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Effect Of Periodontal Biotype On Implant Success And Peri-Implantitis Risk: Integrating The Roles Of Dental Assistants And Operating Room Technicians

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Abstract

Dental implant therapy has become a cornerstone of modern oral rehabilitation, offering functional and aesthetic solutions for tooth loss. However, the success of implants and the prevention of peri-implant disease depend on multiple patient- and procedure-related factors, including the periodontal biotype — a critical determinant of soft tissue thickness, contour, and resilience. Thin periodontal biotypes are more susceptible to soft tissue recession, bone loss, and peri-implantitis compared to thick biotypes. At the same time, the increasingly team-based nature of implantology means that not only dentists and periodontists but also dental assistants and operating room (OR) technicians play key roles in ensuring optimal outcomes.

This review explores the effect of periodontal biotype on implant success and peri-implantitis risk, while integrating the often-overlooked contributions of dental assistants and OR technicians in the perioperative pathway. Dental assistants are pivotal in patient education, chairside management, sterilization, and maintenance of soft-tissue health, while OR technicians ensure sterile surgical environments, instrument readiness, and adherence to aseptic protocols. By linking clinical evidence on biotype with a multidisciplinary workflow model, this review highlights how leveraging the complementary skills of all team members can reduce peri-implant complications and improve long-term outcomes.

1. Introduction

Modern implantology achieves >90% 10-year survival in well-selected cases, but success—the sustained absence of disease with stable tissue contours—depends on more than osseointegration (Pjetursson et al., 2012). Two adversaries undermine longevity: soft-tissue recession/papilla loss (compromising aesthetics and hygiene) and peri-implant disease(mucositis and peri-implantitis). Meta-research demonstrates notable disease burdens: peri-implant mucositis in ~43% of patients and peri-implantitis in ~19–22% after 5+ years, driven by plaque, history of periodontitis, smoking, and prosthetic/technical factors (Derks & Tomasi, 2015; Berglundh et al., 2018; Heitz-Mayfield & Mombelli, 2014; Renvert et al., 2019).

The periodontal biotype (or phenotype), historically categorized clinically (thin-scalloped vs. thick-flat), correlates with gingival thickness (GT), keratinized mucosa (KM) width, and buccal plate thickness—parameters that predict the magnitude of crestal remodeling and recession after extraction and implant placement (Olsson & Lindhe, 1991; Spray et al., 2000). Systematic analyses show thin GT (<2 mm) is associated with greater early crestal bone loss and recession; conversely, ≥2 mm soft-tissue thickness at the implant neck significantly attenuates bone loss (Linkevicius et al., 2009; Linkevicius et al., 2015). Post-extraction ridge collapse is also phenotype-sensitive: buccal bone ≤1 mm predicts pronounced horizontal/vertical loss (Chappuis et al., 2015). Aesthetic stability is similarly phenotype-linked: papilla height relates to the bone crest–contact point distance (≤5 mm optimal) and is more difficult to maintain in thin biotypes (Tarnow et al., 1992; Evans & Chen, 2008; Jung et al., 2012).

Saudi Arabia reports high periodontitis prevalence (Albandar, 2002), intersecting with rising implant demand under Vision 2030. Given phenotype-related risk and regional disease burden, biotype-stratified pathways and team-based execution become central. However, literature and curricula frequently understate how dental assistants and OR technicians operationalize phenotype-sensitive protocols at scale—asepsis fidelity, delicate retraction, moisture control, reprocessing discipline, and perioperative patient coaching—all of which modulate biotype behavior and infection risk under daily clinical realities (CDC, 2021; AAMI ST79; ISO 17664; WHO, 2009).

This review synthesizes global evidence on biotype and peri-implant outcomes; details how dental assistants/OR technicians anchor safe, phenotype-aware workflows; and proposes a multidisciplinary model spanning risk assessment, surgery, prosthetics, and maintenance, aligned to EFP S3 and ITI guidance and adaptable to Saudi systems.

