DOWNLOAD PDF BIOLOGICAL MECHANISMS OF TOOTH MOVEMENT AND CRANIOFACIAL ADAPTATION

Chapter 1 : Dr Munira Xaymardan - The University of Sydney

Biological mechanisms of tooth movement and craniofacial adaptation: proceedings of the international conference held at the Great Southern Hotel, Columbus, Ohio, May , Zeev Davidovitch The Ohio State University, College of Dentistry, - Medical - pages.

King The goal of this chapter is to examine the relationships between orthodontic biomechanics and the underlying biological processes. The topics discussed include the factors affecting the rate of tooth movement, anchorage considerations, causes of relapse, and root resorption. All relevant biological principles underlying orthodontic tooth movement can be characterized as tissue remodeling. The process of orthodontic tooth movement is a resultant dynamic change in the shapes and composition of the investing bone and soft tissues. The dental and peridental tissues dentin, cementum, periodontal ligament [PDL], and alveolar bone all have active reparative mechanisms and will adapt under the normal forces of orthodontic appliances. This is accompanied by transiently increased tooth mobility and, occasionally, radiographic evidence of mild root resorption. Experienced clinicians also expect a certain amount of relapse to occur following orthodontic treatment. Other types of natural tooth migration commonly encountered are eruption of primary and succedaneous teeth as well as mesial or distal drifting of teeth. These physiological processes are not necessarily stimulated by biomechanical signals. In rare instances teeth fail to erupt or move in response to forces i. Each of these common clinical findings can be explained with a better understanding of the underlying biological principles that determine tooth movement. Tooth Movement Clinical Responses Kinetics of Orthodontic Tooth Movement From a clinical perspective orthodontic tooth movement has three distinct phases: The classic curve has three phases: Minimal tooth movement occurs during the first two phases and most of the tooth movement occurs during the acceleration and linear phases, when alveolar bone remodeling occurs. The timeline is approximate, with considerable individual variation due to mechanical and biological differences. The initial reaction of a tooth following force application is almost instantaneous within a fraction of a second and reflects the immediate movement of the tooth within the viscoelastic PDL cradle. These movements are generally predictable by biophysical principles and typically do not involve extensive amounts of tissue remodeling or deformation of the investing alveolar bone. Age is another factor affecting displacement. It is suggested that this might reduce the biological response of the PDL and thus delay tooth movement in adults. A, Varying root length L will cause shifts in the positions of the distance of the center of rotation CROT to the cervix a and the distance of the center of resistance CRES to the cervix x. Ultimately the patterns of tooth displacement will be determined by the change in the position of CRES produced by changes in alveolar bone height or root length. Patterns of initial tooth displacements associated with various root lengths and alveolar bone heights. Am J Orthod Dentofacial Orthop. The second phase of the orthodontic tooth movement cycle is characterized by the absence of clinical movement and is generally referred to as the delay or latency phase. During this period there is no tooth movement but extensive remodeling occurs in all tooth-investing tissues. The absolute amount of force applied is not as relevant as the relative force applied per unit area. Depending on the localized compression of the PDL, there can be either 1 a partial occlusion of the blood vessels in the area or 2 an absolute occlusion of blood vessels when high excessive forces have been applied. In cases of partial blockage, the blood vessels delivering nutrients to the area have the capacity to adapt to the new environment and can undergo angiogenesis to bypass occluded areas. However, complete occlusion of vascular flow leads to temporary necrosis of the immediate area and follows a completely different pathway of tooth movement, which is slower to be initiated, starting after approximately 1 to 2 weeks. In either situation, structural and biochemical changes initiate a cascade of cellular mechanisms required for bone remodeling. Aging has been shown to substantially affect the proliferative activity of the PDL cells and subsequent tooth movement, particularly during the delay phase. Yet once tooth movement had reached the linear phase, the rate of tooth movement became equal in both groups. This indicates that the

