Tissue Response to Mechanical Load in Dental Implants
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Abstract
Dental implants have reported success rates of over 90% over long periods of time. However failures still occur and seem to be unpredictable. The sequence of events that take place at the interfacial zone between an implant and the host tissue deserves much attention. The placement of an implant disturbs the normal physiological distribution of forces, fluids, and cell communication. One factor that is being increasingly considered in failure of dental implants is occlusal loading. The purpose of this article is to describe the current understanding of the biological and biomechanical response of host tissues to mechanical loads especially of the dental implant-bone interface.

Keywords: Dental Implants; Host Tissues; Mechanical Loads; Osseo Integration.

Introduction
Modern day dentistry has a common goal of restoration of teeth of the patients to achieve optimum function, aesthetics, speech and comfort either by restoration of decayed teeth or by replacement of missing teeth. Implant dentistry has achieved this goal and reports of its success range between 78-100%. Success in implant dentistry can be directly correlated to the quality and quantity of bone at an implant recipient site. Surgical traumas together with anatomical conditions are believed to be the most important etiological factors for early implant losses (3.6% of 16,935 implants). On the basis of the published literature, there appears to be a number of scientific issues which are yet not fully understood. Therefore, further clinical follow-up and meta-analysis retrieval studies are required in order to achieve a better understanding of the mechanisms for failure of Osseointegrated implants.

Biological Response
The implant–tissue interface is a dynamic region of interaction, which changes its characteristics from its genesis to its maturity. If the implant is stable in the bone at the time of placement, then the interface is more likely to result in osseointegration. Relative movement (or micromotion) between the implant and the bone at the time of placement is more likely to favor the development of a fibro-osseous interface. The concept of osseointegration was developed by Branemark in the middle of the 1960s and led to the predictable long-term success of oral implants. One of the first definitions of osseointegration, given by Albrektsson et al., was a “direct functional and structural connection between bone and the surface of a load-bearing implant”. Bone apart from providing strength and rigidity also participates in maintaining hormonally regulated calcium homeostasis in the body. It has been proven that bone responds to both hormonal and biomechanical (functional loading) regulation. Researchers have theorized that the actual strain that is perceived by the bone tissue initiates a chain of events that results in a biological response. The objective of a good implant design would be to establish and maintain a strain environment within the host bone tissue and at the interface that favors osseointegration of the implant. Micromotion between the implant and the bone results in the formation of a fibro-osseous interface but some researchers have observed that low frequency micromotion may stimulate bone growth.

Mechano-transduction: Mechano-transduction is a multistep process that includes:
- a. Mechanocoupling i.e. transduction of mechanical forces into signals sensed by sensor cells,
- b. Biochemical coupling i.e. conversion of mechanical signal into a biochemical signal to elicit a cellular response such as gene activation,
- c. Transfer of a signal from sensor to effector cells, and
- d. The effector cell response.

The amazing osteocyte: Osteocytes play a vital role in the osseointegration and bone formation around the implant fixture. These cells are embedded within the lacunae in bony matrix and act as mechanosensors and...
help translate mechanical loads into biochemical signals.\textsuperscript{16} The extensive lacuno-canalicular network of osteocytes enables them to communicate with each other, as well as with periosteal and endosteal osteoblasts. Furthermore, osteocytes have higher sensitivity to mechanical stimulation than osteoblasts.\textsuperscript{17} Osteoblastic differentiation is characterized by the early expression of alkaline phosphatase and later by expression of the DNA-binding transcription factor Runx-2/Cbfa-1. This Runx-2/Cbfa-1 is responsible for osteoblastic production of collagen, the largest constituent of unmineralized osteoid. Mineralization of osteoid forms the new bone, resulting in intramembranous ossification.

Figure 1: Soft tissue overlying the bone surrounding the implant fixture. (Source: Google images, Health base, healthcare beyond boundaries)\textsuperscript{16}

Figure 2: A strain environment within the host bone tissue and at the interface that favors osseointegration of the implant (Source: Internet images, Osseointegration, Orthotics and Prosthetics)\textsuperscript{14}

\textbf{Osteopromoter:} An implant is placed into bone and expected to heal successfully. However, if the quality of the bone is compromised, an osteopromoter may be considered. They are:

1. TGF-β: During the inflammatory phase it is released from platelets and stimulates mesenchymal cell proliferation. TGF-β has been shown to participate in all phases of bone healing.
2. PDGF: It is known to stimulate the reproduction and chemotaxis of connective tissue cells and matrix deposition, and is angiogenic.
3. Insulin-like growth factor (IGF): IGF has demonstrated a capacity to increase bone cell mitosis and increase the deposition of matrix.
4. Platelet-rich plasma (PRP): Platelets are known to contain a number of growth factors, of which TGF-β and PDGF are two. As platelets degranulate these factors are released which initiates bone healing. PRP can be used as an adjunct to bone regeneration.

Regulation of osteoblastic activity by osteocytes has been proposed to occur through gap junctions, with the stimulation of gap junctions mediated by prostaglandin E\textsubscript{2} (PGE\textsubscript{2}).\textsuperscript{19} Loading of bone decreases osteocyte apoptosis, whereas disuse and supra physiological strains increases it followed by haversian remodeling.

