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A hypothetical mechanism of bone remodeling and modeling under electromagnetic loads

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Abstract

A hypothetical regulation mechanism for bone modeling and remodeling under electromagnetic field is proposed. In this hypothesis, the bone modeling and remodeling mechanism is described as follows: the circular loads that we bear during ordinary daily activities generate micro-damage in cortical bone and these micro-cracks are removed by osteoclasts. Then growth factors, which are in latent forms in osteocytes, are activated by osteoclasts and released into bone fluid. These growth factors stimulate osteoblasts to refill the cavities. An electromagnetic field can stimulate the multiplication of growth factors and accelerate the bone remodeling process indirectly. It can be seen that many features reported in adaptive bone modeling and remodeling are explained by the proposed hypothesis. Further, a computational model is established based on the hypothesis, which can simulate the bone modeling and remodeling process under multi-field loads.

Keywords: Bone modeling and remodeling; Electromagnetic field; Biomaterial; Biomechanics

1. Introduction

During the past decades, it is recognized that more and more artificial bone materials are applied to clinical practice. The most important feature of those materials is their mechanical and biological compatibility with natural bone tissues. Although lots of work has been done on the mechanical and biological compatibility, very little progress has been made in the area of functional adaptation of bone tissues. To understand how environment can affect process of bone remodeling, mechanisms on reconstruction of bone materials is of great importance to not only the clinical practice, but also the design and manufacture of bone materials. This can result in a new bone material with better biological and mechanical compatibility. It can also direct the cultivation of bone tissues in vitro.

Functional adaptation of living bone refers to the ability of the tissue to respond to changes in its environment. For cortical bone tissue, one potential response is remodeling, that involving turnover of bone in small packets by basic multi-cellular units (BMU). Another response of cortical bone tissue is modeling, that refers to biological processes that produce functionally purposeful sizes and shapes of skeletal organs. Mostly in bone the processes involve independent resorption and formation modeling drifts. The chief purpose seems to be to fit organs to their mechanical usage so that the usage does not break them or make them hurt, and for a lifetime [1]. Pre-1964 literature did not distinguish between modeling and remodeling, lumping them together as remodeling. Since bones remodel themselves without the control of nervous system, the most interesting feature of this process is that the bone tissue seems to be capable of sensing the surrounding environment and controlling bone formation and resorption. Since the remodeling phenomenon was discovered by Wolff [2] in 1892, it has attracted widespread attention from biological scientists and mechanical engineering. Many hypotheses as to this mechanism have been proposed, among which the theory of adaptive elasticity [3–5], the electricity theory [6,7] and the fatigue damage theory [8–10] are the most popular and widely used.

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In recent years, much work has been done on the response of bone tissue to an extremely low frequency electromagnetic field [11–13]. Evidence has shown that a pulsed extremely low-frequency electromagnetic field can stimulate bone tissue to remodel itself. This feature has been widely applied in the treatment of skeletal diseases such as osteoporosis, tendonitis, osteonecrosis, fracture and non-union. It is noted that electromagnetic filed can also change the structures of bone materials. How the mechanism of bone's functional reconstruction is still open question and is of great importance to clinical practice. In this paper, we present a pilot study investigating the mechanism of bone modeling and remodeling under an electromagnetic field. A hypothesis for the regulating mechanism of bone modeling and remodeling is proposed to illustrate how the electromagnetic field affects the bone modeling and remodeling process. Bone research remains interdisciplinary by nature, and a deeper understanding of bone biology will ultimately lead to advances in the treatment of diseases and injuries to bone itself.

2. Hypothetical mechanism of bone remodeling

2.1. Bone growth factors

It has been reported that growth factors such as platelet derived growth factor (PDGF), insulin-like growth factor (IGF), bone morphogenetic protein (BMP), and transforming growth factor beta (TGF β) play an important role in bone formation and remodeling [14–17]. They are found in considerable quantities in bone matrix. Normally they are retained in osteocytes. Once the osteocytes are resorbed, the growth factors can be released into the bone fluid and can stimulate osteoblasts to refill resorption cavities. Experiments have shown that a pulsed extremely low-frequency electromagnetic field can stimulate the multiplication of growth factors [18–20]. Then indirectly it can accelerate the remodeling process via growth factors.