clinically observed increase in orthodontic treatment time for adults can be attributed primarily to the delay phase prior to the onset of tooth movement but the rate of migration is equally efficient once tooth movement has started. The third phase of the orthodontic tooth movement cycle is characterized by rapid tooth displacement. Tooth movement is initiated in deference to the adaptation of the supporting PDL and alveolar bone changes. Studies on the bone-resorptive osteoclast response following orthodontic appliance activation indicate that when appliance reactivation occurs during the appearance of reactivation osteoclasts, a second cohort of osteoclasts can be recruited immediately. This causes immediate, significant tooth movement with no greater risk of root resorption. Lower forces can induce tooth translation without a lag phase at rates that are still clinically significant. Equally as important as magnitude, however, is the timing of the force application. The force regimen has more influence on the rate of orthodontic tooth movement than the force magnitude. Conversely the application of intermittent forces creates a fluctuating environment of cellular activity and quiescence Fig. Additionally, it is recognized that very low forces produce lower rates of tooth movement then higher forces Fig. Exceeding this optimal force does not result in substantially greater rates of tooth movement. This threshold may differ between individuals as demonstrated in experiments on beagle dogs where it was noted that 25 cN of force caused greater tooth movement than 10 cN of force in one animal but not in another Fig. Tooth movement with light continuous and discontinuous forces in beagle dogs. Eur J Oral Sci. Ankylosis In rare cases a tooth may not move at all, regardless of the amount of external force applied on it. The contact point is a direct fusion of the cementum layer to the cortical bone of the tooth socket. Apart from idiopathic ankylosis, the primary cause of ankylosis is extrinsic localized trauma. Consequences of this condition include progressive resorption of the root with replacement by bone replacement resorption and arrested growth of the alveolar process in growing patients. Individuals with congenitally missing succedaneous permanent teeth characteristically exhibit infraocclusion and overretention of ankylosed deciduous teeth. If these localized regions of boneâ€”tooth attachment can be overcome with sufficient force application, the remainder of the tooth that does have PDL support can proceed toward a normal pattern of tissue remodeling and tooth movement. Principles of Anchorage in Orthodontics The biomechanics of orthodontics are not always designed for the purpose of moving teeth. Several types of anchorage are used in orthodontics: Dental anchorage is a term applied to the intentional minimization of migration of specific teeth through the supporting alveolar bone structure. The following section elaborates on dental anchorage since it is based on the premise of biological adaptation to orthodontic forces. Dental anchorage can be increased either by increasing the number of teeth consolidated into the anchor unit or by intentionally angulating specific teeth to better resist movement, or both. In general, teeth with greater root surface area tend to move less when used to move teeth with less root surface area. This occurs because the ability to resist movement is directly related to the periodontal fibers and bone surface area engaged in withstanding tooth movement. When forces are light and distributed over large surface areas, the compression on underlying periodontal structures leads to a partial vascular occlusion of the system and a transient ischemia. Although limited, there is still oxygenation to the area, enabling the microsystem to adapt and recruit new blood vessels for initiation of frontal resorption to occur. Movement of teeth with frontal resorption occurs within 3 to 4 days. However, when hyalinization of bone occurs in areas of periodontal compression during force application, there is a significant retardation of tooth movement while undermining resorption occurs. In this case the resistance to tooth movement is due to complete vascular occlusion in the compression area causing localized necrosis of bone and undermining resorption. When this happens the teeth start moving only after 12 to 15 days of bone remodeling. Therefore anchorage preparation is affected by the magnitude of forces applied, the total root surface area of the teeth upon which the forces are applied, and the angulation of the teeth. Increasing numbers of adults are seeking orthodontic treatment today and in these cases anchorage becomes a critical concern. Extraoral anchorage appliances are not usually a feasible alternative for these individuals. Therefore the clinician must maximize all available resources, such as the engagement and colligation of second molars and third molars, if present into the dental anchorage unit as well

as the use of palatal anchorage devices such as the Nance acrylic button appliance. Also in multidisciplinary cases, implants and other fixed restorative devices can and should be incorporated into the treatment plan for use as anchorage units during orthodontics. Finally, the introduction of TADs has provided substantial advantages for anchorage preservation in adults and adolescents alike and has opened up novel approaches to orthodontic biomechanics in complex cases. There is no evidence that the intra-alveolar phase of tooth movement during physiological eruption, drift, or relapse is mediated by pathological processes. Hyperemic changes are not restricted to the periodontal tissues adjacent to compression but have also been variously described in the adjacent marrow spaces and the dental pulp. Tooth Movement Without Injury The most obvious course of physiological tooth movement is the intra-alveolar eruption of teeth. As the crown of a tooth completes mineralization and begins its process of migration through the alveolar bone, it becomes enclosed in a crypt. This crypt is translated bodily by a combined effort of osteoclastic bone resorption along the path of eruption and osteoblastic bone formation on the path that the crown has already taken. The rate-limiting factor of the earliest intraosseous stage of tooth eruption is bone resorption and eruption can be accelerated or retarded by the local delivery of factors that alter the rate of osteoclastic activity. As teeth erupt into the oral cavity, and even throughout life, there is a natural tendency for them to continue moving along the path of least resistance until they encounter an obstacle of resistance. Usually this barrier comes in the form of interproximal contact from an adjacent tooth or occlusal contact from a tooth in the opposing arch. In the absence of this resistance there will be continued mesiodistal tipping or supraeruption, depending on the location of deficient contact. Studies have shown that mesial drifting of a tooth can have clinical significance on its morphological composition. In the process of mesial migration, tensional forces on the distal root surfaces may account for the increased cementum thickness on the distal surfaces of mesially drifted teeth. There was concurrent deposition of disorganized woven bone at the top of the interradicular septa, at the bottom of the sockets, and along the modeling sides. Tooth Movement with Injury Necrotic lesions at compression sites in the PDL space have been described in the early literature documenting the histological changes accompanying orthodontic tooth movement. This period is coincident with the delay phase of the tooth movement cycle. Specialized phagocytic cells are recruited and migrate to the site to remove these necrotic lesions. These cells remove the injured tissue from the periphery, resulting in the resorption of not only the necrotic soft tissue lesion but also the adjacent alveolar bone and cementum. In addition to the PDL, such sites can be found naturally in the craniofacial complex at sutures and artificially at sites of osteodistraction. Tensile forces are known to initiate an exuberant osteogenic response at these locations, with the first bone being deposited on the stretched soft tissue scaffold Fig. Through remodeling processes new compact bone is eventually deposited at these sites. This so-called consolidation process is slow to occur and therefore tends to lag behind the tissue removal activity that is simultaneously occurring at compression sites. The clinical result is the prevalence of increased mobility in teeth that are actively being treated orthodontically. This difference in timing between tissue removal and osteogenesis also accounts for the need to retain teeth that have recently been moved. A, Initial changes are characterized by stretching of the principal periodontal ligament PDL fibers, seen here as linear orientation of cell nuclei adjacent to the tooth. B, Later changes show deposition of bone on the stretched PDL fibers, oriented perpendicular to the tooth and socket wall arrows. Bn, Alveolar bone; T, tooth root. C, The three-dimensional organization of these initial bony spicules can be appreciated with a scanning electron micrograph of the alveolar bone on the socket wall after removal of the tooth and PDL. The micrograph is looking into the socket with the tension socket wall on the right. In addition to bone remodeling, histological evidence has shown that initial root resorption occurs in the peripheries of the necrotic PDL following orthodontic treatment Fig.

