Multiscale remodeling model for orthodontic movement

Emílio G. F. Mercuri*, Mildred B. Hecke **, Lídia Carvalho **

*Environmental Engineering Department, Federal University of Paraná (UFPR); mercuri@ufpr.br; Administration building, 3rd floor, room 7, School of Engineering, Polytechnic Center, Av. Cel. Francisco H. dos Santos, 100, 81530-980; Curitiba; Brazil;

**Bioengineering Group, Numerical Methods in Engineering Post Graduate Program (PPGMNE/UFPR); mildred@ufpr.br; Curitiba; Brazil;

***INESC-TEC and Faculty of Sciences of the University of Porto (FCUP); lidia.r.carvalho@inesctec.pt; Rua do Campo Alegre, 687, 4150-179 Porto, Portugal

Abstract

Bone remodeling is responsible for the removal of the micro-damage and reconstruction of the mineralized matrix to increase the useful life of mineralized tissues. This study sought to characterize the hierarchical, anisotropic, heterogeneous and multiscale constitutive behavior of the alveolar bone and cementum of teeth. A dynamic interaction model was adopted to describe the blasts and clasts interactions and the influence of paracrine signaling on the millimeter-sized representative volume element. The homogenization procedure can provide the macroscopic mechanical properties based on the composition of the microstructure of the material. A computer code was developed and implemented in MATLAB to represent the orthodontic remodeling around a central incisor. The spatial discretization of the two-dimensional geometry was performed using the Finite Element Method and the temporal evolution of biological variables and volume fractions was solved by the fourth-order Runge-Kutta method. The mechanical stimulus used to trigger cellular activity was the strain energy density at the microscale. The results show the temporal evolution of the volume fractions distribution in the cortical bone and cementum and also reveal the strain energy density distribution in the periodontal ligament (PDL) during remodeling. The distribution of strains in the PDL is influenced by remodeling of neighboring tissues. This research reveals some novelty in simulating the remodeling around tooth in orthodontic movement, not only around the bone media but also around the tooth root. The study was able to mimic the mechano-transduction at the mesoscale. It contributes to understand the mechanisms governing dental movement so orthodontists might better control tooth movement. DOI: https://doi.org/10.24243/JMEB/4.2.204

1 Introduction

Bone and cellular cementum are living tissues capable of adapting to the mechanical environment and are characterized as composite materials, because of its complex composition and hierarchical levels of organization. Biological factors that influence the bone removal and replacement balance are diverse. Among them are the cells, cytokines, growth factors, hormones, proteins and lipids that interact in this complex local phenomenon that undergoes systemic influences and external stimuli [1]. The tooth root cementum is a thin, mineralized tissue that covers the root

*Corresponding Author : Emílio G. F. Mercuri
Email Address: mercuri@ufpr.br
dentin and is surrounded by the periodontium (PDL). It consists as a cellular cementum on the cervical root and cellular cementum covering the apical root. Acellular cementum is critical for tooth attachment to the adjacent periodontal ligament (PDL), while cellular cementum is hypothesized to play a role in post-eruptive tooth movement and adaptation to occlusion [2].

Orthodontic forces induce an aseptic inflammatory response [3] and generate stress and strain in the extracellular matrix that can activate cells from tissues surrounding the tooth, facilitating tooth movement. It’s a diverse biological environment, wherein applied forces yield remodeling of both mineralized and non-mineralized paradental tissues, including the associated blood vessels and neural elements [4]. This research seeks to understand the mechanisms that govern the dental movement so orthodontists might be able to accelerate or decelerate tooth movement by using physical, chemical, or surgical methods and still yet manage to keep treatment safe. There is a lack of information in the literature regarding models capable of representing the remodeling around the teeth. The authors did not find models able to capture clinically observed phenomena such as tooth root and cementum reabsorption.

The objective of this work is to propose a constitutive model for mineralized tissues that can represent the remodeling process of the orthodontic movement. The mathematical formulation is based on the micromechanics of the continuum and the biological processes are described by a cellular interaction model between osteoclasts, osteoblasts, cementoclasts and cementoblasts. Specific objectives are: (i) develop a model for the interaction of blasts and clasts from cellular cementum and alveolar bone; (ii) perform the coupling of the micromechanical stimulus with the model of cellular interactions; (iii) apply the developed theory, using homogenization and localization rules, to modify extravascular volume fractions by clasts and blasts cells activities.