2.2. Bone electricity

Since 1957, when some bone tissues were found to have a piezoelectric effect [21], the electric properties of bone material have been widely investigated. It is believed that electric signals in bone tissue play an important role in the bone modeling and remodeling process [6,7,22–24]. These signals are generated in two ways: piezoelectricity and streaming potentials. Streaming potentials derive from the bone fluid flow, which is generated by bone material deformation and blood circulation. Evidence has shown that an increase in venous pressure results in an increase in the passage of fluid from capillary to bone matrix [25]. Increased extravascular perfusion could be a factor in increasing periosteal bone formation. This flux of fluid may increase streaming potentials in bone, acting as a signal to bone cells to increase bone formation. Experiments by Lanyon [26] have shown that cyclic loading induces more bone adaptation than static loading. Turner [27] performed experiments on cyclic loading of bone and determined that the stimulus for bone remodeling is proportional to the applied strain rate magnitude. Strain rate magnitude can be directly deduced from strain magnitude and frequency of loading. These phenomena can also be explained by bone electricity. It can be seen that both piezoelectricity and the streaming potentials have relations to strain.

2.3. Bone mechanostat

Early in 1987, Frost [28] proposed a hypothesis for describing bone remodeling which he updated twice, in 1996 [29] and 2003 [30]. In his hypothetic model termed "mechanostat", mechano-biologic negative feedback mechanisms would work under the control of a subject's mechanical usage. In doing so they would adjust skeletal architecture in a way that tended to prevent mechanical usage from causing structural failure of skeletal tissues and organs. It was proposed that mechanically dedicated message traffic would dominate the effects of most non-mechanical agents. Most (not all) non-mechanical agents would have permissive roles in affecting skeletal architecture and health. They could optimize or impair mechanical usage effects, but could not replace or duplicate them. The mechanism of this process is shown in Fig. 1, where "MU" denotes the skeleton's usual mechanical usage. Most systemic (S) agents reach the skeleton from the blood. Local (L) agents include local molecular-biological agents, related phenomena, and local innervations. "*MFL*" indicates a mechanical feedback loop, here one each for modeling (*MFLm*) and remodeling (*MFLr*).

The updated "mechanostat" indicated that signals were dependent on strains. Aided by sense systems that detect and process the signals, threshold ranges of the strain-dependent signals (the MESm for modeling and the MESr for disuse-mode remodeling) help to switch the two whole-bone-strength functions on and off. Fig. 2 shows how these features would usually affect bone strength. The horizontal line at the bottom suggests typical peak hone strains from zero on the left to the fracture strain on the right (Fx), plus the locations of the remodeling, modeling, and micro-damage thresholds (MESr, MESm, and MESp, respectively). The horizontal axis represents no net gains or losses of bone strength. The lower dotted line curve suggests how disuse-mode remodeling would remove bone next to

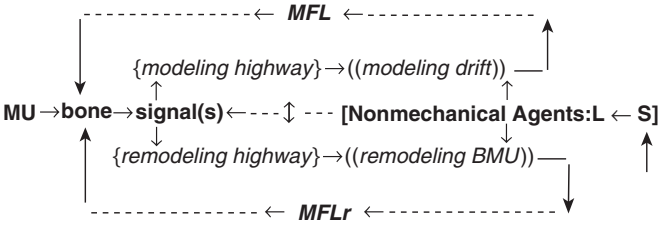

Fig. 1. Mechano-biologic negative feedback mechanisms [26].

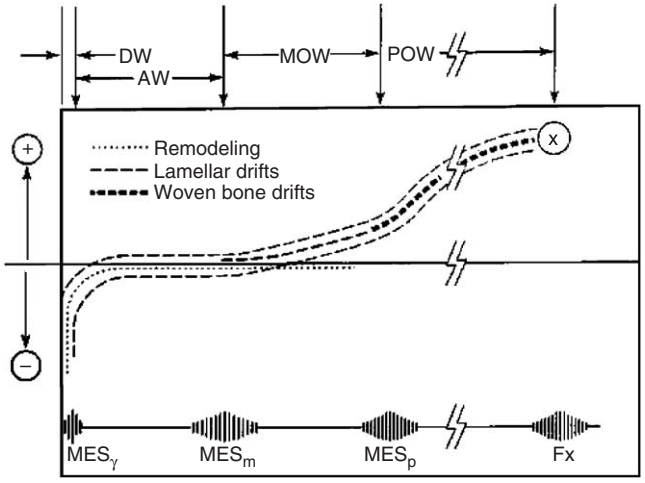

Fig. 2. Combined modeling and remodeling effects on bone strength [30].

marrow when strains remain below the MESr range, but otherwise would tend to maintain existing bone and its strength. The upper dashed line curve suggests how modeling drifts would begin to increase bone strength where strains enter or exceed the MESm range. The dashed outlines suggest the combined modeling and remodeling effects on bone strength. Beyond the MESp range, woven bone formation usually replaces lamellar hone formation. At the top, DW = disuse window and AW = adapted window, as in normally adapted young adults; MOW = mild overload window, as in healthy growing mammals; and POW = pathologic overload window. The strain span between the MESr and MESm represents the span between those features in bone's general biomechanical relations.

