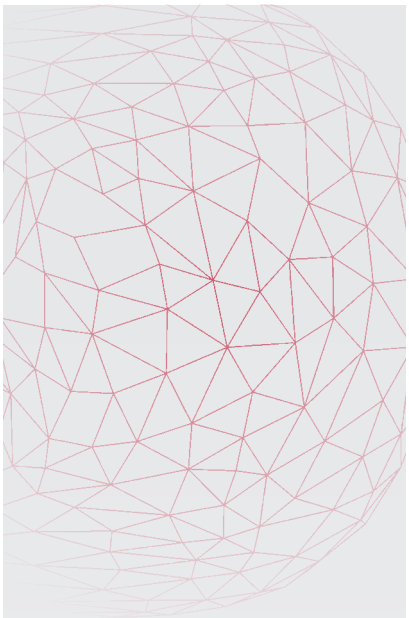

"A Requiem for the Periodontal Ligament": A Nuanced Perspective

In 1991, George Zarb and Tomas Albrektsson, in an editorial titled "A Requiem for the Periodontal Ligament," posited that we must not presume that bacterial plaque is the major etiologic factor in implant failure.[1] They warned against the periodontal model mindset that may preclude other research considerations. Eighteen years later, Albrektsson et al[2] revisited the topic in a follow-up editorial and questioned the alarm sounded in reports of peri-implantitis (PI) incidence. Instead, they purported that the irreversible condition of PI, infrequently found, may be due to compromised healing and adaptation, which may follow genetic disorders, poor bone quality, or traumatic surgical techniques. In a later paper, an imbalance of the immune system—with a dysregulation between osteoblastic and osteoclastic cell formation—was offered to explain the cause of marginal bone loss in PI.[3] The authors also noted that there is a lack of reliable clinical evidence that overloading must be an incriminating reason for bone loss around implants. They then echoed the challenge to accept the fundamental differences between the attachment mechanism for a tooth and a controlled healing one for implants.

The two seminal editorials now beg the following questions, based on additional research: What is known about the pathogenesis, risk factors, and incidence of PI? Why have the responses to established treatment of PI been refractory? What are the emerging therapies? How do implants manage occlusal loads differently than teeth? Given these differences, why has there been resistance to conceptualizing implants separately from the periodontal model?

Whether bacteria are the main or secondary etiologic factors, the histopathology of PI and periodontitis have been shown to be distinguished by diverse microbiota. While aggressive gram-negative bacteria are found in both disease entities, the PI sites were also colonized by specific gram-positive rods and anaerobic gram-negative rods.[4] While these two conditions share similar clinical features, PI progresses in a faster nonlinear pattern due to a lack of mesenchymal stem cell repair as well as reduced vascularity and a poor attachment apparatus compared to the periodontal niche.[5] Aggregate risk factor analysis for PI can more accurately assess a patient's prognosis.[6] The presence of periodontitis (OR = 3.84) and cigarette smoking (OR = 2.07) are highly suggestive, and therapeutic ionizing radiation above 55 Gy, antiresorptive agents, H_2 antagonists, antidepressant medications, diabetes mellitus, osteoprotegerin gene polymorphisms, high plaque index, and lack of keratinized tissue are suggestive of PI. In addition, poor surgical technique or positioning, unhygienic prosthetic design, and excess cement increase the risk.[7] The prevalence of subject-based PI has been reported to be approximately 20% in the Americas and greater in Europe.

A notable finding of PI is its crevicular fluid, which contains higher active matrix metalloproteinase-8 levels than the crevicular fluid of similar deep long-term periodontal sites of natural teeth.[5] Given the regenerative capacity and largess of nutrients and defense cells found in the periodontal ligament (PDL), it is notable that the marginal bone loss around implants is greater than that around periodontally compromised teeth in the same patient over 10 years, if properly treated and maintained.[8]

