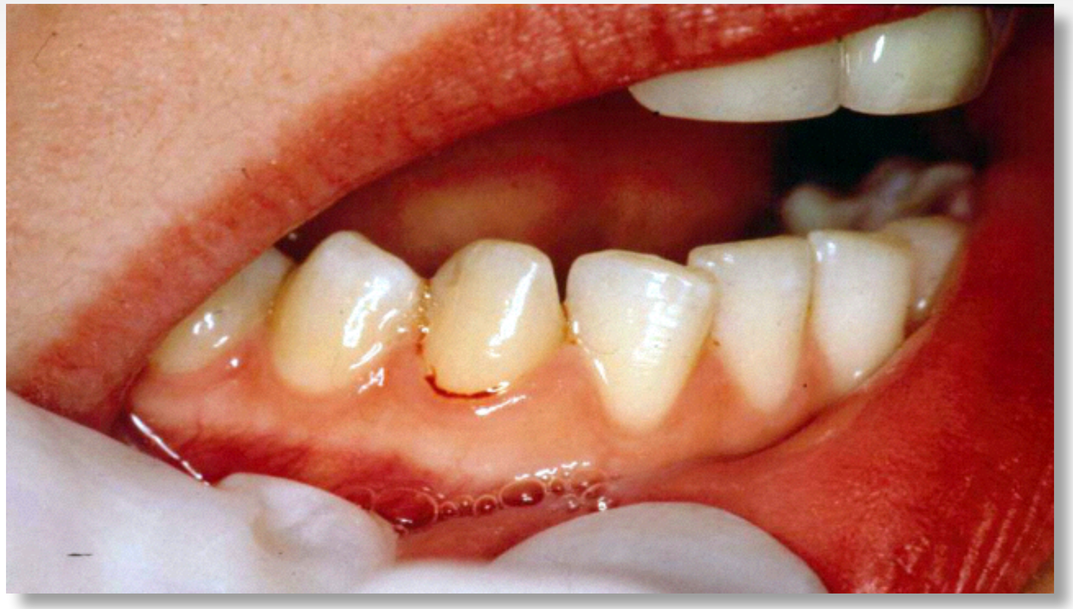
KEYWORDS
gingival diseases, gingival hemorrhage, gingivitis

INTRODUCTION

In this review, the term "gingivitis" applies to plaque-induced gingivitis alone, rather than non-dental-biofilm induced forms of gingivitis, which carry the relevant prefix, such as "necrotizing", "plasma cell", "viral", "fungal" or "bacterial" gingivitis. These conditions are reviewed by Holmstrup et al.[1]