DOWNLOAD PDF BIOLOGICAL MECHANISMS OF TOOTH MOVEMENT AND CRANIOFACIAL ADAPTATION

Chapter 2 : Faculty Directory â€º UConn Health

*Biological Mechanisms of Tooth Movement and Craniofacial Adaptation. Proceedings of the Second International Conference Held at Tara's Ferncroft Conference Resort. Danvers, Massachusetts October , [Ze'ev Davidovitch, James Mah] on calendrierdelascience.com *FREE* shipping on qualifying offers.*

By year Book Chapters Cox, S. A method for investigating the cellular response to cyclic tension or compression in three-dimensional culture. Journal of the Mechanical Behavior of Biomedical Materials, 88, Characterization of inter-crystallite peptides in human enamel rods reveals contribution by the Y allele of amelogenin. Journal of Structural Biology, 1 , Translational Vision Science and Technology, 7 2 , A novel cell-stiffness-fingerprinting analysis by scanning atomic force microscopy: Histochemistry and Cell Biology, 6 , Journal Of Oral Pathology and Medicine, 44 8 , Arecoline is cytotoxic for human endothelial cells. Journal Of Oral Pathology and Medicine, 43 10 , Clinical and Experimental Ophthalmology, 42 6 , PloS One, 9 6 , Materials Science and Engineering C, 33 6 , Opposite cytokine synthesis by fibroblasts in contact co-culture with osteosarcoma cells compared with transwell co-cultures. Cytokine, 62 1 , Translational Vision Science and Technology, 2 5 , TNF-alpha and TGF-beta synergistically stimulate elongation of human endothelial cells without transdifferentiation to smooth muscle cell phenotype. Cytokine, 61 1 , A Finite Element Analysis. PloS One, 8 3 , Description of comprehensive dental services supported by the Medicare Chronic Disease Dental Scheme in the first 23 months of operation. Finite element analysis suggests functional bone strain accounts for continuous post-eruptive emergence of teeth. Archives of Oral Biology, 57 8 , Membrane and cytoplasmic marker exchange between malignant neoplastic cells and fibroblasts via intermittent contact: Journal of Pathology, 4 , Cell Stem Cell, 9 6 , Comparison of microsuture, interpositional nerve graft, and laser solder weld repair of the rat inferior alveolar nerve. Journal of Oral and Maxillofacial Surgery, 69 6 , ee Dental Infection and Vascular Disease. Seminars in Thrombosis and Hemostasis, 37 3 , A novel primate model of delayed wound healing in diabetes: Journal of Clinical Microbiology, 48 5 , Salivary arecoline levels during areca nut chewing in human volunteers. Journal Of Oral Pathology and Medicine, 39 6 , Vascularity during wound maturation correlates with fragmentation of serum albumin but not ceruloplasmin, transferrin, or haptoglobin. Wound Repair and Regeneration, 18, Physiotherapeutic treatment improves oral opening in oral submucous fibrosis. Journal Of Oral Pathology and Medicine, 38, The anti-apoptotic activity of albumin for endothelium is inhibited by advanced glycation end products restricting intramolecular movement. Cellular and Molecular Biology Letters, 14 4 , Journal of Oral and Maxillofacial Surgery, 66, Negative Feedback for Endothelial Apoptosis: Journal of Vascular Research, 45 3 , Effects of pulsed electromagnetic field vibration on tooth movement induced by magnetic and mechanical forces: Australian Dental Journal, 52 4 , Interferon-a and interferon-y sensitize human tenon fibroblasts to mitomycin-c. Investigative Ophthalmology and Visual Science, 48 8 , Resection of an orbital rim intraosseous cavernous hemangioma and reconstruction by chin graft and resorbable fixation plate. Ophthalmic Plastic and Reconstructive Surgery, 23 3 , The anti-apoptotic activity of albumin for endothelium is mediated by a partially cryptic protein domain and reduced by inhibitors of G-coupled protein and PI-3 Kinase, but is independent of radical scavenging or bound lipid. Journal of Vascular Research, 44 4 , The application of synchrotron radiation induced x-ray emission in the measurement of zinc and lead in Wistar rat ameloblasts. Archives of Oral Biology, 52 10 , The Crisis in Dental Health. Developing Practice, 18, Investigative Ophthalmology and Visual Science, 47 3 , Marked differences in the structures and protein associations of lymphocyte and monocyte CD4: Resolution of a novel CD4 isoform. Immunology and Cell Biology, 84 2 , Surgical management of an ameloblastoma in soft tissues of the cheek. Expression of mRNA for osteoprotegerin and receptor activator of nuclear factor kappa beta ligand RANKL during root resorption induced by the application of heavy orthodontic forces on rat molars. American Journal of Orthodontics and Dentofacial Orthopedics, 4 , Marked structural and functional heterogeneity in CXCR4: Immunology and

