

Periodontal Phenotype: From Diagnosis to Personalized Treatment Planning—A Contemporary Review

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ABSTRACT

Background: The concept of periodontal phenotype has evolved from the traditional description of gingival biotype to a broader assessment incorporating gingival and underlying osseous characteristics. It is increasingly relevant to periodontal, mucogingival, orthodontic, restorative, and implant treatment planning.

Objective: This review critically examines the contemporary concept of periodontal phenotype, methods of assessment, clinical significance, phenotype modification, and its role in personalized treatment planning.

Materials and methods: Contemporary consensus documents, systematic reviews, meta-analyses, and clinically relevant studies concerning periodontal phenotype, gingival thickness, keratinized tissue, bone morphology, periodontal plastic surgery, orthodontics, restorative dentistry, and implant therapy were considered, with emphasis on recent evidence and landmark publications.

Results: Periodontal phenotype is best considered a multidimensional anatomical characteristic comprising gingival thickness, keratinized tissue width, and underlying bone morphology. It varies between individuals and intraoral sites. Thin phenotypes are associated with greater susceptibility to gingival recession and may increase anatomical vulnerability during orthodontic movement and selected surgical procedures; however, phenotype alone does not establish periodontal disease risk or determine treatment outcome. Direct clinical measurement remains important, while ultrasound, CBCT, intraoral scanning, and multimodal digital technologies offer increasingly sophisticated assessment. Evidence supports soft-tissue augmentation for selected indications, but routine modification of every thin phenotype is not justified. Around implants, augmentation can increase tissue thickness and may improve recession and esthetic outcomes, although its influence on peri-implant disease prevention remains uncertain.

Conclusions: Periodontal phenotype should be incorporated into comprehensive, site-specific risk assessment rather than used as an isolated predictor. Phenotype assessment can identify anatomical limitations, guide treatment selection, determine the need for tissue augmentation, and facilitate individualized monitoring. Future research should standardize definitions and measurement methods and establish validated relationships between phenotype and clinically meaningful long-term outcomes.

Keywords: Periodontal phenotype; Gingival thickness; Keratinized tissue; Gingival recession; Periodontal plastic surgery; Dental implants; Personalized dentistry; Bone morphotype.

INTRODUCTION

Periodontal tissues exhibit substantial anatomical variation, historically described using the concepts of “thin” and “thick” gingival biotypes. Although clinically useful, the term **biotype** suggests a relatively fixed biological category and does not adequately encompass the multiple anatomical factors influencing periodontal tissue behavior. Contemporary terminology therefore favors **gingival phenotype** and, more broadly, **periodontal phenotype**.

The 2017 World Workshop incorporated gingival phenotype into the classification of mucogingival conditions and promoted assessment of gingival thickness and keratinized tissue width. The broader periodontal phenotype additionally considers the underlying osseous architecture. Thus, phenotype is no longer simply a visual description of gingival thickness but a multidimensional anatomical characteristic involving soft tissues, bone, tooth morphology, and tooth position.

This concept has particular relevance to periodontal plastic surgery, orthodontics, restorative dentistry, and implant therapy. A thin gingival phenotype may increase susceptibility to recession, while thin facial bone and unfavorable tooth position can further increase anatomical vulnerability. Similarly, peri-implant soft-tissue thickness and facial bone morphology may influence esthetic stability.

However, phenotype should not be confused with periodontal disease risk. Periodontitis is multifactorial, and biofilm, smoking, systemic conditions, oral hygiene, previous disease, and patient-related factors remain fundamental determinants of disease development and progression.

The clinical value of phenotype therefore lies primarily in **risk stratification and treatment planning**. The relevant question is not whether a thin phenotype is inherently pathological, but whether its anatomical characteristics create a meaningful risk for a particular intervention.

This review discusses the evolution and components of periodontal phenotype, methods of assessment, biological and clinical significance, phenotype modification, and its application to individualized periodontal, orthodontic, restorative, and implant treatment planning.

EVOLUTION AND DEFINITION OF PERIODONTAL PHENOTYPE

Gingival biotype and gingival phenotype

Early classifications differentiated periodontal tissues into thin-scalloped and thick-flat patterns based on gingival morphology, crown form, and scalloping. De Rouck et al. subsequently proposed a clinically useful classification incorporating gingival thickness, keratinized tissue width, gingival scalloping, and tooth morphology.