2.4. Adaptive bone modeling and remodeling

As we can see from the "mechanostat" model described above, it is a relatively mature hypothesis for bone modeling and remodeling mechanism. But it is far from perfect. It does not describe how local mechanical signals are detected and how they are translated to bone formation and resorption. Nor does it indicate what the signals and non-mechanical agents are during modeling and remodeling processes. Furthermore, although it distinguishes the strain thresholds of each mode, the reason for the existence of these thresholds existed is beyond its explanation capacity. In this paper, we define electric signals as the stimuli and growth factors as non-mechanical agents. Then the modeling and remodeling process of bone under electromagnetic loads can be shown, as follows.

Compact bone structures are susceptible to failure when subjected to cyclic loadings, which often generate micro-fractures. So the remodeling process should have a function to repair damage in osteonal bone. It is known that the bone resorption function is mainly attributed to osteoclasts and bone formation to osteoblasts. When bone tissue is damaged, osteoclasts remove necrotic osteocytes. Growth factors such as BMP or TGF β exist in latent forms in osteocytes. They are activated at the site of bone resorption by osteoclasts and released into the bone fluid. Osteoblasts are then stimulated by these growth factors to form bone and fill up resorption cavities. It has been proposed that under normal circumstances the generation of damage by loading and its repair by remodeling are able to reach an equilibrium state in which the damage burden waiting to be repaired is tolerable [30]. If the loading increases, more micro-cracks are generated and more osteocytes are removed. This results in more growth factors in the bone fluid to accelerate bone formulation and maintain the equilibrium state. It has also been observed that when loadings are excessive, accelerated remodeling not only removes damage at a higher rate, but also increases the rate of damage production [31]. In contrast, a decrease in loading can also result in fewer micro-cracks and subsequently less presence of growth factors. It can thus be seen that bone tissue can remodel itself well to protect itself from damage and keep its mass unchanged.

But where do the two strain thresholds come from and why can bone tissue change its mass and structure? Here we hypothesize that when the electric signals change within a certain range, the quantities of growth factors hidden in osteocytes remain unchanged. When the electric signals exceed this range, the quantities of growth factors in osteocytes will increase or decrease. Then the bone tissue begins to model itself. If the growth factors increase, more new bone tissue can be deposited and the bone mass will also increase. This can be considered as MESm. Similarly, MESr comes from the decrease of growth factors.

The theory developed here can be used to analyze the magnetoelectromechanical behavior of bone tissue in the modeling and remodeling process. Therefore, utilizing the nature of the feedback system of bone tissue, we can accelerate the healing process of fractures and non-unions by artificially introducing an environment similar to the fracture site of the bone.

3. Theoretical model of bone modeling

Based on the hypothesis above, we propose a computable model for the bone modeling and remodeling process. In this model, we define porosity, p, as the measure of bone changes. The relationship between porosity and the elastic modulus can be found as [32]

$$E = (8.83\times10^5)p^6 - (2.99\times10^6)p^5 + (3.99\times10^6)p^4 - (2.64\times10^6)p^3 + (9.08\times10^5)p^2 - (1.68\times10^5)p + 2.37\times10^4. \quad (1)$$

Then the rate of change of porosity, $\dot{p}$, is assumed to be a function of the mean bone resorbing (Q_R) and refilling (Q_F) rates for each BMU, and the density of resorbing (N_R) and refilling (N_F) BMUs/volume [33,34]

$$\dot{p} = Q_R N_R - Q_F N_F. \quad (2)$$

Here, the resorption (Q_R) and refilling (Q_F) rates are assumed to be linear in time. In cortical bone the BMU forms a cylindrical canal about 2000 μm long and 150–200 μm wide. It gradually burrows through the bone with a speed of 20–40 μm/day. At the tip, on the order of 10 osteoclasts dig a circular tunnel (cutting cone) in the dominant loading direction. An activated osteoclast is able to resorb 200,000 μm^3/day [35]. And then several thousand osteoblasts will fill the tunnel (closing cone) to produce an (secondary) osteon of renewed bone. In this way, between 2% and 5% of cortical bone is remodeled each year [36]. So it is easy to estimate the value of Q_R and Q_F.