Cell Biology, 83 2 , Ultrastructural changes in endothelium during apoptosis indicate low microembolic potential. Journal of Vascular Research, 42 5 , Infection and Immunity, 72 4 , Histological aspects of human enamel fissure caries studied by CLSM. Microscopy and Analysis, 17 1 , Induction of contact-dependent endothelial apoptosis by osteosarcoma cells suggests a role for endothelial cell apoptosis in blood-borne metastasis. Journal of Pathology, 3 , Adipogenic healing in adult mice by implantation of hollow devices in muscle. The Anatomical Record, 1 , Microvasculature in gingivitis and chronic periodontitis: Microscopy Research and Technique, 56 1 , Nitric oxide and prostacyclin are released by cultured apoptotic human umbilical vein endothelial cells consistent with an anti-micro-thrombotic potential. Thrombosis and Haemostasis, , Synergistic induction of apoptosis in human endothelial cells by tumour necrosis facftor-a and transforming growth factor-B. Cytokine, 18 5 , Fluorometric and mass spectrometric analysis on nonenzymatic glycosylated albumin. Biochemical and Biophysical Research Communications, , Human endothelial cells maintain anti-aggregatory activity for platelets during apoptosis. Thrombosis and Haemostasis, 85, Reactive pocket epithelium in untreated chronic periodontal disease: Journal Of Oral Pathology and Medicine, 30, Dysregulated inflammation predicts reduced connective tissue growth factor expression in a baboon wound healing model of type 1 diabetes. Bolitho nee Donald , C. The anti-apoptotic activity of albumin for endothelium: The biological effects of mechanical vibration on alveolar and cranial bone remodeling - a combined study of tooth movement and defect healing. To update your profile click here. For support on your academic profile contact.

Chapter 3 : Larry D. Crouch, Ph.D. | College of Dentistry | University of Nebraska Medical Center

Get this from a library! Biological mechanisms of tooth movement and craniofacial adaptation: proceedings of the international conference held at the Great Southern Hotel, Columbus, Ohio, May ,

The need for cancer resections and the widespread occurrence of accidental and battlefield injuries affecting the craniofacial complex, all require significant surgical intervention to restore the proper form and function of the jaws and dentition. As such, the need for effective Tissue Engineering and Regenerative Medicine TERM therapies for craniofacial jaw and tooth regeneration are a significant health concern. To date, reliable methods to repair human jaws and teeth are quite limited, and exhibit significant deficiencies including extensive healing times, the need for multiple sequential surgeries, limited and often inadequate cell and bone tissue sources, donor site morbidities associated with autologous bone harvesting, and tooth regeneration therapies that are largely limited to ankylosed dental implants, which are not suitable for many individuals who have undergone extensive mandibular and maxillary bone resorption, and which do not exhibit any of the properties of natural teeth. As such, identifying reliable methods to regenerate alveolar bone and teeth is a significant health priority for treatment for a variety of genetic, age-associated and injury based craniofacial defects. Here we will build upon our previously published reports to validate reliable methods to use decellularized tooth bud scaffolds for coordinated alveolar bone and tooth regeneration. Validate bioengineered 3D hydrogel tooth bud constructs for tooth and jaw bone regeneration. Here we will build upon our published reports to validate GelMA and silk hydrogel scaffolds for coordinated alveolar bone and tooth regeneration. In addition, heterotopic ossification HO of soft tissues is a common health issue associated with aging, and in calcific arterial valve disease CAVD , osteoarthritis, and atherosclerosis. Create and validate FOP zebrafish. The objective of the studies proposed here is to test how inhibition of each of these genes affects FOP disease progression in two aims: Identify and sequence a validation cohort of additional mild FOP patients. We also believe that tyrosine-derived polycarbonate based scaffolds TyrPCs are a superior scaffold choice for craniofacial jaw and tooth repair, due to their superb properties including: Craniofacial jaw and dental defects represent a serious health issue, since proper function and aesthetics of the craniofacial complex is required for eating, communication, and psychosocial interactions. The purpose of our study is to devise improved methods for repair of craniofacial jaw and tooth defects in a timely and highly functional manner. The scope of our study is to devise improved methods for efficient and effective repair and regeneration of craniofacial jaw and tooth defects incurred in the line of duty. Fabricate scaffolds for optimized alveolar bone formation Demonstrate hDSC viability on scaffolds using in vitro methods. Demonstrate dental hDSC viability on scaffolds using in vivo methods. Demonstrate alveolar bone formation on in vivo hDSC seeded scaffolds. Publications Original Reports in chronological order: Somatic Cell and Molecular Genetics 12 2: Experimental Cell Research Molecular and Cellular Biology 7: Both P1 and P2 protamine genes are expressed in mouse, hamster and rat. Biochimica et Biophysica Acta Molecular and Cellular Biology 8: Journal of Cell Biology Molecular Reproduction and Development 1: Journal of Heredity J Bone Min Res 10 8: Mechanisms of Development Developmental Dynamics 2: Journal of Dental Research 80 Biochem Biophys Res Commun 5: Neuroscience Methods, 2: Journal of Dental Research, 81 Tissue engineering of complex tooth structures on biodegradable polymer scaffolds. Developmental Dynamics Cover Photograph Cells, Tissues and Organs Journal of Dental Research 83 7: Archives in Oral Biology, 50 2: Developmental Biology 2: Developmental Dynamics 4: Tissue Engineering 11 Epub Mar American Journal of Pathology 6: Dent Clin North Am Dent Clin North Am 50 2: EMBO Journal 26 Epub Oct Tissue Eng Part A. Journal of Dental Research 87 8: Tissue Engineering Methods 47 2: Journal of Oral and Maxillofacial Surgery 67 2: BMC Evolutionary Biology 10 1: The Journal of Applied Ichthyology Journal of Dental Research 90 2: Dec 13, Epub ahead of print. Epub Aug 1. Epub Apr Journal of Biomedical Materials Research A. J Mol Histology 43 1: Nov 23, [Epub ahead of print] Andreeva, V. Gene Expression Patterns Jan 25 [Epub ahead of

