MINI REVIEW

BONE MASS AND THE MECHANOSTAT

Nikita Singh[1], Varun Wadhwani [2], Vaishnavi Rajaraman [3]
[1]Student, Maulana Azad Institute of Dental Sciences, New Delhi-110 002
[2]Student, Maulana Azad Institute of Dental Sciences, New Delhi-110 002
[3]Student, Bharati Vidyapeeth Dental College and Hospital, Navi Mumbai, Bharati Vidyapeeth University, Mumbai - 400614

ABSTRACT
The Mechanostat is generally a term describing the way in which the mechanical loading influences the bone structure by changing the mass (amount of bone) and the architecture (its arrangement) to provide a structure that resists the habitual loads with an economical amount of material. As changes in the skeleton are accomplished by the processes of formation (bone growth) and resorption (bone loss), the mechanostat models affect the influences on the skeleton by those processes, through their effector cells, osteocytes, osteoblasts, and osteoclasts. The term was invented by Harold Frost: an orthopedic surgeon. The still-evolving mechanostat hypothesis for bones inserts tissue-level realities into the former knowledge gap between bone's organ-level and cell-level realities. The mechanostat proposal is a seminal idea which fits diverse evidence but it requires critique and experimental study. This current review describes in detail about the mechanostat theory and bone changes during the different phases.

KEYWORDS
Mechanostat, bone remodeling, microstrain, stress, window

How To Cite This Article: NikitaS et al. Bone Mass and The Mechanostat**.** Int J Prostho Rehabil 2021; 2: 2: 6-8

Received: 23-06-21; Accepted: 25-07-21; Web Published: 22-12-21

Address of correspondence

Dr. Varun Wadhwani
Student, Department of Prosthodontics, Maulana Azad Institute of Dental Sciences, New Delhi-110 002

Mail id: varunw020595@gmail.com

Introduction

Bone is an organ that is able to change in relation to a number of factors, including hormones, vitamins, and mechanical influences. However, biomechanical parameters, such as duration of edentulous state, are predominant.[1–4] Awareness of this adaptability has been reported for more than a century. In 1887 Meier qualitatively described the architecture of trabecular bone in the femur. In 1888 Kulmann noticed the similarity between the pattern of trabecular bone in the femur and tension trajectories in construction beams.

History

Wolff, in 1892, further elaborated on these concepts and published, “Every change in the form and function of bone or of its function alone is followed by certain definite changes in the internal architecture, and equally definite alteration in its external conformation, in accordance with mathematical laws.” The modified function of bone and the definite changes in the internal and external formation of the vertebral skeleton as influenced by mechanical load were reported by Murry.[5] Therefore the external architecture of bone changes in relation to function, and the internal bony structure is also modified.

MacMillan and Parfitt[6] have reported on the structural characteristics and variation of trabeculae in the alveolar regions of the jaws. For example, the maxilla and mandible have different biomechanical functions. The mandible, as an independent structure, is designed as a force absorption unit. Therefore, when teeth are present, the outer cortical bone is denser and thicker, and the trabecular bone is coarser and denser. In contrast, the maxilla is a force distribution unit. Any strain to the maxilla is transferred by the zygomatic arch and palate away from the brain and orbit. As a consequence, the maxilla has a thin cortical plate and fine trabecular bone supporting the teeth. They also noted that the bone is densest around the teeth (cribriform plate) and denser around the teeth at the crest compared with the regions around the apices. Alveolar bone resorption associated with orthodontic therapy also illustrates the biomechanical sensitivity of the alveolar processes.[7] Generalized trabecular bone loss in the jaws occurs in regions around a tooth from a decrease in mechanical strain.[8]Orban[9] demonstrated a decrease in the trabecular bone pattern around a maxillary molar with no opposing occlusion compared with a tooth with occlusal contacts on the contralateral side. Bone density in the jaws also decreases after tooth loss. This loss is primarily related to the length of time the region has been edentulous and not loaded appropriately, the initial density of the bone, flexure and torsion in the mandible, and parafunction before and after tooth loss. In general, the density change after tooth loss is greatest in the posterior maxilla and least in the anterior mandible.