The term **gingival phenotype** is preferred because the observable tissue characteristics can be influenced by anatomical and environmental factors rather than representing a fixed biological “type.” Gingival phenotype is principally characterized by **gingival thickness (GT)** and **keratinized tissue width (KTW)**.

Importantly, GT and KTW are not interchangeable. A site may have a broad band of keratinized tissue but remain thin, or have relatively thick tissue despite a narrower keratinized zone. Consequently, binary classification alone may oversimplify clinically relevant anatomy.

Periodontal phenotype

The periodontal phenotype extends beyond gingival characteristics and incorporates:

- gingival thickness;
- keratinized tissue width;
- facial/buccal bone thickness;
- tooth dimensions and morphology;
- root prominence;
- tooth position and inclination.

This multidimensional approach is particularly relevant because soft- and hard-tissue anatomy interact. A thin gingiva may be clinically more vulnerable when accompanied by a thin facial bone plate, prominent root, or tooth positioned close to the alveolar boundary.

Phenotype should therefore be distinguished from **periodontal risk**. It describes anatomy; risk represents the probability of an adverse clinical outcome based on the interaction of anatomy, disease, patient characteristics, and treatment.

The American Academy of Periodontology best evidence review found greater recession prevalence and severity in patients with thin and narrow gingiva but found insufficient evidence to recommend routine conversion of healthy thin tissues into thick tissues. Thus, thin phenotype should not be considered pathological in itself.

COMPONENTS AND DETERMINANTS OF PERIODONTAL PHENOTYPE

Gingival thickness

GT is an important component of periodontal phenotype and varies considerably between individuals, teeth, and sites. Thin tissue generally provides less soft-tissue volume and may be more susceptible to mechanical and surgical trauma. Nevertheless, no universal numerical threshold reliably separates “safe” from “unsafe” tissue.

Keratinized tissue width

KTW represents the apicocoronal dimension of keratinized gingiva. Its relationship with periodontal stability is complex. Narrow KTW does not automatically indicate disease or future recession, particularly when plaque control is excellent. It should therefore be considered together with tissue thickness, inflammation, recession, tooth position, and patient-related factors.

Supracrestal tissue attachment

The contemporary term **supracrestal tissue attachment** replaces “biologic width” and includes the junctional epithelium and supracrestal connective tissue attachment. Restoration margins that encroach on this tissue may promote inflammation and periodontal tissue loss.

Osseous architecture

Facial bone thickness and the presence of dehiscence or fenestration are important determinants of the periodontal envelope. Thin facial bone may increase the vulnerability of periodontal tissues during tooth movement or surgical procedures.

Tooth morphology and position

Crown dimensions, root prominence, tooth inclination, and proximity to the alveolar boundary influence periodontal architecture. Tooth position may therefore be more clinically relevant than gingival thickness alone.

Patient- and site-related determinants

Phenotype varies according to age, sex, tooth type, arch location, and local anatomical factors. Previous orthodontic treatment, malposition, trauma, and existing recession may further modify clinical risk. This supports **site-specific rather than generalized phenotype assessment**.

ASSESSMENT OF PERIODONTAL PHENOTYPE

No single assessment method is ideal for every clinical situation.

Visual assessment

Visual examination considers gingival contour, scalloping, tissue color, translucency, and crown morphology. It is rapid and non-invasive but subjective and unable to quantify tissue thickness reliably.

Probe transparency

The transparency method involves determining whether a periodontal probe is visible through the gingival margin. It is simple and inexpensive but provides categorical rather than continuous measurements and may be influenced by tissue pigmentation, inflammation, and probe characteristics.

Transgingival probing

Direct measurement under local anesthesia provides quantitative GT measurements and remains an important reference method. Its disadvantages are invasiveness, discomfort, and potential measurement error from probe angulation or tissue compression.

Ultrasonography

Ultrasound offers a non-invasive method of measuring gingival thickness without ionizing radiation. Systematic evidence suggests potential usefulness for soft-tissue assessment, although performance varies between sites and standardization of equipment and protocols remains necessary.

CBCT

CBCT is particularly useful for assessing facial bone thickness, root position, dehiscence, and fenestration. It should not be prescribed solely for routine gingival thickness measurement because of radiation exposure. However, when CBCT is already justified—for example, for complex implant or orthodontic planning—it provides valuable information about the hard-tissue phenotype.