Based on the analysis above, we propose following equation for calculating the number of osteoclasts N_R. It can be obtained by integrating over an appropriate period of the BMU activation frequency (f_a) history:

$$N_R = \int_0^t f_a(t')\,dt' + N_R^0, \tag{3}$$

where t is the time at which N_R is calculated. As proposed above, the resorption of osteocytes is activated by micro-damage. So the BMU activation frequency, f_a (BMUs/volume/time), is assumed to be a function of the existing state of damage,

$$f_a = f_{a(max)}(1 - e^{k_r \Phi}), \tag{4}$$

where $f_{a(max)}$ is the maximum activation frequency, $f_{a(max)} = 0.8$ BMUs/mm^3/day, and $k_r = -1.6$ defines the shape of the curve. Φ is defined here as environmental stimulus:

$$\Phi = C_{ij} s_{ij}^q R_L + (C_i E_i + G_i B_i) f_e, \tag{5}$$

where C_{ij}, C_i and G_i are the damage rate coefficients, s_{ij}, E_i and H_i are strains, intensity of electrical field and intensity of magnetic field, respectively. The value for the exponent q is set at a nominal value of 2/3. The mechanical loading rate, R_L, is assumed to be 3000 cycles per day (cpd), and f_e is the frequency of the electromagnetic field. N_R^0 represents the number of BMUs required to resorb the naturally timeworn osteocytes besides those that were destroyed by micro-damage.

The population N_F is found by multiplying the quantities of resorbed osteocyte by k_f:

$$N_F = k_f N_R, \tag{6}$$

where k_f is the correlation coefficient of the refilling BMUs which indicates the relation between the refilling and the resorbing process. k_f is defined as a piecewise function of Φ and p in our analysis:

$$k_f = \begin{cases} c_0, & \Phi_L \leqslant \Phi \leqslant \Phi_U, \\ c_1, & \Phi > \Phi_U, \\ (c_0 - c_2)\left(\frac{p}{p_0}\right)^n + c_2, & \Phi < \Phi_L. \end{cases} \tag{7}$$

Considering that the quantity of growth factors retained in osteocytes changes along with the environmental loads, as mentioned above, Φ_L and Φ_U can be considered as MESr and MESm, respectively. When $\Phi_L \leqslant \Phi \leqslant \Phi_U$, the growth factors remain unchanged ($k_f = c_0 = 1.0$) and the bone tissue is in the remodeling state. When $\Phi > \Phi_U$ (the upper limit of Φ), more growth factors ($k_f = c_1 = 1.2 > c_0$) are generated, which results in bone modeling. When $\Phi < \Phi_L$ (the lower limit of Φ), fewer growth factors ($k_f = (c_0 - c_2)(p/p_0)^n + c_2 < c_0$) result in a disuse mode of bone tissue, where $c_2 = 0.1$ in our analysis. The formula, $(c_0 - c_2)(p/p_0)^n$, indicates the influence of biological factors. If this formula vanishes, when Φ converges to zero, p will approach 1, which means that all the bone tissue is resorbed. However, as is well known, although a mass of bone loss is observed, bone tissue is not completely resorbed in the body of a patient who stays in bed for a long period of time. It is reasonable to predict that there must be some other factors contributing to bone remodeling as well as the mechanical factor. We assume it to be biological factors, which prevent complete bone resorption. The porosity of the remaining bone tissue is assumed to be $p_0 = 50\%$ and $0 < p \leqslant p_0$. $n = 5$ defines the shape of the curve (see Fig. 3).

It can be seen from Fig. 3 that as the porosity p increases, k_f also increases, which indicates that the bone tissue secretes more growth factors to deposit more bone material and restrain bone resorption.