2. Mathematical model for cells interactions

Some authors [5] presented a system of ordinary differential equations that modeled cellular interactions. Seven cell groups were considered, representing the different temporal stages of the bone remodeling process. However, many aspects of cellular interactions have not been described by their model and remain open. In this sense, other authors [6] made an adaptation to the previous model, adding a new differential equation to describe the variation of bone volume. In the same sequence, another work [1] introduced new functions that allow changing the sensitivity of the system to mechanical signals, such as strain energy. The main influence of strain energy is on the proliferation of osteoblasts and the production of RANKL, a protein that controls cell proliferation. Although cells micromechanical environment is considered a dynamic milieu of biophysical stimuli that includes strain, stress, shear, pressure, fluid flow, streaming potentials and acceleration [7], only the strain energy was considered in this study.

For the simulations it was considered that the cells were present only in cortical bone and cellular cementum. These are the only dynamic materials which can change properties in the present work. The variables considered were: $B_u; B_p; B_a; C_p$ and $C_a$, where $B_u$ represents undifferentiated blasts (undifferentiated progenitors of osteoblasts or undifferentiated progenitors of cement to blasts); $B_p$ indicates the precursors of blasts (precursors of osteoblasts or precursors of cementoblasts); $B_a$ is the symbol for active blasts (active osteoblasts or active cementoblasts); $C_p$ denotes the pre-clasts (pre-osteoclasts or pre-cementoclasts) and $C_a$ represents the active clasts (active osteoclast or active cementoclast). Cell populations were expressed in terms of concentrations defined as the number of cells in a representative volume element (RVE) divided by the volume of the RVE. The unit used for concentrations was pM, Picomolar ($10^{-12}$ mol/dm$^3$).

The concentrations of blastic or clast cells increase or reduce the volume fractions $f_i$, respectively. The volume fractions were defined by $f_i = V_i/V_{total}$, where $V_i$ represented the volume fraction of phase $i$ and $V_{total}$ was the total volume, $V_{total} = \Sigma_i V_i$. For cortical bone and for the cementum, it was considered the micromechanical representations presented by [1], where the expression $f_{exvas} + f_{vas} = 1$ was valid. $f_{exvas}$ was the volume fraction of the extravascular bone matrix or extravascular cement matrix and $f_{vas}$ was the volume fraction of haversian canals or intertrabecular space or cementum vascularity.

The system of coupled ordinary differential equations of the model is presented in Eqs. (1)-(4):
\[
\frac{dC_{Ba}}{dt} = \mathcal{D}_{Bu} C_{Bu} \pi_{act,Ba}^{TGF-\beta} + \mathcal{P}_{Bp} C_{Bp} \Pi_{\varepsilon_{exvas}} - \mathcal{D}_{Bp} C_{Bp} \pi_{rep,Bp}^{TGF-\beta}
\]  
\[
\frac{dC_{Ba}}{dt} = \mathcal{D}_{Bu} C_{Bu} \pi_{act,Ba}^{TGF-\beta} - \mathcal{A}_{Bu} C_{Ba}
\]  
\[
\frac{dC_{Ca}}{dt} = \mathcal{D}_{Cp} C_{Cp} \pi_{act,Ca}^{RANKL} - \mathcal{A}_{Ca} C_{Ca} \pi_{act,Ca}^{TGF-\beta}
\]  
\[
\frac{df_{exvas}}{dt} = k_{form} C_{Ba} - k_{res} C_{Ca}
\]

where \(\mathcal{D}_{Bu}, \mathcal{D}_{Bp}\) and \(\mathcal{D}_{Cp}\) are the rates of differentiation of progenitors of undifferentiated cells. \(\mathcal{P}_{Bp}\) is the proliferation rate of pre-osteoblasts or pre-cementoblasts. Constants \(\mathcal{A}_{Bu}\) and \(\mathcal{A}_{Ca}\) represent the apoptosis rates of active cells. The symbols \(\pi_{act,Bu}^{TGF-\beta}\), \(\pi_{act,Ca}^{TGF-\beta}\) and \(\pi_{rep,Bp}^{TGF-\beta}\) are the activation and inhibition functions that regulate the differentiation of blasts and apoptosis of clasts by TGF-\(\beta\), while \(\pi_{act,Cp}^{RANKL}\) is the activation function regulating the differentiation of osteoclasts by the RANK-RANKL-OPG signaling pathway. The parameters \(k_{form}\) and \(k_{res}\) represent the rates of bone formation and resorption, i.e., percentage of bone turnover per pM of cells per time. The mechano-regulation function \(\Pi_{\varepsilon_{exvas}}\) will be discussed in the next subsection. Fig.1 illustrates the mathematical modeling of the biomechanical regulation functions, which are explained in section 3.

\[P_{RANKL-exvas} \text{ [pM/day]}\]

Fig.1 Mechano-transduction diagram for cells interaction based on the MSED [8].