Digital technologies

Intraoral scanning permits three-dimensional documentation of gingival contours and longitudinal monitoring of recession and tissue-volume changes. Combining IOS with CBCT may provide a more comprehensive representation of hard- and soft-tissue anatomy.

Artificial intelligence may eventually automate phenotype mapping and integrate photographs, scans, CBCT data, and periodontal records. However, technical accuracy does not necessarily demonstrate clinical predictive value.

Table 1. Methods for periodontal phenotype assessment

Method	Main parameter	Invasiveness	Advantages	Limitations	Clinical use
Visual examination	General morphology	None	Rapid, inexpensive	Subjective	Screening
Probe transparency	Approximate GT	None	Simple	Categorical	Routine assessment
Transgingival probing	GT	Minimally invasive	Quantitative	Invasive	Surgical planning
Ultrasound	GT/soft tissues	None	No radiation	Equipment/operator dependent	Emerging
CBCT	Bone thickness	Radiation	3D hard-tissue assessment	Radiation; limited soft-tissue contrast	Implant/orthodontic planning
Intraoral scanning	Surface morphology	None	Digital and repeatable	Does not directly visualize bone	Monitoring
Multimodal imaging	Soft + hard tissues	Variable	Comprehensive	Cost/complexity	Advanced planning

BIOLOGICAL AND CLINICAL SIGNIFICANCE

Gingival recession

The association between thin gingival tissues and recession is one of the most consistently reported phenotype relationships. However, recession is multifactorial. Tooth position, facial bone morphology, traumatic toothbrushing, inflammation, orthodontic movement, frenal factors, smoking, and pre-existing recession can all contribute. Therefore, thin phenotype should be considered a **risk modifier rather than an independent cause**.

Periodontal inflammation and disease

Phenotype may influence the clinical appearance of inflammation, while inflammation itself can alter tissue dimensions through edema. Consequently, phenotype assessment is ideally performed after inflammation has been controlled.

Current evidence does not demonstrate that thin phenotype independently causes periodontitis. Disease diagnosis and risk assessment must continue to focus on established etiological and modifying factors.

Orthodontic treatment

Orthodontic movement, particularly labial movement or proclination of mandibular incisors, may challenge the limits of the alveolar housing. Patients with thin gingiva combined with thin facial bone or unfavorable tooth position may be more vulnerable to recession.

Systematic reviews have identified periodontal phenotype, tooth movement, periodontal status, and site-specific anatomy as potential risk factors, although heterogeneity prevents definitive conclusions regarding causality. Phenotype assessment may therefore be particularly useful before extensive tooth movement.

Restorative dentistry

Subgingival restorations must respect supracrestal tissue attachment and permit adequate plaque control. Tissue phenotype, restorative margin position, crown contour, and emergence profile should be considered together. A thin phenotype may provide less tolerance for unfavorable restorative contours, but excellent prosthetic design and maintenance remain essential regardless of phenotype.

PERIODONTAL PHENOTYPE IN PERIODONTAL PLASTIC SURGERY

Gingival recession treatment

Phenotype assessment is particularly relevant when treating gingival recession. Prognosis depends strongly on recession morphology and interproximal attachment, but tissue thickness can influence surgical planning and postoperative stability.

Connective tissue grafting remains an established method for increasing soft-tissue thickness and can be combined with coronally advanced flap procedures when tissue augmentation is desirable.

Evidence does not indicate that thin phenotype makes root coverage impossible. Systematic reviews have reported high rates of root coverage in both thin and thick tissues, emphasizing that phenotype is only one component of treatment predictability.

Connective tissue grafting

Connective tissue grafts increase tissue thickness and can improve long-term marginal tissue stability. Network meta-analyses demonstrate that connective tissue grafts and several substitutes increase gingival thickness compared with flap procedures alone.

The rationale for augmentation is therefore strongest when increased tissue volume has a specific clinical objective—for example, existing recession, inadequate tissue stability, or an anticipated anatomical challenge.

Phenotype modification

The AAP best evidence review found insufficient evidence to recommend routine conversion of a healthy thin phenotype into a thick phenotype. Phenotype modification should consequently be **indication-driven rather than classification-driven**.

The distinction is clinically important:

Thin phenotype ≠ automatic indication for surgery.

Instead:

Thin phenotype + identified clinical risk = consideration of phenotype modification.