This constitutive model is based on first-order, non-homogeneous, nonlinear differential equations (Eq. (2)), which, respectively, govern the evolutionary state variables porosity and damage. The environmental stimulus Φ is regarded as the forcing function. The rate equation (Eq. (2)) involves the BMU activation frequency, f_a, which itself is not an independent state descriptor, as it is algebraically related to p and Φ in Eqs. (4) and (7). The algorithm is implemented using a simple forward Euler scheme to integrate Eq. (2) with respect to time. The integral in Eq. (3) is calculated using the history of the daily

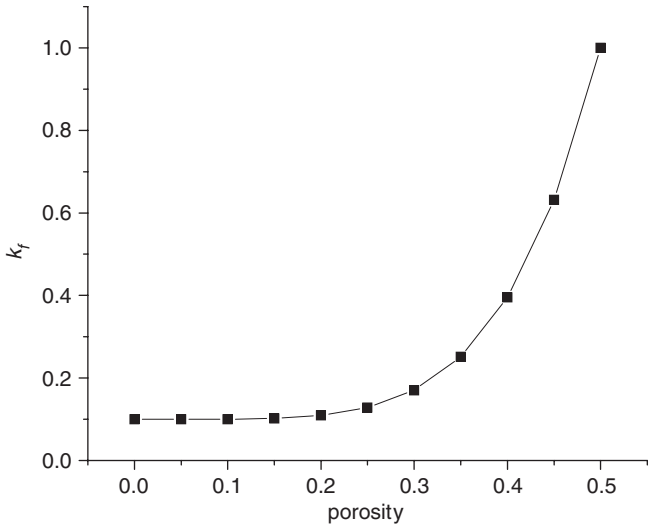

Fig. 3. The relationship between k_f and p in disuse-mode remodeling.

average activation frequency. Then it can be used to analyze the bone modeling and remodeling process. Numerical simulation follows in the next section.

4. Numerical examples

For the sake of simplicity, we consider a cubic bone section subjected to uniaxial compressive pressure P and pulsed electromagnetic loads. The length of its side a is 1 cm. We also assume that the strain s_{ij}, the electric field E_i and the magnetic field H_i all return to zero at the end of each load cycle, so that their ranges and peaks are the same. The model is given an initial porosity of 4.43% because this allows equilibrium between the Haversian canals removed and added by new BMUs [37,38]. This porosity produces an initial modulus of 17.8 GPa as determined by Eq. (1). A time increment of 0.5 day is examined to integrate Eqs. (2). The state variables and constants are shown in Table 1.

Bone resorption and formation can reach a proper equilibrium during the remodeling process, which keeps the bone mass unchanged. But it should be mentioned that environmental factors affect the bone remodeling process. An increase in Φ can result in a faster bone remodeling, and vise versa. In this study, we investigate only the modeling and disuse-mode remodeling of bone tissues. We distinguish following six loading cases:

(1) $P = 1.8, 1.9, 2.0, 2.1\,\mathrm{KN}, \quad E_i = 0, \quad H_i = 0.$

The results for this loading case are shown in Fig. 4. It can be seen that overloads can activate bone modeling. The porosity of bone tissue decreases when the environmental stimuli exceed the MESm, which is defined as the modeling threshold. Overloads result in a denser and stronger bone structure. The elastic module E increases due to the decrease in porosity. Then the environmental stimulus decreases at the same time as the strains become smaller. When porosity returns to the remodeling threshold it will not change any further. The result shows that bone tissue can model itself to force its strains to revert to the remodeling range. It can also be seen that the greater the pressure, the less porous the bone material. But it should be mentioned that if the loading is so great that the strain cannot be reduced to the remodeling range when the porosity reaches its lower limit, the bone structure would model itself in another way. This case is beyond the scope of this paper and will be discussed in our subsequent work.

(2) $P = 0, 0.05, 0.10, 0.15\,\mathrm{KN}, \quad E_i = 0, \quad H_i = 0.$

This case is investigated to demonstrate the bone disuse-mode remodeling process. The corresponding results are presented in Fig. 5. It can be seen from the figure that, as the loadings decrease, the bone materials become more porous to resist the decrease of environmental stimulus. But, as mentioned before, the porosity of bone tissue should be below a certain (or critical)