print]. J Mol Histol Apr 4 [Epub ahead of print]. Biomaterials Jul;33 Journal of Dental Research Dec 91 Odontology Sept PLoS Genetics Jan 30; 10 1. Global process, tissue specific defects. Commissioned to Rare Diseases. Epub Feb Advances and perspectives in tooth tissue engineering. J Tissue Eng Regen Med. Developing a biomimetic tooth bud model. Dental cell sheet biomimetic tooth bud model. Journal of Dental Research Feb;96 2: Epub Dec Journal of Dental Research, Jan 1: Cold Spring Harb Perspect Med. Pray and Yelick, P. J Biomed Mater Res A. Developmental Dynamics, Invited Review Feb; 2: Epub Dec 1. Developmental Biology, Dev Biol. Zebrafish Journal In Review. Journal of Dental Research In Revision. Zebrafish Journal In Preparation. In Preparation Lee, Y. Books and Other Monographs: Developmental Dynamics In Review. Biomaterials and Regenerative Medicine. Published by Cambridge University Press. Epub April Journal of Regenerative Medicine 4 5: Lin, Y and Yelick, P. Dental Clinics of North America. Cellular and Developmental Biology. Nuclear Structure and Function. Zbarsky, Plenum Press, New York.

Chapter 4 : Commitment to Privacy - Virginia Commonwealth University

If searching for the ebook Biological Mechanisms of Tooth Movement and Craniofacial Adaptation. Proceedings of the Second International Conference Held at Tara's Ferncroft Conference Resort.