3. Micromechanics and self-regulation of mechanical stimulus

Mechano-transduction consists of two cell activities: first they act as micro strain sensors at the extravascular matrix level and second they send signals to other cells to activate the remodeling process. The microstrain energy density (MSED) at the microscale was the chosen stimulus for a cell to produce cytokines that, once signaled, stimulate cellular differentiation.

The representative volume element of the cortical bone and cementum is composed of a vascularized phase and a phase representing the extravascular matrix. The volume fraction and stiffness tensor of the vascularized phase are written as \(f_{vas}\) and \(\varepsilon_{vas}\), respectively. For the extravascular matrix, the volume fraction is \(f_{exvas}\) and the stiffness tensor is \(\varepsilon_{exvas}\). The elastic relations for the stress \(\sigma\) and strain \(\varepsilon\) of each phase are: \(\sigma_{exvas} = \varepsilon_{exvas} : \varepsilon_{exvas}\) for extravascular phase and \(\sigma_{vas} = \varepsilon_{vas} : \varepsilon_{vas}\) for vascular phase. The tensors \(\varepsilon_{exvas}\) and \(\varepsilon_{vas}\) for cortical bone and cementum were the same as those proposed by [1] with adaptation to the state of plane stress. The homogenization procedure to obtain macroscopic strain \(\mathbf{E}\) and macroscopic stress \(\mathbf{\Sigma}\) is showed in Eqs. (5)-(6).

\[\mathbf{E} = f_{exvas} \varepsilon_{exvas} + f_{vas} \varepsilon_{vas}\]
\[\mathbf{\Sigma} = f_{exvas} \sigma_{exvas} + f_{vas} \sigma_{vas}\]
Homogenized (macroscopic) stress and strain, $\Sigma$ and $E$, are related by homogenized (macroscopic) stiffness tensor $C^{\text{hom}}$ according to Eq. (7).

$$\Sigma = C^{\text{hom}} : E$$

(7)

The localization rule, used to obtain the extravascular strain, is showed in Eq. (8):

$$\varepsilon_{\text{exvas}} = A_{\text{exvas}}^{\text{est}} : E$$

(8)

The overall homogenized stiffness $C^{\text{hom}}$ is obtained according to Eq. (9) for the $r = 2$ phases.

$$C^{\text{hom}} = \sum_r f_r C_r : A_r^{\text{est}}$$

(9)

The concentration tensors $A_r^{\text{est}}$ can be adequately estimated through the matrix-inclusion problem of [9]-[11] in terms of the Mori-Tanaka scheme [12].

The biomechanical regulation functions, $\Pi_{\varepsilon_{\text{exvas}}}$ and $P_{\text{RANKL} \cdot \varepsilon_{\text{exvas}}}$ are shown in Eq. (10) and Eq. (11). The function of anabolic mechano-regulation (bone or cement formation), $\Pi_{\varepsilon_{\text{exvas}}}$, is defined as:

$$\Pi_{\varepsilon_{\text{exvas}}} = \begin{cases} 
\Pi_{\varepsilon_{\text{exvas} \cdot \text{st}}} & \text{if } w_{\epsilon_{\text{exvas}}}^1 < w_{\epsilon_{\text{exvas}}}^f \\
1 + \lambda_1 \left( \frac{w_{\epsilon_{\text{exvas}}}^f}{w_{\epsilon_{\text{exvas}}}^1} - 1 \right) & \text{if } w_{\epsilon_{\text{exvas}}}^f < w_{\epsilon_{\text{exvas}}} < w_{\epsilon_{\text{exvas}}}^2 \\
\lambda_2 \left( w_{\epsilon_{\text{exvas}}} - w_{\epsilon_{\text{exvas}}}^3 \right) + \Pi_{\varepsilon_{\text{exvas}}}^{\text{max}} & \text{if } w_{\epsilon_{\text{exvas}}} < w_{\epsilon_{\text{exvas}}}^f < w_{\epsilon_{\text{exvas}}}^4 \\
\Pi_{\varepsilon_{\text{exvas} \cdot \text{st}}} & \text{if } w_{\epsilon_{\text{exvas}}} > w_{\epsilon_{\text{exvas}}}^f 
\end{cases}$$