Table 1
Model state variables and constants

	State variables	
E	Elastic modular (Mpa)	
p	Porosity	
N_R	Number of resorbing BMUs (BMUs/mm^3)	
N_F	Number of refilling BMUs (BMUs/mm^3)	
f_a	BMU activation frequency (BMUs/mm^3/day)	
s_{ij}	Strain ($\mu\varepsilon$)	
Φ	Environmental stimulus (cpd)	
k_f	Correlation coefficient of the refilling and resorbing process	
	Constant	**Values in this study**
V	Volume of bone tissues (mm^3)	1000
Q_R	Resorbing rate of bone tissues (mm^3/day)	2.0×10^{-4}
Q_F	Refilling rate of bone tissues (mm^3/day)	1.0×10^{-6}
$f_{a(max)}$	Maximum BMU activation frequency (BMUs/mm^3/day)	0.8
k_r	Activation frequency dose-response coefficient	-1.6
R_L	Mechanical loading rate (cpd)	3000
C_{ij}, C_i, G_i	Damage rate coefficients	0.04,0.04,0.08
f_e	Frequency of electromagnetic field (Hz)	100
N_R^0	Number of naturally timeworn osteocytes (BMUs/mm^3)	0.4
c_0	Value of k_f during remodeling process	1.0
c_1	Value of k_f during modeling process	1.2
c_2	Value of k_f in disuse-mode remodeling	0.1
p_0	Porosity of unresorbable bone tissues	0.5

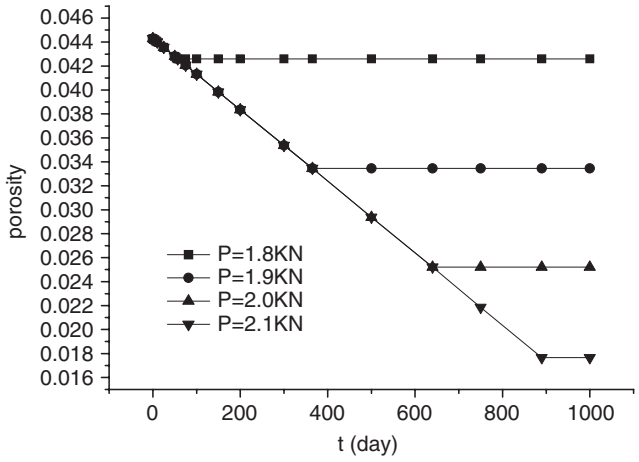

Fig. 4. Variation of porosity p for several overloads.

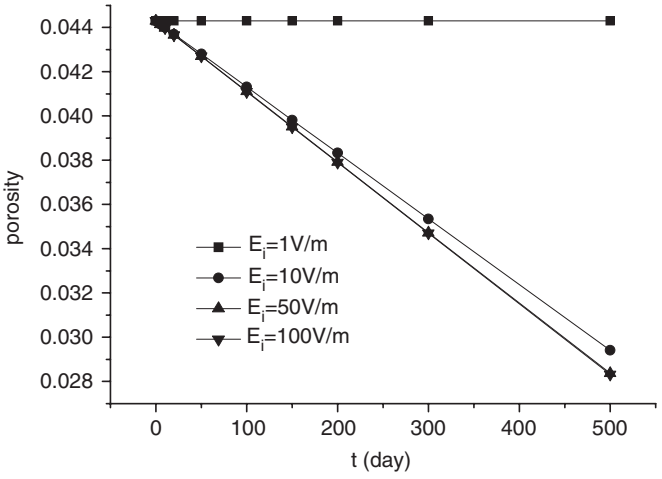

Fig. 6. Variation of porosity p for several electrical changes.

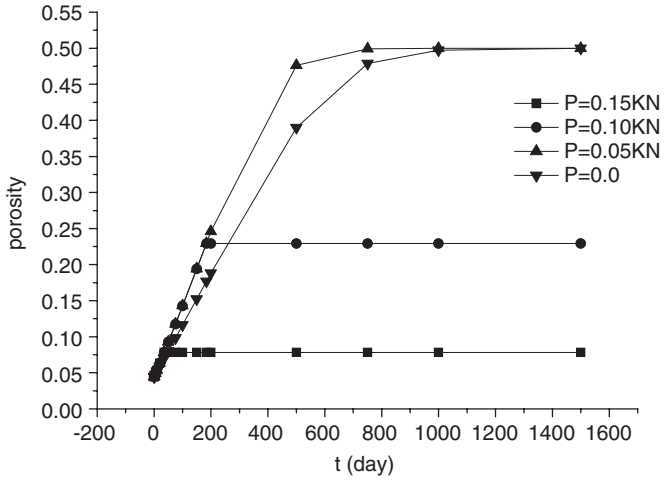

Fig. 5. Variation of porosity p in the disuse-mode case.