Nanocalcium sulfate and collagen biomaterials: Effects on osteoblastic cells. Dent Oral Craniofac Res. The effect of ascorbic acid on bone cancer cells in vitro. Int J Dent Oral Health. Tissue Eng Part A. Synthesis and characterization of nanocrystalline calcium sulfate for use in osseous regeneration. Effects of platelet-derived growth factor, vitamin D and parathyroid hormone on osteoblasts derived from cancer patients on chronic bisphosphonate therapy.. Int J Mol Med. Platelet-derived growth factor enhancement of two alloplastic bone matrices. Effects of sphingosinephosphate and lysophosphatidic acid on human osteoblastic cells. Prostaglandins Leukot Essent Fatty Acids. Role of protein kinase C alpha in primary human osteoblast proliferation. J Bone Miner Res. Judith Lampasso, Marzec, N. Journal of Bone and Mineral Research. Role of Voltage-Activated Calcium Channels. International Journal of Oral Biology. Int J Oral Biol. Molecular characterization of the alpha1 subunit of the L type voltage calcium channel expressed in rat calvarial osteoblasts. Prostaglandins Leukotrienes and Essential Fatty Acids. Carpio, LC, Dziak R. Carpio, L, Dziak R. Archives of Oral Biology. Zhang, W, Dziak R. Current Opinions in Periodontology. Inositol trisphosphate receptor gene expression and hormonal regulation in osteoblast-like cell lines and primary osteoblastic cell cultures. Regulation by Protein Kinase C. Carpio L, Dziak R. Plasminogen Activators in Osteoblastic Cells. Calcium Currents in Ostroblastic Cells: Dependence Upon Cellular Growth Stage. Stephan E, Dziak R. Biochemical and Molecular Mediators of Bone Metabolism. Prostaglandins, Leukotrienes and Essential Fatty Acids. Evidence for B2 Receptors. Effects of inositol trisphosphate on calcium mobilization in bone cells. Ren, W, Dziak R. Effects of Leukotrienes on Osteoblastic Cell Proliferation. Hagel-Bradway, S, Dziak R. Biochemical and Biophysical Research Communications. Role of Calcium and cAMP. Tatakis DN, Dziak R. Cytosolic Calcium and Phosphoinositides. Leukotrienes in Orthodontic Tooth Movement. American Journal of Orthodontics. Effects of Phenytoin on Osteoblastic Cell Calcium. J Bone Mineral Research. Benzo B ThiopheneCarboxylic Acid: Biological effects of a purified lipopolysaccharide from Bacteroides gingivalis.. Calcitonin Effects on Bone Cell Calcium. Research Communications in Chemical Pathology and Pharmacology. Dziak R, Stern, PH. Cytoplasmic Glucorticoid Receptors in Bone Cells. Calcium Transport in Isolated Bone Cells. Dziak R, Stern PH. Biochem Biophys Res Comm. Dziak R, Brand JS. Bone Cell Isolation Procedures. Plenum Publishing, New York.. Rosemary Dziak, Farr, D. Gum Gingiva in Encyclopedia of Human Biology. Effects of leukotrienes on osteoblastic cells: Cytosolic calcium and cyclic AMP studies. International Journal of Oral Biology.. See all 91 more Books and Book Chapters: Calcification in Biological Systems. Encyclopedia of Human Biology. Prostaglandins as Mediators of Bone Cell Metabolism.. Calcium in Biological Systems. Host Bacterial Interactions in Periodontal Diseases. Out of date dental implants; Surface optimization and re-sterilization.. Berk J, Dziak R. Effects of bisphosphates Etidronate on human alveolar bone derived osteoblasts. Toxicity of formocresol and ferric sulfate on proliferating osteoblasts. Effects of enamel matrix protein on human osteoblastic cells. Inhibition of p38 MAPK reduces alkaline phosphotase in differentiating osteoblasts. Effects of cranioplastic relining resins on proinflammatory cytokine production. Symposium Robert J Genco. Tissue-Engineering of Bone Graft Material: Modifications of Calcium Sulfate. Effects of synsthetic nonceramic hydroxyapatite on human osteoblastic cells. Sphingolipids mitogenic effects on osteoblastic cells via MAPK activation. J Bone Min Res. InsP3 receptor subtype analysis in human osteosarcoma cells.. Inositol trisphosphate receptor subtype analysis in human osteosarcona cells.. Selective expression of InsP3 receptor gene in human osteosarcoma cells.. Lampasso JD, Dziak R.

Chapter 5 : 5 Biological Mechanisms in Orthodontic Tooth Movement | Pocket Dentistry

Biological mechanisms of tooth movement and craniofacial adaptation proceedings of the second international conference held at Tara's Ferncroft Conference Resort, Danvers, Massachusetts, October ,