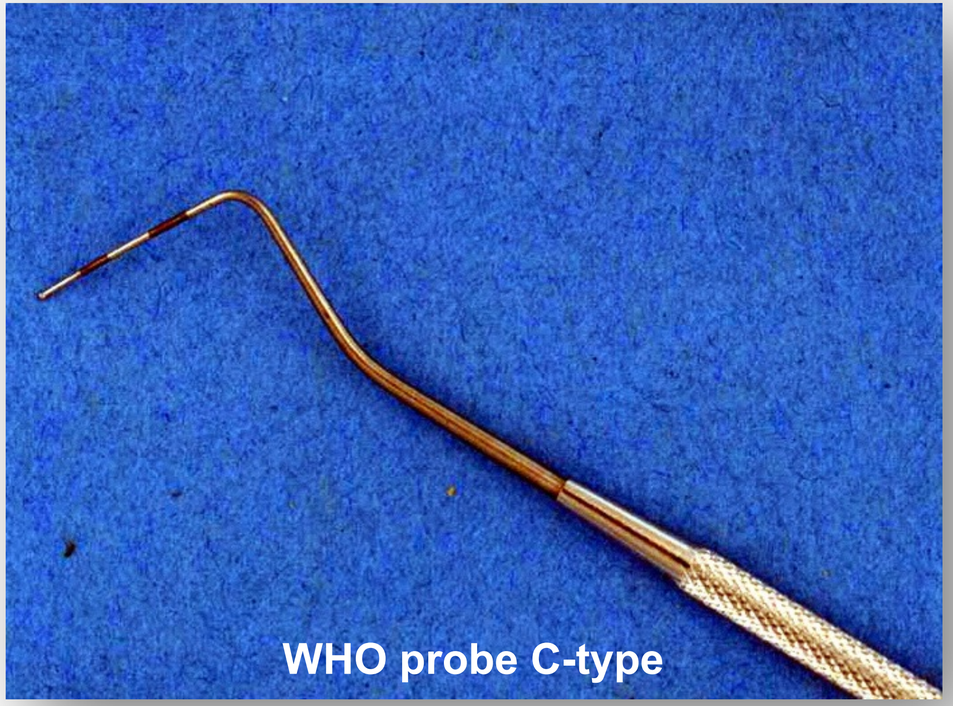
Gingivitis is generally regarded as a site-specific inflammatory condition initiated by dental biofilm accumulation[2–4] and characterized by gingival redness and edema[5] and the absence of periodontal attachment loss.[6] Gingivitis is commonly painless, rarely leads to spontaneous bleeding, and is often characterized by subtle clinical changes, resulting in most patients being unaware of the disease or unable to recognize it.[7]

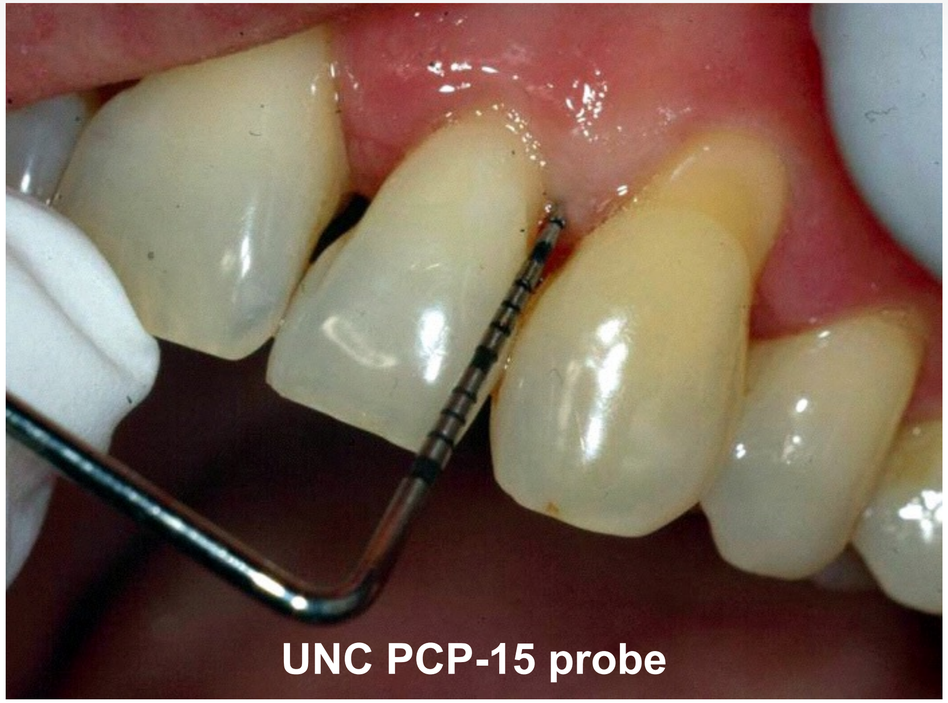
J Clin Periodontol. 2018;45(Suppl 20):S44–S67.