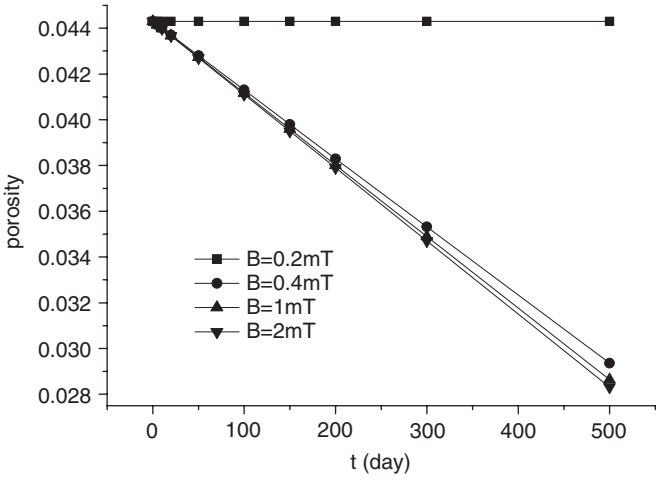

Fig. 7. Variation of porosity p for several magnetic loadings.

level. Here we define the value as 50%. When the porosity approaches this value, biological factors will stimulate the osteocytes to excrete more growth factors to resist the loss of bone mass. This is clearly shown in Fig. 5. It can also be shown that when the loading vanishes, the velocity of bone remodeling is not as fast as in bone materials subjected to compressive loads. This can be attributed to the lack of environmental stimuli, resulting in a reduction of osteoclasts. Then fewer osteocytes are resorbed and fewer growth factors are released, which slows down the loss of bone mass.

(3) $P = 1.0\,\mathrm{KN}, \quad E_i = 1, 10, 50, 100\,\mathrm{V/m}, \quad f_e = 15\,\mathrm{Hz}.$

Fig. 6 shows the effect of electrical loading on the bone modeling process. It can be seen that when the environmental stimuli are not sufficient, the remodeling state of bone tissue will remain unchanged. As the electrical loading increases to a particular level, bone modeling can be triggered. A more intense electrical field can produce a less porous and denser bone structure. But when the electrical loading is sufficiently high, a further increase will have very little effect on the bone modeling process. This is also due to an insufficiency of osteoclasts. The capacity of the body to produce osteoclasts restricts the upper limit of growth factors. So the electrical loading that can effectively stimulate bone modeling must have both an upper and a lower limit. However, all these conclusions are based on the hypothetical model. At this stage we cannot give the exact value of these thresholds; that requires further experimental investigation in this field. On the other hand, the result also indicates that bone materials become increasingly denser after electrical fields are loaded. Although the remodeling balance may be finally reached, bone tissue exposed to an electromagnetic field for a long time may suffer a high risk of bone hypertrophy.

(4) $P = 1.0\,\mathrm{KN}, \quad B_i = 0.2, 0.4, 1, 2\,\mathrm{mT}, \quad f_e = 15\,\mathrm{Hz}.$

Fig. 7 shows the effect of magnetic loading on the bone modeling process. The results are similar to those

for electrical loading. There are upper and lower thresholds in magnetic loads, and long exposure to a magnetic field can also cause bone hypertrophy.

(5) $P = 1.0\,\mathrm{KN}$, $E_i = 10\,\mathrm{V/m}$, $B_i = 2\,\mathrm{mT}$, $f_e = 2, 8, 15, 75\,\mathrm{Hz}$.

This case concerns the frequency of electromagnetic fields loaded on bone tissues. The results (see Fig. 8) are similar to the previous two cases. All three figures (Figs. 6–8) indicate that an electromagnetic field can trigger bone modeling. The effect of the loading on bone modeling is dependent on the intensity and frequency. This feature can be applied in clinical practice to treat bone diseases, such as osteoporosis and non-union, with a pulsed extremely low-frequency electromagnetic field.

(6) $P = 0.15, 0.2, 1.2\,\mathrm{KN}$, $E_i = 10\,\mathrm{V/m}$, $B_i = 2\,\mathrm{mT}$, $f_e = 15\,\mathrm{Hz}$.

As the results in case (5) show, an electromagnetic field can influence bone modeling. This effect has been used in the treatment of bone disease. As yet we do not know how bone tissue remodels itself after electromagnetic loads cease. However, post-treatment maintenance is equally important as curative effects. Here we consider three cases to study the remodeling and disuse mode, respectively. $P = 0.15\,\mathrm{KN}$ defines the disuse mode and the two later cases are remodeling examples. Electromagnetic fields are loaded, and are unloaded after 500 days.

