A Case of Mistaken Identity

When the concept of osseointegration was introduced to North America, it would foreshadow a tectonic shift in dental treatment planning. But, almost 40 years later, we are just beginning to understand the difference between implants and teeth. A collective cognitive bias has throttled a clear analysis. Tversky and Kahneman, social scientists, have observed that when a new paradigm is discovered, there is a tendency to use associative substitution (eg, implants = teeth) to simplify the mental processing of a novel discovery.[1] Evidence is then selectively culled to buttress this premise. The impact of seminal publications has revealed this heuristic process.

Bo Rangert et al, in 1997, demonstrated high stress levels on implants and surrounding bone when modeling posterior implant-supported cantilevered fixed partial dentures.[2] What reinforced the association between implants and teeth was a 15-year retrospective study in 2002, in which Terry Walton reported that tooth-borne cantilevered fixed partial dentures had significantly greater failure rates.[3]

Carl Misch published a review article in 2005 on short implants, documenting that machined implants < 10 mm had a higher failure rate than longer ones.[4] Using biomechanical methods to reduce stress, such as splinting or increasing the diameter, he reported 99% implant survival with 7- to 9-mm implants. He theorized that the natural teeth follow a similar biomechanical approach to accommodate the higher occlusal forces in the posterior regions of the mouth. With molar teeth, the diameter is increased, and the roots are splinted together. In Misch's article, a tooth model was used to improve the design and use of short implants.

Both examples have promoted a larger narrative that teeth and implants have similar biomechanical behavior. Early published investigations burnished the associative substitution view of implants, but there were inherent flaws in the premise. In the first case, simple finite element analyses could not accurately model the threshold for damaging microstrain in the peri-implant bone with cantilever designs. In the second instance, machined implants were compensated by strategies commandeered from tooth geometries, but when textured surfaces came into vogue, it was apparent that the tooth model was not essential to success.

More recently, a systematic review on the marginal bone loss around implant-supported restorations, with and without a cantilever, discerned no significant difference.[5] In 2019, a clinical trial with a 5-year follow-up demonstrated that with textured surfaces, unsplinted 7- to 9-mm implants of standard diameter achieved similar survival outcomes compared with longer implants, even when crown-to-implant ratios are 2:1.[6]

If strong hierarchical evidence indicates that implants may manage forces better than teeth, a new understanding of their differences is in order. How is it that an ankylosed root analog without a periodontal ligament (PDL) transmits loads more favorably to the supporting bone? Notably, while loads within a certain physiologic range of stress (< 90 kPa) prompt an osteogenic differentiation of the healthy PDL, excessive compressive stress can induce local hypoxia and a cascade of osteoclastic factors.[7] Here, the rate-limiting factor is the PDL.

While the PDL does not have unflagging shock absorbing function, its biologic response to noxious stimuli is exceptional. The PDL confers superior defense to the gingival complex compared with the peri-implant microenvironment. The dominant cell population of the PDL is multipotent fibroblasts, which can differentiate into cementoblasts and osteoblasts. The rich vascular network can increase in number and size in response to inflammation.

Conversely, the peri-implant interface is bereft of the regenerative capabilities found in the periodontal environment. The poorly sealed connective tissue compartment offers less protection to downgrowth of bacteria and foreign bodies like cement, which can accelerate horizontal recession. The reduced blood supply compromises the source of nutrients and local defense against pathogens.

The inferior resistance of the peri-implant milieu to protect against biologic challenges is substantiated by lower success rates of implants compared with teeth in longitudinal investigations, even when the teeth are periodontally compromised, if properly treated and maintained.[8] The high incidence of peri-implant disease and its refractory response to treatment can better be understood in the light of a vastly different defensive shield compared with teeth.

Perpetuating the myth that implants mimic teeth has resulted in a less conservative approach to dental treatment planning. The conventional wisdom that one implant per tooth is a far better treatment than

doi: 10.11607/jomi.2021.1.e

employing an implant-supported fixed partial denture or a cantilever prosthesis has led to more invasive and costly procedures. The use of longer implants requiring augmentation, instead of shorter implants, has led to an increase in morbidity, complications, and cost. The preemptive extraction of periodontally compromised teeth and replacement with implants has not been shown to necessarily preserve marginal bone in compliant patients.[8] These data are the basis for evidence-based minimally invasive treatment.[9]

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