(10)

where $\Pi_{\varepsilon_{\text{exvas} \cdot \text{st}}}$ is the steady state value of the anabolic mechano-regulation function. The constants $\lambda_1$ and $\lambda_2$ are related to the inclinations of the straight lines in Fig. 2. $\lambda_1$ has no unit and $\lambda_2$ have units of Pa$^{-1}$. The function $\Pi_{\varepsilon_{\text{exvas}}}^{\text{max}}$ is the maximum value of the anabolic mechano-regulation function (elastic proliferation is limited, $\Pi_{\varepsilon_{\text{exvas}}}^{\text{max}} = 1$). The values $w_{\epsilon_{\text{exvas}}}^0$, $w_{\epsilon_{\text{exvas}}}^f$, $w_{\epsilon_{\text{exvas}}}^3$, and $w_{\epsilon_{\text{exvas}}}^4$ represent the deformation energy densities at the microscale in the beginning, middle, and end of the formation region (see Fig. 1). The function of catabolic mechano-regulation (bone or cement resorption), $P_{\text{RANKL} \cdot \varepsilon_{\text{exvas}}}$, is defined as:

$$P_{\text{RANKL} \cdot \varepsilon_{\text{exvas}}} = \begin{cases} 
0 & \text{if } w_{\epsilon_{\text{exvas}}} < w_{\epsilon_{\text{exvas}}}^1 \\
k_1 \left( 1 - \frac{w_{\epsilon_{\text{exvas}}}}{w_{\epsilon_{\text{exvas}}}^1} \right) & \text{if } w_{\epsilon_{\text{exvas}}}^1 < w_{\epsilon_{\text{exvas}} < w_{\epsilon_{\text{exvas}}}^2} \\
k_2 \left( \frac{w_{\epsilon_{\text{exvas}}}}{w_{\epsilon_{\text{exvas}}}^2} - 1 \right) & \text{if } w_{\epsilon_{\text{exvas}}} > w_{\epsilon_{\text{exvas}}}^2 
\end{cases}$$

(11)

where $k_1$ and $k_2$ are the slopes of dashed lines (Fig. 1). The values $w_{\epsilon_{\text{exvas}}}^1$ and $w_{\epsilon_{\text{exvas}}}^2$ represent the initial and final microstrain energy densities of the reabsorption phase (or catabolic phase), that is, in which clastic activity prevails. The function $P_{\text{RANKL} \cdot \varepsilon_{\text{exvas}}}$ is responsible for the resorption action of the elasts and is part of the function $\pi_{\text{act} \cdot c_p}^{\text{RANKL}}$, as described by Eqs. (12)-(14).

$$\pi_{\text{act} \cdot c_p}^{\text{RANKL}} = \frac{C_{\text{RANKL} - \text{RANK}}}{\beta_{\text{RANKL} + \text{RANKL} \cdot \varepsilon_{\text{exvas}} + P_{\text{RANKL} \cdot \varepsilon_{\text{exvas}}} + \beta_{\text{RANKL} \cdot \varepsilon_{\text{exvas}}}}$$

(12)

$$C_{\text{RANKL} - \text{RANK}} = \frac{1 + k_\text{RANKL} - \text{RANK}}{1 + k_\text{RANKL} - \text{RANK}}$$

(13)
RANKL_{eff} = n_{act,B}^{PTH} \left( R_{B_p}^{RANKL} C_{B_p} + R_{B_a}^{RANKL} C_{B_a} \right) \tag{14}

More information about the equations and constants of the model can be found in [13]. Table 1 shows the values of the parameters used in the numerical simulation. Besides the second parameter obtained from [8], all the others were numerically calibrated by the authors.

<table>
<thead>
<tr>
<th>Table 1</th>
<th>Parameters of the model</th>
</tr>
</thead>
<tbody>
<tr>
<td>Symbol</td>
<td>hydrostatic traction</td>
</tr>
<tr>
<td>$\Pi_{\text{exvas, st}}$</td>
<td>0.2</td>
</tr>
<tr>
<td>$\Pi_{\text{max}}$</td>
<td>1.0</td>
</tr>
<tr>
<td>$R_{\text{max}}^{\text{RANKL}, \text{exvas}}$</td>
<td>0.8</td>
</tr>
<tr>
<td>$w_{\text{exvas}}^1$</td>
<td>0.4</td>
</tr>
<tr>
<td>$w_{\text{exvas}}^2$</td>
<td>$1.2 \times 10^6$</td>
</tr>
<tr>
<td>$w_{\text{exvas}}^3$</td>
<td>$5.5 \times 10^1$</td>
</tr>
<tr>
<td>$w_{\text{exvas}}^4$</td>
<td>$1.5 \times 10^2$</td>
</tr>
<tr>
<td>$w_{\text{exvas}}^5$</td>
<td>$4.4 \times 10^5$</td>
</tr>
<tr>
<td>$w_{\text{exvas}}^6$</td>
<td>$1.2 \times 10^6$</td>
</tr>
</tbody>
</table>