- **BoP = only objective measure**
- **We cannot measure severity**
- **So limited to extent (<10%, 30% rule)**

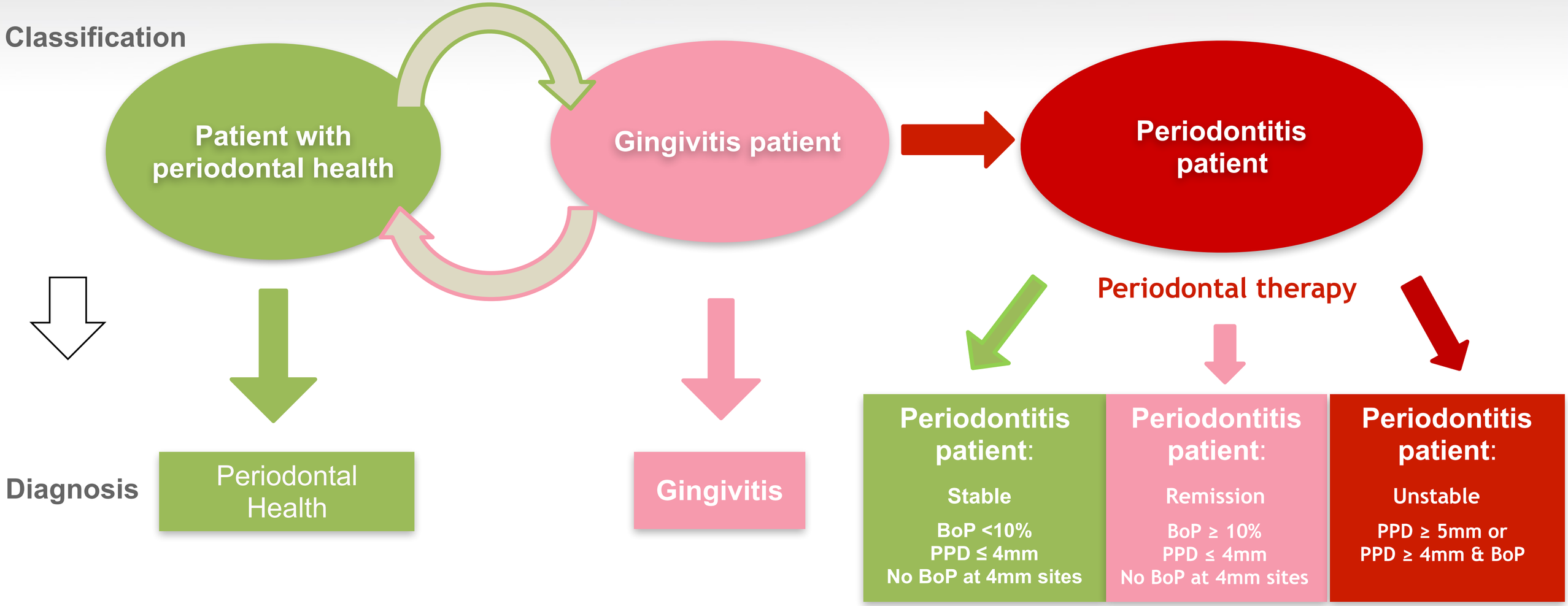
Periodontal probes:
There is a critical need for a new ISO standard