Cortical and trabecular bone throughout the body is constantly modified by either modelling or remodelling.[10]Modelling has independent sites of formation and resorption, and results in the change of the shape or size of bone. Remodelling is a process of resorption and formation at the same site that replaces previously existing bone and primarily affects the internal turnover of bone,

©2021International Journal of Prosthodontic Rehabilitation | Published byMMPublishers

including that region where teeth are lost or the bone next to an endosteal implant.[11,12] These adaptive phenomena have been associated with the alteration of the mechanical stress and strain environment within the host bone.[13,14] Stress is determined by the magnitude of force divided by the functional area over which it is applied. Strain is defined as the change in length of a material divided by the original length. The greater the magnitude of stress applied to the bone, the greater the strain observed in the bone.[15]Bone modelling and remodelling are primarily controlled, in part or whole, by the mechanical environment of strain. Overall, the density of alveolar bone evolves as a result of mechanical deformation from microstrain.

Frost's Theory

Frost [16]proposed a model of four histologic patterns for compact bone as it relates to mechanical adaptation to strain. The pathologic overload zone, mild overload zone, adapted window, and acute disuse window were described for bone in relation to the amount of the microstrain experienced. These four categories also may be used to describe the trabecular bone response in the jaws.

The bone in the acute disuse window loses mineral density, and disuse atrophy occurs because modelling for new bone is inhibited and remodelling is stimulated, with a gradual net loss of bone. The microstrain of bone for trivial loading is reported to be 0 to 50 microstrain. This phenomenon may occur throughout the skeletal system, as evidenced by a 15% decrease in the cortical plate and extensive trabecular bone loss consequent to immobilized limbs for 3 months.[17] A cortical bone density decrease of 40% and a trabecular bone density decrease of 12% also have been reported with disuse of bone.[18,19] Interestingly, bone loss similar to disuse atrophy has been associated with microgravity environments in outer space, because the microstrain in bone resulting from the Earth's gravity is not present in the "weightless" environment of space.[20] In fact, an astronaut aboard the Russian Mir space station for 111 days lost nearly 12% of his bone minerals.[21]

The adapted window (50–1500 microstrain) represents an equilibrium of modelling and remodelling, and bone conditions are maintained at this level. Bone in this strain environment remains in a steady state, and this may be considered the homeostatic window of health. The histologic description of this bone is primarily lamellar or load-bearing bone. Approximately 18% of trabecular bone and 2% to 5% of cortical bone are remodelled each year[22] in the physiologic loading zone, which corresponds to the adapted window. This is the range of strain ideally desired around an endosteal implant, once a stress equilibrium has been established Bone turnover is required in the adapted window, as Mori and Burr [23] provide evidence of remodelling in regions of bone microfracture from fatigue damage within the physiologic range.

The mild overload zone (1500–3000 microstrain) causes a greater rate of fatigue microfracture and increase in the cellular turnover rate of bone. As a result, the bone strength and density may eventually decrease. The histologic description of bone in this range is usually woven or repaired bone. This may be the state for bone when an endosteal implant is overloaded and the bone interface attempts to change the strain environment.[11] During the repair process the woven bone is weaker than the more mature, mineralized lamellar bone. Therefore, while bone is loaded in the mild overload zone, care must be taken because the "safety range" for bone strength is reduced during the repair.[12]

Pathologic overload zones are reached when microstrains are greater than 3000 units. Cortical bone fractures occur at 10,000 to 18,000 microstrain (1%–2% deformation). Therefore, pathologic overload may begin at microstrain levels of only 18% to 40% of the ultimate strength or physical fracture of cortical bone. The bone may resorb and form fibrous tissue, or when present, repair woven bone in this zone, because a sustained turnover rate is necessary. The marginal bone loss evidenced during implant overloading may be a result of the bone in the pathologic overload zone. Implant failure from overload may also be a result of bone in the pathologic overload zone.

Conclusion

Declining bone density increases the risk of fractures of bones in older adults. A person's physical performance is known to be closely related to bone density, and a relationship between the physical performance and the oral function is also known to exist. However, there currently is a lack of evidence regarding the relationship between bone density and the oral function.