4. Material and methods

Fig. 2 shows the different thresholds of the functions used in the model for hydrostatic traction and hydrostatic compression as different stimuli. The annotations R, DZ and F represent resorption, dead zone and formation, respectively. The first invariant of the strain tensor $I_1$ was used to characterize if the stress state was hydrostatic compression ($I_1 < 0$) or hydrostatic traction ($I_1 > 0$). The x-axis (Fig. 2) is in logarithmic scale because there was great dispersion of the MSED in the finite elements analysis. The distribution of MSED for both cortical bone and the cellular cementum are illustrated in Fig.2.

A Matlab code, named Remold 2D, was developed to represent the coupling of a micromechanical model with the biological model of cellular interaction. Stress and strain tensors were assessed by the Finite Element Method and the temporal evolution of biological variables and volume fractions was solved by the Runge-Kutta fourth-order method (RK4). The RK4 method has a local truncation error of the order of $h^5$, where $h$ is the time step of the model.

The geometric model of a frontal incisor was obtained from the scanning of a radiograph and it can be seen in the left image of Fig. 3. The two-dimensional model represents a cut in the mesial-distal direction, in the middle.
section of the central incisor. For this reason, the visualization of the orthodontic bracket, where the forces are applied, was omitted. The finite element model consists of 12,474 elements and 25,283 nodes. A force of 2.0 N [8] in the mesial-distal direction was applied to simulate the orthodontic load and zero displacement boundary conditions were applied in the contour of the trabecular bone (right image of Fig. 3).

![Image of geometric model and finite element mesh with applied load conditions.]

**Fig.3 Left - Geometric model; Right - Finite element mesh with applied load conditions.**

Table 2 shows the values of the initial constitutive constants used in the simulation of the orthodontic movement. Except cortical bone and cellular cementum, with dynamic properties, all the other tissues were modelled as linear elastic. However, biomechanically, the ligament demonstrates nonlinear viscoelasticity [14]-[15], this was not the focus of this research and for that reason it was considered linear elastic. The micromechanical representation for cortical bone and cellular cementum followed the work of [16] which considered the extravascular subscale with representative volume element of approximately 100μm. The volume fractions already described (f_{exvas} and f_{vas}) where considered as inputs for a one-step homogenization scheme which provides the upscaled macroscopic stiffness tensor. In this multiscale framework it was used an orthotropic stiffness matrix for the extravascular matrix of cortical bone and cementum, the same anisotropic behavior proposed by [1].

<table>
<thead>
<tr>
<th>Material</th>
<th>Young's Modulus</th>
<th>Poisson Coefficient</th>
</tr>
</thead>
<tbody>
<tr>
<td>trabecular bone [12]</td>
<td>1.37 GPa</td>
<td>0.30</td>
</tr>
<tr>
<td>cortical bone [12]</td>
<td>13.70 GPa</td>
<td>0.30</td>
</tr>
<tr>
<td>periodontal ligament [13]</td>
<td>0.68 MPa</td>
<td>0.47</td>
</tr>
<tr>
<td>dentin [12]</td>
<td>18.60 GPa</td>
<td>0.31</td>
</tr>
<tr>
<td>pulp [12]</td>
<td>1.37 GPa</td>
<td>0.30</td>
</tr>
<tr>
<td>enamel [13]</td>
<td>20.00 GPa</td>
<td>0.30</td>
</tr>
<tr>
<td>cellular and acellular cementum [14]</td>
<td>30.00 GPa</td>
<td>0.30</td>
</tr>
</tbody>
</table>

The system of Eqs. (1)-(4) were solved with the RK4 time marching scheme, with a time step of $h = 0.1$ day for 1000 iterations, totaling 100 days of simulation of the evolution of cellular concentrations. The concentrations of the cells $B_u$ and $C_p$ were constant and equal to $C_{B_u} = C_{C_p} = 1.0 \times 10^{-2}$ pM. The initial concentrations of the variable cell populations were: $C_{B_p} = 7.682 \times 10^{-5}$ pM, $C_{B_a} = 1.081 \times 10^{-4}$ pM and $C_{C_a} = 1.799 \times 10^{-4}$ pM.