WHO probe C-type


UNC PCP-15 probe

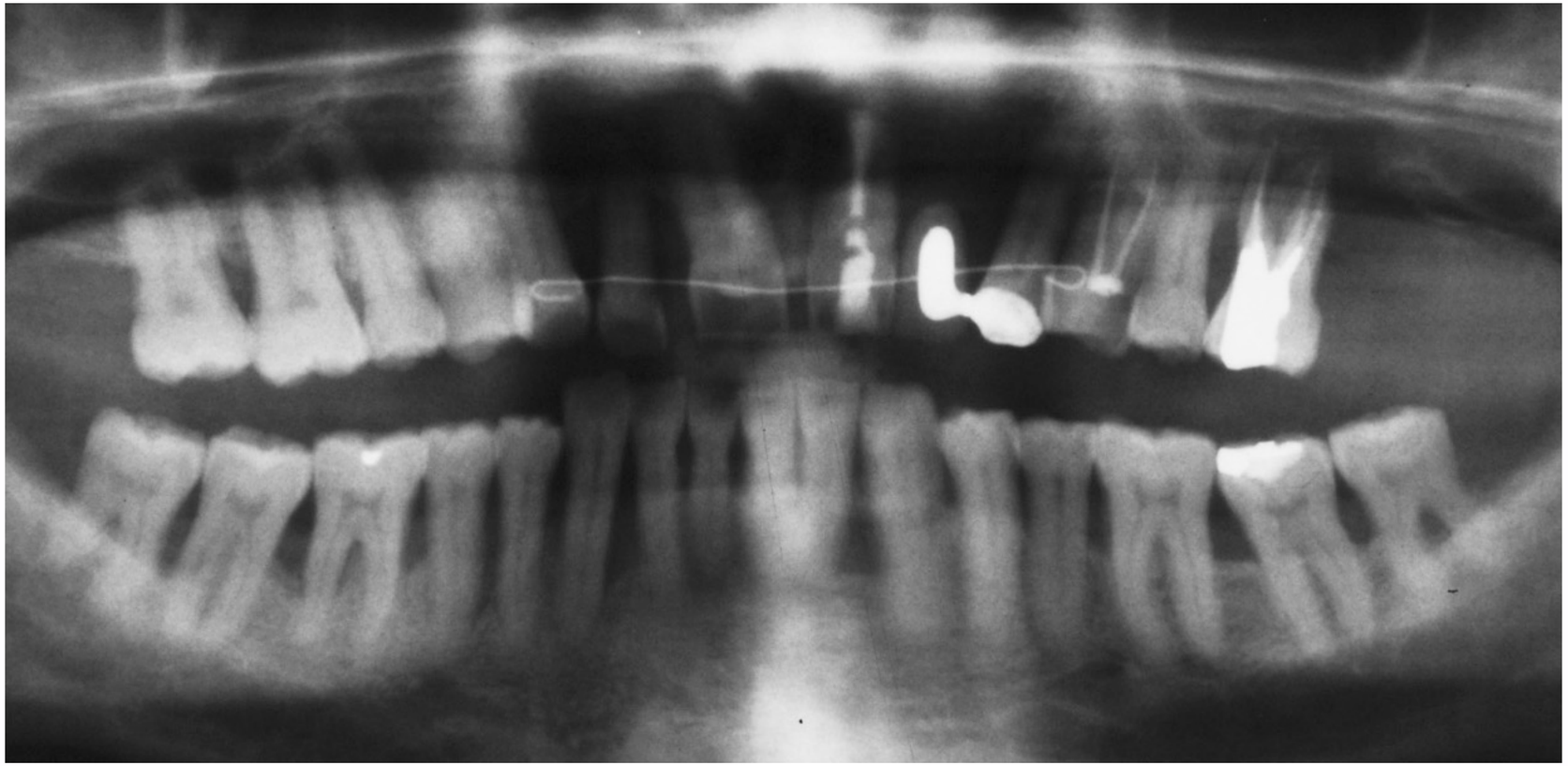
What is needed is a probe that provides “constant force” and goes to 15mm

Health and gingivitis on an intact periodontium and on a reduced periodontium: underpinning principles