Inconsistent results have been reported from nonsurgical treatment, resective or reconstructive surgery, and combined approaches for the

doi: 10.11607/ijp.2024.4.e

resolution of PI. Ironically, when PDL pluripotent stem cells isolated from root surfaces of extracted teeth were introduced in an in vivo model, more than 70% of the defects were reintegrated using the delayed placement protocol of implants.[9] Efforts are also ongoing to produce predictable methods to regenerate peri-implant bone with tissue engineering and regenerative medicine.[10] The development of an approach to foster a foreign body equilibrium with new implant surfaces and assessment of the osteoimmunobiology of the host, and the discovery of M2 type macrophages, is promising.[11]

On the mechanical front, the PDL is the rate-limiting factor in managing load-related stress > 90 kPa (within physiologic range). Above this level, the PDL generates a local hypoxia and fluid flow, initiating an osteoclastic resorption.[5] However, in the implant scenario, despite the load being transferred directly to the surrounding bone, the fatigue failure of the bone is at least three times that of the PDL. Therefore, adverse effects from nonaxial loads on implants have not been reported to be as pronounced as in teeth.

While the PDL confers a robust biologic reserve and defense to teeth that implants do not possess, surprisingly, implants benefit from not having a PDL in mediating higher occlusal loads. This explains why posterior implant cantilever prostheses and crown-to-implant ratios of 2:1 provide predictable outcomes, unlike their tooth-borne counterparts. The impact of this understanding has led to a more minimally invasive approach to treatment planning rather than an intuitive default overengineering to compensate for a tooth analog without a suspensory ligament.

The inertia to accept the differences between an implant and a periodontal model has been explained by social scientists Tversky and Kahneman.[12] They illuminated the human tendency for a mental shortcut (heuristics) by using associative substitution (teeth) when presented with a novel discovery (osseointegration).

An honest eulogy for the PDL would be to praise its biologic defense prowess while appreciating its limits to mediate higher occlusal loads. By keeping the periodontal model alive for implants, it has delayed our ability to assess the true strengths and weaknesses of the dental implant complex.

Steven J. Sadowsky, DDS
University of the Pacific Arthur A. Dugoni School of Dentistry, San Francisco, California, USA.

REFERENCES

1. Zarb G, Albrektsson T. Osseointegration—A requiem for the periodontal ligament?-An editorial. Int J Periodontics Restorative Dent 1991;11:88–91.
2. Albrektsson T, Brunski J, Wennerberg A. 'A requiem for the periodontal ligament' revisited. Int J Prosthodont 2009;22:120–122.
3. Albrektsson T, Dahlin C, Reinedahl D, Tengvall P, Trindade R, Wennerberg A. An imbalance of the immune system instead of a disease behind marginal bone loss around oral implants: Position paper. Int J Oral Mxillofac Implants 2020;35:495–502.
4. Sadowsky SJ. Peri-implantitis after 40 years: Evidence, mechanisms, and implications: A mapping review. J Prosthet Dent 2023:S0022-3913(23)00114-2.
5. Sadowsky SJ, Brunski JB. Are teeth superior to implants? A mapping review. J Prosthet Dent 2021;126:181-187.
6. Curtis DA, Lin GH, Fishman A, et al. Patient-centered risk assessment in implant treatment planning. Int J Oral Maxillofac Implants 2019;34:506–520.
7. Giok KC, Veettil SK, Menon RK. Risk factors for peri-implantitis: An umbrella review of meta-analyses of observational studies and assessment of biases. J Dent 2024;146:105065.
8. Rasperini G, Siciliano VI, Cafiero C, Salvi GE, Blasi A, Aglietta M. Crestal bone changes at teeth and implants in periodontally healthy and periodontally compromised patients. A 10-year comparative case-series study. J Periodontol 2014;85:e152–e159.
9. Park SY, Kim KH, Kim S, et al. Comparison of experimental peri-implantitis models after application of ex vivo BMP2 gene therapy using periodontal ligament stem cells. Sci Rep 2020;10:3590.
10. Goker F, Larsson L, Del Fabbro, Asa'ad F. Gene delivery therapeutics in treatment of periodontics and peri-implantitis: A state of art review. 2019;20:3551.
11. Li Y, Li X, Guo D, et al. Immune dysregulation and macrophage polarization in peri-implantitis. Front Bioeng Biotechnol 2024;12:1291880.
12. Tversky A, Kahneman D. Judgment under uncertainty: Heuristics and bias. Science 1974;185:1124–1131.