Fig. 9 shows the simulation results. As we can see from the figure, after the electromagnetic field is loaded, the porosity of bone tissue decreases due to bone modeling. But unloading the electromagnetic field results in different effects. In disuse mode, the bone structure becomes more porous and finally returns to its initial state. In remodeling mode, when the loading is relatively small, the variation in porosity is similar to that in disuse mode. On the other hand, if the loading is large enough, the porosity will remain unchanged. The first result is due to an insufficiency of environmental stimuli. After the electromagnetic field is removed, there is no other loading to stimulate bone modeling except the initial mechanical loading. Thus bone tissue reverts to disuse-mode remodeling and bone mass loss is triggered again. Thus it can be concluded that although electromagnetic treatment is effective, active exercises are necessary to maintain the curative effect. The second result can be explained as follows. The electromagnetic load makes the bone structure more rigid. The initial mechanical loading cannot stimulate bone remodeling sufficiently after the electromagnetic field is unloaded. The bone tissue begins to remodel itself in disuse mode, which causes bone loss and increased porosity. This

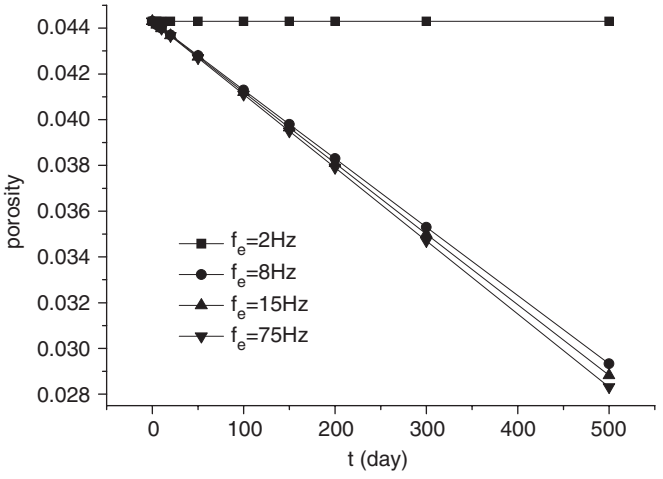

Fig. 8. Variation of porosity p for bone modeling in bone subjected to electromagnetic fields of different frequencies.

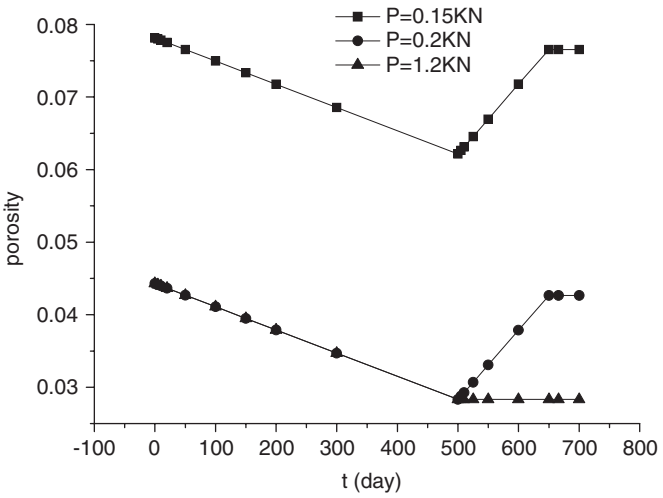

Fig. 9. Variation of porosity p for bone material subjected to several multi-field loads.

indicates that although an electromagnetic field can induce bone hypertrophy, it can be automatically cured after the field is removed. But this occurs only in some cases. In other cases, as shown in the third case ($P = 1.2\,\mathrm{KN}$), the bone mass gain is permanent.

5. Conclusion

A new hypothesis for bone modeling and remodeling is proposed in this work. A mathematical model is established based on the hypothesis. The behavior of bone modeling and remodeling under multi-field loads is simulated using the theoretical model. Simulation of overloaded bone modeling and disuse-mode bone remodeling are investigated. The effect of pulsed extremely low-frequency electromagnetic field on bone modeling and remodeling is also studied.

Numerical results show that an electromagnetic field of proper intensity and frequency can effectively trigger bone modeling. Clinical practice validates this hypothesis. However, the critical values of the intensity and frequency are still not known, and require further experimental research. Results also indicate that electromagnetic treatment may cause bone hypertrophy, although in some cases it can be healed automatically. Exercise after treatment is very important to avoid recurrence of bone loss. Further more, Figs. 4–9 show clearly how the bone structure and properties can be changed with environment. It should be mentioned that all the results obtained are based on the proposed model. Experimental validation is obviously necessary and this will be performed in the near future.

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