2. Periodontal Biotype and Implant Success

2.1 Definition, measurement, and global evidence.

Biotype (phenotype) encompasses gingival thickness**,** KM width**,** and bone morphotype. Clinical probing with an endodontic file/needle ("probe transparency") stratifies thin vs. thick; ultrasound and CBCT add precision, though CBCT is primarily for hard-tissue mapping (Jepsen et al., 2018). Global cohorts and RCTs indicate: (a) thin GT <2 mmcorrelate**s** with greater early crestal bone loss following implant uncovery/restoration (Linkevicius et al., 2009, 2015); (b) soft-tissue augmentation at the collar improves mid-term marginal bone stability and mucosal levels (Zucchelli & Mounssif, 2015; Cairo et al., 2014); (c**)** KM ≥2 mm is associated with better plaque control, less mucositis, and less recession, though disease can still be controlled with excellent hygiene (Lin et al., 2013; Sanz-Martín et al., 2019).

2.2 Timing and technique choices shaped by phenotype.

Immediate implant placement in thin biotypes risks buccal concavities and recession despite meticulous technique; enhanced predictability often requires contour management (socket shield/partial root retention), bone and connective tissue grafting, and emergence-profile provisionalization (Chen & Buser, 2009; Cosyn et al., 2016). Early or delayed placement with contour-augmenting GBR and soft-tissue thickening is frequently superior in thin phenotypes where the buccal plate is thin or dehiscent (Buser et al., 2013; Chappuis et al., 2015). Thick phenotypes tolerate immediate protocols more predictably when buccal plate ≥1.5–2 mm and the soft tissue is ≥2 mm (Kan et al., 2011; Evans & Chen, 2008).

2.3 Macro-/micro-design and prosthetic interfaces.

Platform switching reduces crestal remodeling by moving the microgap inward, attenuating inflammatory cell infiltrate at the bone crest (Lazzara & Porter, 2006). Conical connections show less micro-movement and bacterial pumping than external hex, benefiting thin tissues (Jung et al., 2012). Subcrestal placement (0.5–1 mm) in thin phenotypes can compensate for remodeling, but over-subcrestal risks biologic width encroachment and bone loss; prosthetic emergence must be convex-to-flat to avoid mucosal overload (Jung et al., 2012; Gallucci et al., ITI). Globally, ITI consensus supports phenotype-driven abutment selection, transmucosal height matching the vertical soft-tissue thickness, and early soft-tissue conditioning with provisionals (Buser et al., 2013; Gallucci et al., ITI consensus).

3. Peri-Implantitis: Etiology and Risk Factors—Biotype as a Modifier

3.1 Disease spectrum and prevalence.

Peri-implant mucositis is plaque-induced inflammation without bone loss; peri-implantitis includes progressive bone loss after initial remodeling (Berglundh et al., 2018). Worldwide meta-analyses reveal variability due to inconsistent definitions, but peri-implantitis commonly affects ~10–20% of patients long-term (Derks & Tomasi, 2015; Heitz-Mayfield & Mombelli, 2014).

3.2 Risk factors and phenotype interaction. Strong predictors include poor plaque control, history of periodontitis, smoking, uncontrolled diabetes, residual cement, and mal-contoured prostheses (Renvert et al., 2019; Monje et al., 2016; Romandini et al., 2021). Thin biotypeamplifies susceptibility by: (a) lowering the biologic width buffer; (b) increasing recession risk that exposes rough surfaces; (c) limiting vascular supply for immune surveillance and healing (Linkevicius et al., 2009; Sanz-Martín et al., 2019). In thin phenotypes, even minor prosthetic overcontours or cement remnants can trigger persistent inflammation with faster breakdown (Kotsakis et al., 2016). Global S3-level guidance (EFP) emphasizes risk control before and after placement—particularly smoking cessation, periodontal stabilization, and maintenance, with phenotype-specific soft-tissue augmentation when needed (Berglundh et al., 2018; EFP S3 CPG).

4. Dental Assistants' Role—Operationalizing Biotype-Aware Care

4.1 Preoperative mapping and education.

Assistants standardize intake documentation: phenotype notes (probe transparency, GT impression), KM width, smoking status, glycemic history, periodontitis stage, and oral hygiene indices—creating a phenotype-risk dashboard that prompts biotype-specific informed consent (Jepsen et al., 2018; EFP S3). They deliver motivational interviewing to improve plaque control and smoking cessation and demonstrate atraumatic interdental hygiene suited to thin tissues (super-soft brushes, rubber interdental picks) to avoid papillary trauma (Sanz-Martín et al., 2019).