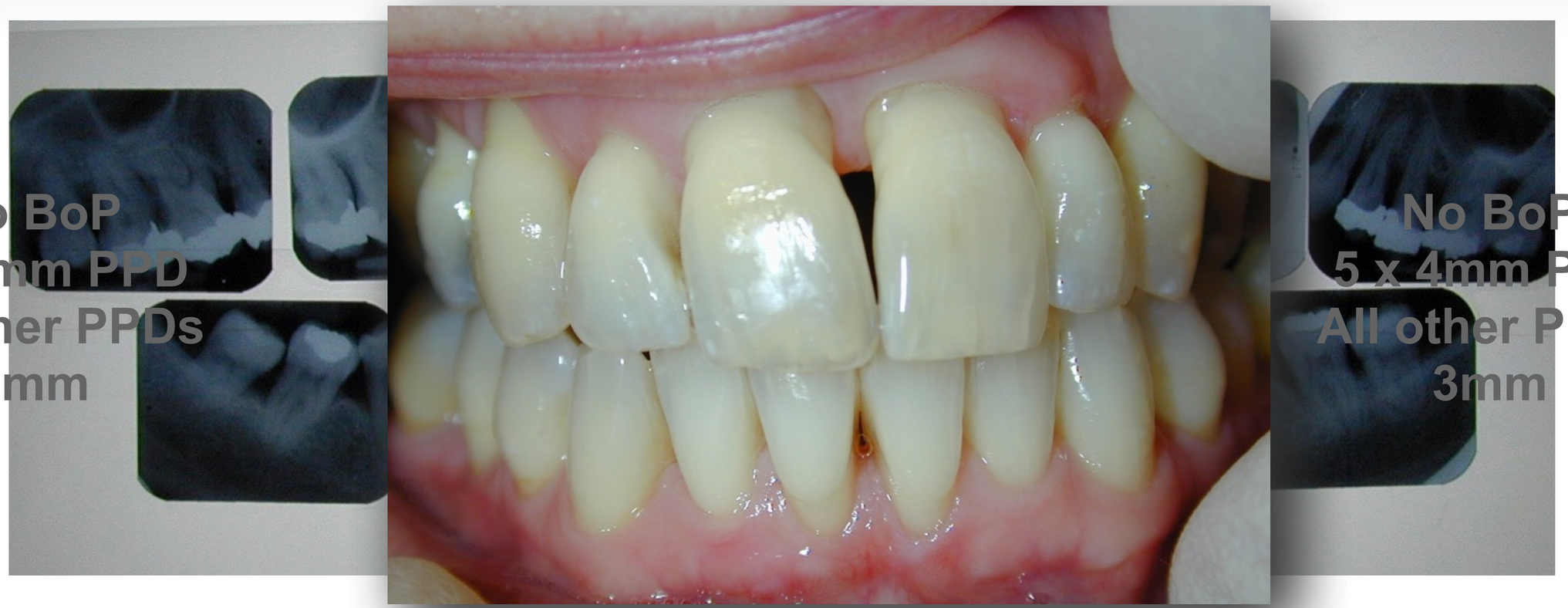


Once a periodontitis patient, always a periodontitis patient.
But such a patient *can* be a case of health.

March 2007



The 4mm non-bleeding site (closed pocket) represents health in a treated patient



No BoP
5 x 4mm PPD
All other PPDs
3mm

No BoP
5 x 4mm PPD
All other PPDs
3mm

2017 case definitions of health and gingivitis

Intact periodontium	Health	Gingivitis
Probing attachment loss	No	No
Probing pocket depths (assuming no pseudo pockets)	≤ 3mm	≤ 3mm
Bleeding on probing	<10%	Yes
Radiological bone loss	No	No
Reduced periodontium in a non-periodontitis patient	**Health**	**Gingivitis**
Probing attachment loss	Yes	Yes
Probing pocket depths *(assuming no pseudo pockets)*	≤ 3mm	≤ 3mm
Bleeding on probing	< 10%	Yes
Radiological bone loss	Possible	Possible
Successfully treated and stable periodontitis patient	**Health**	**Gingival inflammation**
Probing attachment loss	Yes	Yes
Probing pocket depths *(all sites & assuming no pseudo pockets)*	≤ 4mm (no site ≥4mm with BoP)	≤ 3mm
Bleeding on probing	< 10%	Yes (≥ 10%)
Radiological bone loss	Yes	Yes

EFP

New Classification

of periodontal and peri-implant diseases

EFP

European
Federation of
Periodontology

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