

Implant Success and Failure Is Dependent Upon the Bone Response. Show a Little Respect for Those Bone Cells!

As clinicians we see the “macro-results” but must understand the “micro-events” that support and lead to the desired conclusions. The net result is that the implant must be stable and to achieve that goal, bone must be in close apposition to the implant (aka osseointegration). Some may debate the definition of osseointegration, but that is not the purpose of the editorial.

It is known that immediately upon placement and over the subsequent months, a series of cellular and molecular events occurs that enables the host tissues to biologically integrate the implanted alloplastic material into the native or grafted bone.¹ Cortical bone has the ability to immediately withstand torsional loading and provide initial stability, whereas medullary bone does not provide the same degree of initial stability. Medullary bone is rich in vascular canals and thus provides a delivery mechanism for mesenchymal stem cells. The implant surface becomes populated with bone cells that are responsible for forming *de novo* bone in close approximation to the dental implant. For the new bone to form, the local environment undertakes the breakdown of the existing native bone.²

Multiple proximal and systemic factors can affect this process. Implant material biocompatibility, implant placement/position, loading protocol, material degradation/titanium particle release, patient age, local or systemic pathology, and medication can initiate positive or negative effects on the osseointegration process.³ Implant dentistry has achieved a partial understanding of these factors. This knowledge has led to the evolution of hydrophilic and bioactive implant surfaces that aid in early osseointegration. We also understand that limiting (or controlling) the inflammatory process can diminish local tissue trauma¹ and thereby lead to prolonged integration with continual constructive bone remodeling.

The remodeling process is as equally complex as the initial integration. Osteocytes are the regulators of bone metabolism and bone remodeling. Osteocytes control these processes by regulating multiple molecular and cellular activities that in turn control osteoclast and osteoblast function.^{4,5} Bone metabolism is affected by numerous factors including: cholesterol, fatty acids, vitamin D, hyperglycemia, and medication.

The relationship between body fat and bone marrow fat is not fully understood.⁶ It is known that increased levels of adipogenesis in the bone marrow causes decreased osteoblast formation and viability.⁷ Adipocytes release pro-inflammatory cytokines that initiate bone loss.⁸

Vitamin D stimulates osteoblasts to produce bone mineral matrix in addition to coupling bone resorption with formation. The result of this coupling is optimized bone remodeling.⁹

There is an increase in patients who present to our offices with insufficient serum levels of vitamin D. Insufficiency of vitamin D may be a confounding factor for implants.¹⁰ Animal studies discovered that vitamin D supplementation significantly increased peri-implant bone density, bone-implant contact and peri-implant trabecular microarchitecture.¹¹ Further research is necessary to confirm that vitamin D supplementation will significantly improve implant healing.

Diabetes mellitus is known to decrease osteoblast activity resulting in suppressed bone formation. It is confirmed that uncontrolled diabetics have an increased risk of implant failures.¹² Undiagnosed diabetes may help explain some unanticipated implant failures. Hyperglycemia has been related to decreased levels of bone formation markers, such as osteocalcin, bone-specific alkaline phosphatase and C-terminal telopeptide of collagen-type I.¹³ Morashini et al.¹⁴ concluded that the implant failure rate was no higher for well-controlled diabetic patients than non-diabetic patients. However, all diabetic patients experience more crestal bone loss than non-diabetic patients. Additional studies are needed to confirm the affects of both controlled and uncontrolled diabetes on dental implant long-term success.

Some systemic medications are known to have a direct affect on bone cells and therefore, impact bone formation and/or remodeling. If remodeling is affected in a negative manner this may result in bone loss surrounding dental implants.¹⁵ Examples being serotonin reuptake inhibitors and proton pump inhibitors. Both drug classes have been implicated in bone loss and increased implant failure rates.¹⁶

Implant surface corrosion, as the result of dioxide layer titanium degradation is known to cause the release of titanium particles that induce an inflammatory reaction in the peri-implant tissues.¹⁷ It has been observed that chronic wear found with long-term hip replacements can result in the release of titanium particles. These titanium particles are known to cause a foreign body inflammatory reaction.¹⁷

There are multiple reasons for implant failure or success. This editorial highlights some of the reasons, but they are not limited to those mentioned here. The Insua et al.¹ review determined: “Implant osseointegration is a long-term equilibrium between host immune cells and bone biomaterials.”

Patients desire and expect successful implant treatments. They rely upon us to help ensure that success. We have a responsibility to examine all aspects of the dental implant case; leaving no stone unturned when diagnosing and treatment planning a case. Implant dentistry is the most complex discipline in dentistry. Yet there are some who are either unaware of the complexities or choose to ignore the complexities and deny that it should be a specialty in its own right.

For more information on bone response, see the article by Insua et al.¹

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