4.2 Reprocessing and asepsis fidelity.

By enforcing Spaulding classification and ISO 17664 reprocessing instructions—cleaning, disinfection, packaging, sterilization parameters, and traceability—assistants reduce cross-contamination risk that disproportionately harms thin biotype sites (CDC, 2021; ISO 17664; AAMI ST79). They verify implant component sterility, lot numbers, and expiry; stage micro-instruments (tissue forceps, microsutures) that minimize trauma.

4.3 Chairside soft-tissue stewardship.

During surgery and prosthetics, assistants coordinate saliva control, gentle retraction (micro-retractors, Teflon tape), moisture maintenance on grafts, and atraumatic suction to protect fragile mucosa. They manage provisional try-ins to ensure emergent contours do not blanch thin mucosa and track soft-tissue color/bleeding indices at each maintenance visit to trigger early intervention (Jung et al., 2012; Gallucci ITI).

4.4 Maintenance leadership.

Assistants schedule phenotype-stratified recalls (e.g., 3–4 months for thin biotype with periodontitis history), deploy implant-safe ultrasonic tips and air-polishing powders (glycine/erythritol), and coach cement-retained cases to report bleeding promptly (Renvert et al., 2019; Kotsakis et al., 2016). They also administer peri-implant risk scores that combine phenotype, plaque, bleeding, and prosthetic factors to standardize escalation pathways (EFP S3).

5. Operating Room Technicians' Role—Protecting Tissues and the Field

5.1 Field integrity and timing.

OR technicians ensure laminar instrument flow, proper class IV field setup for implant kits, and timely turnover to limit soft-tissue desiccation/ischemia—critical in thin phenotypes prone to recession (Association of Surgical Technologists, 2020). They stage microsurgical trays, CTG/ADM handling tools, and collagen membranes, maintaining hydration and sterility.

5.2 Micro-instrumentation and graft logistics.

They prepare punch-calibrated tissue thickness gauges, micro-blade handles, tunneling kits, and PTFE microsutures(5-0/6-0) that reduce incision trauma and strangulation—technique sensitivities repeatedly linked to soft-tissue stability worldwide (Zucchelli & Mounssif, 2015; Cairo et al., 2014). For GBR, they ready low-profile fixation, titanium-reinforced membranes, and particulate/xenograft mixes per surgeon preference.

5.3 Standards compliance and traceability.

OR technicians integrate AAMI ST79 steam sterilization monitoring and ISO 17664 device reprocessing instructions, document cycle parameters, and maintain UDI/lot traceability for implants/abutments to streamline root-cause analysis for any postoperative complication (AAMI ST79; ISO 17664; CDC, 2021).

5.4 Postoperative handover.

They co-deliver written and verbal aftercare, highlighting thin-biotype precautions (no manipulation of provisional, gentle hygiene, high-fluoride pastes if recession risk), and schedule early check-ins to detect suture tension or blanching, tightening the loop with assistants and clinicians (EFP S3; ITI).

6. Integrated Multidisciplinary Workflow—A Biotype-Anchored Model

6.1 Pre-surgical stage.

- **Risk map**: phenotype (GT/KM), CBCT buccal thickness, periodontal staging, systemic risks (A1c), smoking pack-years.
- **Pathway selection**: thin biotype with buccal plate ≤1 mm → staged approach (GBR + CTG), platform-switch conical connection, subcrestal 0.5–1 mm; thick biotype with intact buccal plate → broader latitude for immediate placement/loading if primary stability and intact socket allow (Buser et al., 2013; Chappuis et al., 2015; Linkevicius et al., 2015).
- **Team brief**: assistants/OR techs review instrument maps, graft logistics, sterility plan, and phenotype-specific checkpoints (no dry grafts, minimal retraction).

6.2 Surgical/prosthetic execution.

- **Soft-tissue preservation**: papilla-sparing incisions, partial-thickness tunneling for CTG, microsutures without strangulation (Zucchelli & Mounssif, 2015; Cairo et al., 2014).
- **Bone containment**: GBR where buccal shell <1–1.5 mm; particulate + membrane stabilization; **platform switching** and conical connection to protect crest (Lazzara & Porter, 2006; Jung et al., 2012).