PERIODONTAL PHENOTYPE AND IMPLANT DENTISTRY

Peri-implant soft-tissue phenotype

Peri-implant phenotype includes mucosal thickness, keratinized mucosa, and peri-implant soft-tissue architecture. Although conceptually related to periodontal phenotype, peri-implant tissues differ biologically from periodontal tissues and require implant-specific interpretation.

Facial bone and implant placement

Thin facial bone can undergo substantial remodeling after extraction and implant placement. When combined with a thin soft-tissue phenotype, the risk of facial contour alteration and mucosal recession may be greater.

This is particularly important in the esthetic zone.

Immediate implants

Immediate implant placement does not eliminate post-extraction remodeling. Implant position, facial bone thickness, soft-tissue phenotype, grafting, and provisionalization must therefore be considered together.

Soft-tissue augmentation can increase tissue thickness and may reduce facial recession in selected cases. The decision should be based on the patient's esthetic risk profile rather than phenotype alone.

Implant esthetics

Midfacial recession, tissue transparency, papillary deficiencies, and facial contour changes are influenced by implant position, facial bone morphology, soft-tissue thickness, restorative emergence profile, and surgical technique.

Systematic evidence indicates that soft-tissue augmentation increases peri-implant tissue thickness and may reduce mucosal recession, but evidence for prevention of peri-implantitis or marginal bone loss remains less conclusive.

Peri-implant health

The presence and width of keratinized mucosa may be associated with peri-implant health, particularly plaque control and soft-tissue stability. However, phenotype should not be considered a substitute for implant maintenance, plaque control, prosthetic design, implant positioning, or management of established peri-implant disease.

PHENOTYPE-DRIVEN PERSONALIZED TREATMENT PLANNING

The central clinical value of phenotype assessment is its ability to modify treatment planning.

Step 1: Identify phenotype

Assess, where relevant:

- gingival thickness;
- keratinized tissue width;
- facial bone morphology;
- tooth position;
- root prominence;
- existing recession;

- interproximal attachment;
- supracrestal tissue dimensions.

Step 2: Identify anatomical risk

Consider whether phenotype is accompanied by:

- thin facial bone;
- dehiscence/fenestration;
- prominent roots;
- tooth malposition;
- existing recession;
- traumatic brushing;
- orthodontic movement toward the cortical boundary.

Step 3: Determine procedure-specific risk

The same phenotype may have different implications depending on treatment.

For example:

- Thin phenotype + recession → consider soft-tissue augmentation.
- Thin phenotype + orthodontic proclination → evaluate alveolar boundaries and modify mechanics if necessary.
- Thin phenotype + thin facial bone + immediate implant → consider soft/hard tissue management.
- Thin phenotype + subgingival restoration → optimize margin position and emergence profile.

Step 4: Determine whether phenotype modification is necessary

Modification should be considered when there is a clear clinical objective, such as:

- treatment of existing recession;
- inadequate tissue stability;
- anticipated orthodontic risk;
- implant esthetic risk;
- inadequate peri-implant tissue volume;
- difficulty maintaining plaque control.

Step 5: Select the treatment

Treatment selection should integrate phenotype with periodontal diagnosis, patient expectations, bone morphology, tooth position, esthetic demands, morbidity, and long-term maintenance.

Step 6: Monitor

Post-treatment monitoring should assess tissue dimensions, recession, esthetics, periodontal health, and patient-reported outcomes. Digital scans and standardized photographs may facilitate longitudinal evaluation.

Table 2. Phenotype-driven treatment considerations

Clinical situation	Main risk	Assessment	Possible modification
Recession	Tissue instability	GT, KTW, recession classification	CTG/soft-tissue augmentation
Orthodontics	Recession/alveolar boundary violation	GT, bone, tooth position	Modify mechanics ± augmentation
Immediate implant	Facial recession/contour loss	Soft tissue + bone	Soft/hard tissue augmentation
Esthetic implant	Tissue transparency/recession	Mucosal thickness + bone	Soft-tissue augmentation
Crown lengthening	Marginal instability	Tissue and bone dimensions	Tailored flap/osseous surgery
Subgingival restoration	Inflammation/tissue violation	Tissue dimensions	Margin/emergence-profile modification

Phenotype must be interpreted alongside plaque control, smoking, systemic factors, periodontal diagnosis, tooth position, bone morphology, occlusion, restorative factors, and patient compliance.

EMERGING CONCEPTS AND FUTURE DIRECTIONS

The future of periodontal phenotype assessment lies in **precision periodontology**, in which anatomical phenotype is integrated with disease status and patient-specific risk.