Hartsfield received his D. Following a medical genetics fellowship resulting in an M. Hartsfield studied orthodontics for three years at Harvard University. During this time he began research in the causes of oral and facial defects as a research fellow in pediatrics at the Massachusetts General Hospital, receiving from Harvard his certificate in orthodontics and a Master of Medical Science degree. Following a postdoctoral fellowship in medical genetics at the University of South Florida U. College of Medicine in Tampa, Dr. There he received a Physician Scientist Award from the National Institutes of Health, which supported his continued studies into the causes of birth defects leading to a Ph. Hartsfield has been a reviewer for 24 different dental and medical journals. He was the guest editor in for the Seminars in Orthodontics volume on Genetics in Orthodontics. In preparation for submission. Submitted to Angle Orthod. Orthodontic mechanotransduction and the role of the P2X7 receptor. Biological Mechanisms of Tooth Movement. Anat Rec, in press. Comparison of reliability in anatomic landmarks identification utilizing two-dimensional digital cephalometrics and three-dimensional cone beam computed tomography in vivo. J Dentomaxillofacial Radiol, in press. Angle Orthod, in press. Am J Orthod Dentofacial Orthop, in press. A comprehensive analysis of normal variation and disease causing mutations in the human DSPP gene. The three-dimensional mechanical environment of orthodontic tooth movement and root resorption. Am J Orthod Dentofacial Orthop Major gene and multifactorial inheritance of mandibular prognathism. Treatment complexity index for assessing the relationship of treatment duration and outcomes in a graduate orthodontics clinic. Orthodontics and External Apical Root Resorption. Treatment outcomes in a graduate orthodontic clinic for cases defined by the American Board of Orthodontics malocclusion categories. Orthod Craniofac Res Dental Care for People with Osteogenesis Imperfecta. Follow-Up Study for Classes of American Journal of Orthodontics and Dentofacial Orthopedics, Eur J Orthod Remodeling of Mineralized Tissues, Part I: Physiologic interaction of root resorption, remodeling, tooth movement and ankylosis. Orthodontics Current Principles and Techniques, fourth edition. Dentistry for the Child and Adolescent, eighth edition. Clinical Genetics for the Dental Practitioner. Comprehensive clinical evaluation as an outcome assessment for a graduate orthodontics program. Biomechanics of root resorption. Bone Development and Function: Genetic and Environmental Mechanisms. Crit Rev Oral Biol Med Miniature implants for orthodontic anchorage. J Dent Res Refinement of the NHS locus on chromosome Xp Eur J Hum Genet Development of the vertical dimension: Morphometric characteristics of subjects with oral clefts and their relatives. Cleft Lip and Palate. From Origin to Treatment. Oxford University Press, Oxford. Dental Care for Persons with OI. Am J Med Genet The Ephx1d allele encoding an ArgCys substitution is associated with heat lability. Mouse models for craniofacial anomalies. Davidovitch Z and Mah J, eds. Harvard Society for the Advancement of Orthodontics, Boston. Dentistry for the Child and Adolescent, seventh edition. Cleft Palate Craniofacial J De Novo10q22 interstitial deletion. J Med Genet Assignment of microsomal epoxide hydrolase EPHX1 to human chromosome 1q Cytogenetics and Cell Genetics Partners with reciprocal translocations: Genetic counseling for the "double translocation. Genetic linkage of dentin dysplasia type II to chromosome 4q. J Cranio Genet Dev Biol Multidisciplinary management of congenital and acquired compensated malocclusions: Diagnosis, etiology and treatment planning. J Indiana Dent Asso 76 2: Induction of microsomal epoxide hydrolase activity in inbred mice by chronic phenytoin exposure. Biochem Mol Med Discordant levels of CGG repeat mosaicism in two brothers. Premature exfoliation of teeth in childhood and adolescence. Failure of transcutaneous electrical nerve stimulation in the conventional and burst modes to alter digital skin temperature. Arch Phys Med Rehabil Pleiotropy in Coffin-Lowry syndrome: Sensorineural hearing deficit and premature tooth loss as early manifestations. Biochem Med Metab Biol Lowe syndrome in a female with a balanced X: Mapping of the X chromosome breakpoint. Am J

Hum Genet Intestinal hypoperistalsis megacystis-microcolon type. Blackwell Scientific Publications, Inc. Gingival fibromatosis-deafness, Jones type. Comparison of two methods for linear measurement of ossified tissue in cleared mouse fetuses. J Craniofac Genet Dev Biol 8: Gingival fibromatosis with sensorineural hearing loss: Hypertelorism associated with holoprosencephaly and ectrodactyly. J Clin Dysmorphol 2: Bilateral macrostomia in one of monozygotic twins.

DOWNLOAD PDF BIOLOGICAL MECHANISMS OF TOOTH MOVEMENT AND CRANIOFACIAL ADAPTATION

Chapter 6 : Home - School of Dental Medicine - University at Buffalo

Description: Biological Mechanisms of Tooth Movement, Second Edition is an authoritative reference to the scientific foundations underpinning clinical orthodontics. Led by an expert editor team and with contributions from an international group of contributors, the book covers key topics including bone biology, the effects of mechanical.

By year Book Chapters Xaymardan, M. Bone Marrow Stem Cell: In Anthony Atala Eds. Clinical and Therapeutic Applications, pp. United States of America: Bone Marrow Stem Cells: Stem Cells and Development, 25 17 , Stem Cells International, , Stem Cells and Development, 24 8 , Journal of Developmental Biology, 2 2 , PloS One, 9 6 , Uterine cells are recruited to the infarcted heart and improve cardiac outcomes in female rats. Journal of Molecular and Cellular Cardiology, 52 6 , Cell Stem Cell, 9 6 , Vascularity during wound maturation correlates with fragmentation of serum albumin but not ceruloplasmin, transferrin, or haptoglobin. Wound Repair and Regeneration, 18, Stem Cells, 27 8 , In vitro characterization of adult cardiac progenitors and flux after ischemic injury. BDNF-mediated enhancement of inflammation and injury in the aging heart. Physiological Genomics, 24 3 , Circulation, 18 , Restoration of cardiac angiogenesis in aging female mice: BDNF-mediated enhancement of inflammation andinjury in the aging heart. Adipogenic healing in adult mice by implantation of hollow devices in muscle. The Anatomical Record, 1 , Canadian Cardiovascular Society Consensus Conference American Diabetes Association Bolitho nee Donald , C. For support on your academic profile contact.

DOWNLOAD PDF BIOLOGICAL MECHANISMS OF TOOTH MOVEMENT AND CRANIOFACIAL ADAPTATION

Chapter 7 : Professor Hans Zoellner - The University of Sydney

Biological Mechanisms of Tooth Movement, Second Edition is an authoritative reference to the scientific foundations underpinning clinical orthodontics.