Figure 3: Bony healing at the implant-host interface (Source: Internet images, pocketdentistry.com)\textsuperscript{15}

Figure 4: Fibrous healing at the implant-host tissue interface. (Source: Internet images, pocketdentistry.com)\textsuperscript{15}
Gene Expression: Strain induced alterations in patterns of osteoblast gene expression have been studied by various researchers and it has been reported that the initial response to strain is a rapid increase in c-fos mRNA expression. This indicates increased proliferation, along with a rapid decline in levels of mRNA encoding bone matrix proteins, such as type I collagen, osteopontin, and osteocalcin. Reversal of this trend is usually seen with time as the proliferation tapers off, accompanied by an increase in expression of the matrix proteins. Active osteogenesis involves the expression of genes that result in the production of collagen type I protein. This feature makes the type I collagen molecule a valuable indicator of differentiated osteoblastic activity. Both osteocalcin and osteopontin are regulated by (1,25(OH)2D3), which directly influences the genes of both proteins. This is possible because the genes for both osteopontin and osteocalcin contain regions that recognize vitamin D. Osteoblastic products such as interleukin-1 (IL-1) can stimulate osteoblasts. Frost groups these cells as basic multicellular units (BMUs). These BMUs are most prevalent on periosteal and endosteal surfaces, and the periosteal BMUs are most sensitive to biomechanical stimuli.

Biomechanical response: The magnitude of strain imparted to the dental implant-bone interface is determined by various factors:

- Load direction: Occlusal loads, in general are classified as axial and non-axial forces. Axial forces act perpendicular to the occlusal plane and are suggested to be more favorable as they distribute stress more evenly throughout an implant. Non-axial forces act in a non-perpendicular direction to the occlusal plane are thought to disrupt the bone-implant interface.

- The degree to which the mechanical properties of cortical bone are dependent on its structure is referred to as anisotropy. Cortical bone of the human mandible has been reported as transversely isotropic, with the stiffest direction oriented around the arch of the mandible. A material is said to be orthotropic if it exhibits different properties in all three directions and isotropic if the properties are the same in all three directions.

- Rate of loading: Bone fails at a higher load but with less allowable elongation (deformation) at higher as compared with lower strain rates.

- Duration of loading: Excessive cyclic loading of bones is known to cause micro crack growth and increase fracture risk.
• Bone quality: Bone quality classification proposed by Lekholm and Zarb
  o Type 1 Homogenous compact bone
  o Type 2 Thick layer of compact bone surrounding a core of dense trabecular bone
  o Type 3 Thin layer of cortical bone surrounding a core of dense trabecular bone
  o Type 4 Thin layer of cortical bone surrounding a core of low-density trabecular bone. Type 4 bone is the least favorable for implants.

• Anatomical location: Trabecular architecture being either “rod like” or “plate like”, depending on the anatomical site, could be responsible for the differences between sites. Experimental data suggest that rod like structure is more susceptible to large deformations by bending and rotation of trabeculae than plate like structures. Current clinical practice routinely places the same-size dental implant diameter and geometry in the posterior and anterior mandible. This practice appears contraindicated given the inherent strength variations within human mandibular bone. Regional differences were noted in the human mandibular trabecular bone elastic modulus and ultimate compressive strength, exhibiting up to 47% to 68% higher mean values in the anterior compared with the posterior region of the mandible.

Peri-implant soft tissues
Soft tissue barrier at implants: The early formation of a longstanding, effective barrier capable of biologically protecting the peri-implant structures is mandatory to prevent oral bacteria and their products from penetrating into the body. Wound healing establishes an effective interface between living tissues and a foreign body. The interface consists of two zones, one of epithelium that covers about two mm of the surface and one devoted to connective tissue adhesion.

Healing process: After installation of the transmucosal implant component, the healing of the connective tissue wound involves four distinct processes: (1) formation and adhesion of a fibrin clot to the implant surface, (2) adsorption of extracellular matrix proteins and subsequently of connective tissue cells to the implant surface, (3) transformation of the clot into granulation tissue, and (4) migration of epithelial cells on top of the fibrin clot/granulation tissue. Once the epithelial cells reach the implant surface, their attachment occurs directly via a basal lamina (<200nm) and the formation of hemidesmosomes.

With endosteal dental implants, due to the absence of cementum and to the solid nature of trans mucosal implant components, there is no true anchorage of supra-alveolar connective tissue, but only a brittle adhesion. Consequently, the connective tissue adhesion at implant has a poor mechanical resistance compared to that of natural teeth. So the gingiva at implants is not attached. Since the connective tissue interface is considered of paramount importance in supporting the epithelium and blocking its apical migration, the lack of mechanical resistance can be a threat to the prognosis of dental implants. The following parameters can be hypothesized to have an impact on early soft tissue barrier formation:

1. Size of the exposed blood clot: A careful flap adaptation will reduce the gap, which will enable the epithelial cells to reach the implant surface sooner.
2. Mechanical stability of the wound borders: Any mobility of the flap can tear off the brittle adhesion of the fibrin clot to the transmucosal implant components.
3. Pressure of provisional removable prosthesis: If a removable prosthesis leans on the peri-implant soft tissues, masticatory movements will repeatedly destroy the fragile adhesion of the fibrin clot and induce a rapid apical migration of the biological width.

Conclusion
Dental implants pierce the oral mucosa, thus establishing a transmucosal connection between the external environment and the inner parts of the body. To avoid bacterial penetration through this transmucosal piercing, the early formation of a long-standing effective barrier is of paramount importance and forms a critical part of tissue integration. Primary implant stability between bone and implant may be the essential feature that permits the transfer of stress from the implant to the bone without any appreciable relative motion. Bone resorption around endosseous implants tends to increase with 180μm or more of excess height of a functioning superstructure. There is the possibility of bone resorption around implants caused by excess occlusal trauma,
even when there is no inflammation in peri-
implant tissue.

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**References**


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