- **Emergence control**: provisional contours add 0.5–1.0 mm soft-tissue thickness over time; contouring avoids overcompression in thin tissues (Jung et al., 2012; Gallucci ITI).
- **Cement control**: favor screw-retained where feasible; for cemented crowns, use vented abutments/self-adhesive cements**,** radiopaque cement, and deliberate excess removal protocols—key for thin tissues (Kotsakis et al., 2016).

6.3 Maintenance and surveillance.

- **Recall cadence** by phenotype and risk: thin + periodontitis history (3–4 mo), thick + low risk (6 mo).
- **Instrumentation**: low-abrasive powders, implant-safe scalers; avoid scratching titanium/zirconia.
- **Indices**: bleeding/suppuration charting, PES/WES aesthetics tracking, radiographic bone levels at baseline, 12 mo, and risk-based thereafter (Renvert et al., 2019; Jung et al., 2012).
- **Escalation**: early mucositis → debridement + behavior change; refractory or radiographic progression → surgical access/decontamination, potential soft-tissue augmentation in thin phenotype (Heitz-Mayfield & Mombelli, 2014; EFP S3).

This model mirrors ITI and EFP S3 pathways and scales to Saudi Vision 2030 by embedding standardized checklists, centralized sterilization monitoring, and phenotype-risk dashboards in public/private clinics.

7. Challenges and Future Directions (Global and Saudi Contexts)

7.1 Training and scope.

Global curricula rarely formalize phenotype-aware competencies for assistants/OR techs. Creating credentialed modules (biotype assessment support, micro-instrumentation, ISO/AAMI reprocessing, peri-implant risk scoring) and simulation labs can close practice gaps worldwide and in Saudi Arabia's expanding dental sector (Jepsen et al., 2018; CDC, 2021; AAMI ST79).

7.2 Digital workflows and precision planning.

Widespread adoption of intraoral scanning, dynamic/static guided surgery, and CAD/CAM provisionals enables precise emergence profiles that respect thin tissues. However, this requires allied staff trained in data integrity, guide sterilization, and surgical file–tray reconciliation to avoid chairside drift (Gallucci ITI; Chen & Buser, 2009).

7.3 Materials and surfaces.

Evidence on zirconia vs. titanium abutments suggests comparable survival; zirconia may reduce gray-shine in thin tissues, while platform-switched titanium remains robust mechanically (Jung et al., 2012). Ultra-hydrophilic implant surfaces may accelerate early osseointegration, but do not replace soft-tissue conditioning in thin phenotypes (Buser et al., 2013). Future RCTs should stratify by biotype.

7.4 Public health and registries.

International implant registries linking phenotype, risk factors, and outcomes would refine real-world estimates of recession and peri-implantitis. Saudi Arabia could seed a national implant registry under Vision 2030, leveraging e-health infrastructure to benchmark clinics, surface sterilization gaps, and quantify phenotype impacts on outcomes (EFP S3; Vision 2030).

7.5 Microbiome and host-response personalization.

Worldwide studies increasingly implicate dysbiotic biofilms and hyper-inflammatory phenotypes in peri-implantitis; integrating point-of-care biomarkers (MMP-8, calprotectin) with phenotype could personalize recall intervals and preempt escalation (Renvert et al., 2019). This dovetails with precision prevention—e.g., earlier CTG/ADM in high-risk thin phenotypes.

8. Conclusion

Worldwide evidence converges on a core truth: periodontal biotype strongly conditions implant behavior—from the magnitude of early crestal remodeling to the likelihood of recession and peri-implant disease. Thin phenotypes, especially with limited buccal bone and narrow keratinized mucosa, demand softer hands, slower protocols, and stricter maintenance. Clinicians can engineer success with phenotype-aware timing, bone/soft-tissue augmentation, platform-switched conical connections, and meticulously contoured provisionals. Yet durable success is not a solo act. When dental assistants enforce reprocessing fidelity, protect soft tissues chairside, and lead patient education—and OR technicians maintain field integrity, instrument precision, and sterile logistics—the biologic advantages we design into the plan actually reach the patient. Embedding these allied roles in standardized, biotype-anchored pathways, aligned with EFP/ITI guidance and CDC/ISO/AAMI standards, can reduce peri-implantitis risk and elevate aesthetics worldwide and within Saudi Vision 2030. The next advances will come from team literacies—shared checklists, phenotype dashboards, and registry-driven learning—so that every implant placed has a soft-tissue environment designed and cared for to last.

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