Neuroendocrine modulation of inflammatory processes Stress-induced immunosuppression Molecular regulation of estrogen deficient post-menopausal bone loss Grants: June 1, to May 31, Larry Crouch, July 1, to June 30, Human osteogenic protein-1 induces osteogenic differentiation of adipose-derived stem cells harvested from mice. Arch Oral Biol Speed of human tooth movement is related to stress and IL-1 gene polymorphisms. Am J Orthod Dentofacial Orthop 6: Tooth movement and cytokines in gingival crevicular fluid and whole blood in growing and adult subjects. Am J Orthod Dentofacial Orthop 4: Role of IL in acute lung inflammation. Exogenous and endogenous nitric oxide but not iNOS inhibition improves function and survival of ischemically injured livers. J Invest Surg 14 5: Interleukin-1 gene polymorphisms and orthodontic tooth movement: What does DNA tell you about bone remodeling? J Dent Hygiene J Immunol 5: The velocity of human orthodontic tooth movement is related to stress magnitude, growth status and the ratio of cytokines in gingival crevicular fluid. Davidovitch Z, Mah J. Trends in teaching biochemistry to dental students: Survey results from North American basic science course directors. Preparing students for critical thinking: Accessing and appraising biomedical evidence. Stress magnitude and interleukin-1 affect the speed of orthodontic tooth movement in humans. OPInduced osteogenic differentiation of adipose-derived stem cells. The relationship among stress, GCF cytokines, and velocity of tooth movement in humans. Interleukin-1 gene cluster polymorphisms and velocity of orthodontic tooth movement. Interleukin-1 gene cluster polymorphisms and orthodontic tooth movement. Nicotine suppresses CGRP signaling os osteoblast-like cells. VonWald, L, Crouch, L. Influence of genotype on interleukin-1 receptor antagonist in gingival crevicular fluid. Nicotine and calcitonin gene related peptide receptor mRNA expression in osteoblast-like cells. Proceedings of the Nebraska Academy of Sciences, Cytokine production in whole blood and primary cultures of periodontal ligament cells. Nicotine and calcitonin gene related peptide receptor mRNA expression in osteoblast-like Cells. J Bone Miner Res Suppl 1:

DOWNLOAD PDF BIOLOGICAL MECHANISMS OF TOOTH MOVEMENT AND CRANIOFACIAL ADAPTATION

Chapter 8 : Biological mechanisms of tooth movement and craniofacial adaptation (edition) | Open Library

Biological mechanisms of tooth movement and craniofacial adaptation Article in American Journal of Orthodontics and Dentofacial Orthopedics (1) Â· July with 15 Reads DOI: /mod.

The interface between orthodontics and periodontolgy is as broad as any between two disciplines in clinical dentistry. Controlled tooth movements utilize the dynamic nature of the periodontium which remodels actively when external forces are applied. Periodontal tissues are also continuously at risk from plaque-induced diseases that compromise the support of the dentition. The accumulation of plaque is enhanced when patients wear orthodontic appliances and the dental hygienist has a vital role to play before, during, and after orthodontic treatment. Changes in tooth relationships are often a consequence of periodontal disease. When the condition has been controlled orthodontic treatment may be indicated to, for example, align drifted incisors, or to upright molars that have tipped into extraction spaces. The objective of this book is to explore fully the interrelationships between the periodontium in both health and disease, and orthodontics. It is not a text that is aimed purposely towards the specialist management of orthodontic or periodontal cases. We have instead examined exhaustively the common ground upon which the orthodontist and periodontist are often called upon to meet and collaborate on cases that demand multi-disciplinary care. The text is presented in two parts. Part I comprises three chapters covering the basic sciences. Chapter 1 deals with the anatomy and physiology of the peridontium and investigates the role of dental anatomy in the initiation and progression of periodontal disease. Chapter 2 summarizes the microbiology and pathogeneisis of periodonatla disease and it is evident that many of the biological mediators which are involved in the perpetuation of disease are also implicated in the day-to-day remodelling of the supporting tissues. Chapters 3 studies the histological aspects of orthodontic tooth movements and looks at the biological mechanisms involved. Part II comprises the remaining six chapters. Chapter 4 discusses the exmaination and treatment planning for the orthodontic management of patients who have a history of periodontal disease. Chapter 5 deals with different concepts of occlusion and how common occlusal problems can initiate periodontal problems. The importance of controlling periodontal disease before orthodontic treatment is explained in Chapter 6. Aids to oral hygiene for the orthodontic patient and the periodontal complications of orthodontic treatment are described. Chapter 7 covers the orthodontic management of specific periodontal problems and the periodontal effects of surgical adjuncts to orthodontic therapy are outlined in Chapter 8. The final chapter discusses aspects of relapse, the need for retention, and the soft tissue procedures that have been advocated in the prevention of relapse. This book will serve as a helpful volume for all specialists in the orthodontic and periodontal disciplines. It is also directed towards postgraduate students and practitioners with a special interest in these subjects.

Chapter 9 : Zeev Davidovitch (Author of Biological Mechanisms Of Tooth Movement And Craniofacial Adap

In Z. Davidovitch, J. Maah (eds.): Biological Mechanisms of Tooth Movement and Craniofacial Adaptation (The 4th International Conference). Boston: Harvard Society for the Advancement of Orthodontics,