Triangular plane elements, with 6 Gauss points, were adopted in a plane stress state finite element analysis. The macroscopic properties of the materials, Table 2, such as the modulus of elasticity and the Poisson coefficient, were chosen according to the literature [17]-[19]. The numerical application consisted of a transient two-dimensional...
analysis of the variation of the constitutive properties of cortical bone and cellular cementum. The remodeling cycle of one day was adopted for the variation of volume fractions and cell populations. In each day a constant load case in a static structural analysis was performed, together with the solution of the temporal evolution of the coupled model of differential equations of the cellular dynamics. As usual in a FEA analysis, the elementary stiffness matrices were allocated in the global stiffness matrix and the force vectors and null displacement contour conditions were considered. The system of algebraic equations was solved and the nodal displacements were obtained, a procedure that was performed in each remodeling cycle.

It was found a great variability in the value of the vascular volume fraction of bone and cementum in the literature [20]-[25]. Therefore, the range of variability (minimum and maximum values) of the vascular volume fractions are 0.050 - 0.300 for cortical bone [20]-[21] and 0.007 - 0.158 for cementum [25]. The initial vascular volume fractions adopted in the simulation are $f_{\text{vas}}^{\text{cortical bone}} = 0.17$ and $f_{\text{vas}}^{\text{cementum}} = 0.08$.

5. Results and discussion

Fig.4 shows the volume fraction variation in cortical bone and cellular cementum from day 1 to day 100, in intervals of 10 days. All the other tissues are colored in dark blue with no variation of mechanical properties.

The results of the evolution of the volume fraction in the cortical bone show that the regions where the volume fraction of the bone matrix decreased are mostly in state of compressive stresses and the regions where the volume fraction increased are mostly in traction stress state. These results are in agreement with the classic pressure-tension theory that explains the dental movement, described by [26]-[31].

The level of the strain energy density stimulus varies in materials with different rigidities, the same occurs in adjacent tissues such as cortical bone and PDL. The PDL has an important role as a connective tissue during tooth movement, it absorbs and transmits the loads and presents a stress relaxation behavior [32]. Fig.5 shows the mechanical strain energy density (SED) stimulus in the elements which belong to the periodontal ligament (PDL) on days 1 and 100 of simulation in the left and right sides of the model.
The results of the SED in the PDL show that the ligament does not receive homogeneous loading. In the region of the incisor neck, both on the right and left sides, there is a great variation and intensity of the mechanical stimulus. As the time passes there is an increase of the mechanical stimulus in the region closer to the cervical root and there is a decrease of the SED in the region of the apical root. This observation may be related with the spin that the teeth can make during orthodontic movement.

6. Conclusions

The application of the micromechanical theory with the model of cellular interaction in the microscale allowed to develop a methodology to estimate remodeling, exploring aspects of the bone and cementum microstructure. The coupling result of the micromechanical and biological models provides a research tool for the transient analysis of the evolution of volume fractions due to the action of blasts and clasts cells. The calibrated model was able to give distinct response in compression and traction.

This study reveals some novelty in simulating remodeling produced by orthodontic movement and to mimic the mechano-transduction done by clasts and blasts in the cortical bone and cellular cementum. The results showed a dispersion of the PDL strain energy near the cervical region of the dental root during remodeling. The decrease of the SED in the apical region of the PDL for both the compressive and traction sides indicates that cells may be able to facilitate stress relaxation.

There is still much effort to be made, for example, to model the fluid flow in the alveoli region which is related to the diffusion of proteins and growth factors. However, this work contributes to understand the mechanisms that govern the dental movement, so orthodontists might be able to accelerate or deaccelerate tooth movement and better comprehend resorption in cortical bone or in tooth root during orthodontic treatment.

Funding

This work is financed by FEDER Funds through the Competitiveness and Internationalization Operational Program - COMPETE 2020 under the project "POCI-01-0145-FEDER-006961" and by National Funds through FCT - Foundation for Science and Technology through the project UID / EEA / 50014/2013.

References


[26] Reitan K. The tissue reaction as related to the functional factor. The European Journal of Orthodontics. 2007 Apr 1;29(suppl_1):i58-64. DOI: https://doi.org/10.1093/ejo/cjl110