Digital workflows may allow three-dimensional mapping of gingival contours and longitudinal monitoring of tissue volume. Intraoral scanning is particularly promising because it is non-invasive and repeatable. Combining IOS with CBCT may provide a comprehensive representation of soft- and hard-tissue anatomy when CBCT is clinically justified. Artificial intelligence may eventually integrate clinical photographs, digital scans, CBCT images, and periodontal records to generate site-specific risk maps. However, future research must establish whether such systems improve treatment decisions and patient outcomes rather than merely classifying phenotype accurately.

Major research priorities include:

- standardized phenotype definitions;
- standardized measurement protocols;
- site-specific thresholds;
- prospective long-term studies;
- patient-reported outcomes;
- cost-effectiveness studies;
- validation of digital and AI-based technologies;
- identification of patients who genuinely benefit from prophylactic phenotype modification.

CLINICAL DECISION-MAKING ALGORITHM

Phenotype-Based Clinical Decision-Making in Contemporary Periodontology

Periodontal diagnosis and disease control



Site-specific phenotype assessment

GT + KTW + bone morphology + tooth position + recession + supracrestal tissue dimensions



Identify anatomical risk

Thin tissue / thin bone / dehiscence / malposition / root prominence / existing recession



Identify treatment-specific risk



Is phenotype modification clinically indicated?

No → phenotype-compatible treatment + maintenance

Yes → select appropriate soft/hard tissue modification



Procedure-specific treatment



Postoperative assessment



Long-term monitoring and maintenance

This algorithm reinforces that phenotype assessment is not an endpoint but a component of individualized clinical decision-making.

FUTURE RESEARCH PRIORITIES

The major unanswered questions are:

1. What quantitative phenotype thresholds are clinically meaningful?
2. Can phenotype independently predict recession after adjustment for confounders?
3. Which patients benefit from prophylactic phenotype modification?
4. What is the optimal non-invasive method for routine phenotype assessment?
5. Can digital technologies reliably monitor phenotype longitudinally?
6. Does increased gingival thickness translate into improved long-term clinical stability?
7. What are the independent contributions of soft-tissue phenotype and facial bone thickness around implants?
8. Can AI improve phenotype-based treatment decisions?
9. What patient-reported benefits justify the morbidity and cost of augmentation?
10. Which interventions provide durable benefit beyond conventional maintenance?

Key Clinical Take-Home Messages

1. **Periodontal phenotype is a multidimensional anatomical characteristic, not simply a thin/thick gingival classification.**
2. **Thin phenotype is associated with greater recession susceptibility but does not independently cause periodontitis.**
3. **Phenotype assessment is most valuable when it changes treatment planning.**

4. **Phenotype modification should be indication-driven rather than routinely performed in every thin phenotype.**
5. **Future phenotype assessment will increasingly involve site-specific digital and multimodal technologies integrated with conventional periodontal diagnosis.**

CONCLUSION

Periodontal phenotype represents an important evolution from the traditional concept of gingival biotype toward a multidimensional understanding of periodontal soft- and hard-tissue anatomy. Gingival thickness, keratinized tissue width, facial bone morphology, tooth position, and related anatomical characteristics can influence susceptibility to recession and the predictability of selected periodontal, orthodontic, restorative, and implant procedures.

Current evidence supports an association between thin periodontal tissues and gingival recession, but does not establish thin phenotype as an independent cause of periodontitis. Similarly, a thin phenotype should not automatically be considered an indication for augmentation. Phenotype modification is most appropriately performed when a specific clinical objective exists, such as treatment of recession, anticipated orthodontic risk, or management of esthetic implant risk.

The most clinically useful approach is therefore **phenotype-driven rather than phenotype-determined**. Assessment should identify the anatomical characteristics present at a particular site, determine how they interact with the planned treatment, and establish whether modification is likely to improve a meaningful clinical outcome.

Digital scanning, ultrasound, CBCT, and multimodal imaging may improve the precision of phenotype assessment, although their ultimate clinical value will depend on demonstrating improved treatment decisions and long-term patient outcomes.

Periodontal phenotype should consequently be incorporated into a broader framework encompassing periodontal diagnosis, patient-level risk, tooth position, bone morphology, treatment mechanics, esthetic demands, and maintenance. This integrated approach provides a rational foundation for personalized and increasingly precise periodontal care.