Authors Contribution

Nikita Singh: Manuscript drafting, Literature search, data collection
Varun Wadhwani: Data Analysis, manuscript drafting
Manuscript editing

Acknowledgement

The authors would thank all the participants for their valuable support and thank the dental institutions for the support

Conflict Of Interest All the authors declare no conflict of interest

Source Of Funding None

References

[1] ROBERTS WE. Implants: Bone physiology and metabolism. Cda j. 1987;15:54-61.
[2] Klemetti E, Vainio P, Lassila V, Alhava E. Trabecular bone mineral density of mandible and alveolar height in postmenopausal women. European Journal of Oral Sciences. 1993 Jun;101(3):166-70.
[3] Mercier P, Inoue S. Bone density and serum minerals in cases of residual alveolar ridge atrophy. The Journal of Prosthetic Dentistry. 1981 Sep 1;46(3):250-5.
[4] Atwood DA. Reduction of residual ridges: a major oral disease entity. Journal of Prosthetic Dentistry. 1971 Sep 1;26(3):266-79.
[5] Physical Therapy 1938, 18, 281.

[6] Parfitt GJ. An investigation of the normal variations in alveolar bone trabeculation. Oral Surgery, Oral Medicine, Oral Pathology. 1962 Dec 1;15(12):1453-63.
[7] Harris EF, Baker WC. Loss of root length and creastal bone height before and during treatment in adolescent and adult orthodontic patients. American Journal of Orthodontics and Dentofacial Orthopedics. 1990 Nov 1;98(5):463-9.
[8] Neufeld JO. Changes in the trabecular pattern of the mandible following the loss of teeth. The Journal of Prosthetic Dentistry. 1958 Jul 1;8(4):685-97.
[9] Orban B. Oral histology and embryology. South Med J. 1955;48(1):99.
[10] Yaeger JA. Principles of bone remodeling; an account of post-natal growth and remodeling processes in long bones and the mandible. JAMA. 1963 Jun 15;184(11):906-.
[11]Roberts WE, Smith RK, Zilberman Y, Mozsary PG, Smith RS. Osseous adaptation to continuous loading of rigid endosseous implants. American journal of orthodontics. 1984 Aug 1;86(2):95-111.
[12] Garetto LP, Chen J, Parr JA, Roberts WE. Remodeling dynamics of bone supporting rigidly fixed titanium implants: a histomorphometric comparison in four species including humans. Implant dentistry. 1995 Jan 1;4(4):235-43..
[13]Rhinelander FW. The normal circulation of bone and its response to surgical intervention. Journal of Biomedical Materials Research. 1974 Jan;8(1):87-90.
[14] Currey JD. Effects of differences in mineralization on the mechanical properties of bone. Philosophical Transactions of the Royal Society of London. B, Biological Sciences. 1984 Feb 13;304(1121):509-18..
[15] Bidez MW, Misch CE. Force transfer in implant dentistry: basic concepts and principles. The Journal of oral implantology. 1992 Jan 1;18(3):264-74.
[16] Martin RB, Burr DB. Structure, function, and adaptation of compact bone. Raven Press; 1989.
[17] KAZARIAN LE, VON GIERKE HE. 7 Bone Loss as a Result of Immobilization and Chelation: Preliminary Results in Macaca Mulatta. Clinical Orthopaedics and Related Research®. 1969 Jul 1;65:67-75.
[18]Minaire PP, Meunier P, Edouard C, Bernard J, Courpron P, Bourret J. Quantitative histological data on disuse osteoporosis. Calcified tissue research. 1974 Dec;17(1):57-73.
[19]Uhthoff HK, Jaworski ZF. Bone loss in response to long-term immobilisation. The Journal of bone and joint surgery. British volume. 1978 Aug;60(3):420-9.
[20] Simmons DJ, Russell JE, Winter F, Tran Van P, Vignery A, Baron R, Rosenberg GD, Walker WV. Effect of spaceflight on the non-weight-bearing bones of rat skeleton. American Journal of Physiology-Regulatory, Integrative and Comparative Physiology. 1983 Mar 1;244(3):R319-26..
[21] Oganov VS. Modern analysis of bone loss mechanisms in microgravity. Journal of gravitational physiology: a journal of the International Society for Gravitational Physiology. 2004 Jul 1;11(2):P143-6.
[22] Roberts WE, Stanford CM. Bone physiology, metabolism, and biomechanics in implant therapy. InPrinciples and Practice of Single Implant and Restorations 2014 Jan 1 (pp. 53-73). WB Saunders.
[23] Mori S, Burr DB. Increased intracortical remodeling following fatigue damage. Bone. 1993 Mar 1;14(2):103-9.

This work is licensed under the Creative Commons Attribution-Non Commercial 4.0 International License. To view a copy of this license, visit http://creativecommons.org/licenses/by-nc/4.0/ or send a letter to Creative Commons, PO Box 1866, Mountain View, CA 94042, USA.