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Numerical Simulation of Short- and Long-Term Stability of the Hip Stem: A Review Paper on Remodeling Formulations

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ABSTRACT

Total Hip Arthroplasty (THA) is one of the only available treatments for numerous hip pathologies. Hip protheses do not last for a lifetime. As the age of first implantation increases and the patient pool increases, extending the lifespan of the prothesis is becoming of major societal and economic importance. The main cause of failure, after infection, is aseptic loosening, referring to the resorption of the bone near the implant. To tackle this issue, a precise understanding and simulation of the bone response is needed. Numerous remodeling and healing models exist, and each illustrates different biological phenomena. Creating a model accounting for both processes could help better understand the bone reaction and better predict its reaction to implantation. This article proposes to present a state-of-the art of both the remodeling and healing law, with their respective specificity. The aim is to help select models the most suited to each specific study and to start the discussion concerning models for linking healing and remodeling.

1. INTRODUCTION

Total hip arthroplasty is the total replacement of the hip joint by a prosthetic device. It is the only treatment available for numerous pathologies. This type of intervention impacts a large part of the population, as around two million people in France in 2018 [1] and it represents an important part of the global healthcare marked valued at €8billion in 2021 [2]. These numbers are expected to increase with global life

expectancy. The population with implants is expanding due to several factors. The recent technological improvements permit implantation in both younger and older patients. Lifestyle also has an influence on the number of implant recipients. Sedentary behavior and the increase of obesity create conditions conducive to the need for a hip replacement. These technologies do not last a lifetime. Revision surgeries concerned 8-9% of the patients in 2000 [3]. As the average age of the first implantation decreases, extending the lifespan of the prothesis is a major societal as well as economic goal.

Failure of the implant may be caused by several factors. One of the main causes of revision is aseptic loosening, meaning that the bone near the implant resorbs. This phenomenon represented 70% revision surgeries in Sweden in 2000 [3]. Seven to fourteen years after the operation, bone loss in the proximal femoral zone can be up to 40% [4]. Loosening is especially worrying for elderly patients, with low cell activity. This is mainly caused by stress shielding, which is the bone reaction to the modification of its stress environment [5]. Implantation of the prosthesis traumatizes the bone. The body treats the implant as a foreign body which creates major biomechanical disruptions [6]. The difference in stiffness between prosthesis titanium and bone creates a region of lack of stress transmission at the interface. The bone is thus not sufficiently loaded, leading to its resorption. This creates micro-movements between the implant and the bone jeopardizing joint stability.

The term stability in the context of this paper refers to the relative movement between the stem and the bone induced by external loads [7]. Two types are differentiated: primary or short-term stability refers to that occurring just after the surgery and during the bone healing, while long-term stability is evaluated several years after the implantation. As the biological processes determining primary and long-term stability differ, none of the models found in this study adequately describes both types of stability. A numerical model linking these two phenomena would increase our understanding of the long-term behavior of the hosting bone. Creating such a linked model is the aim of this work and subsequent numerical studies. Although the results of these studies will not be presented here, it has an impact on the models selected. The methodology will be detailed in another section.

The first section focuses on the biological phenomena. The second section focuses on the numerical simulation, describing the methodology of selection of healing and remodeling models encountered.

2. BIOLOGICAL FUNCTIONING OF THE BONE

In the context of arthroplasty, the remodeling process drives the osteointegration (the fusing of the implant with the bone) and the long-term stability, while the healing process drives the primary stability. The remodeling occurs continually and aims to renew the bone tissue and repair microdamage. The healing process occurs in case of trauma to the bone, such as a fracture or surgical operation, and it reconstructs the missing bone. These mechanisms enable the bone to repair itself and adapt its microstructure to the loading condition, and to ensure an optimized weight/mechanical properties ratio. The structure adaptation depends on the loading, possible disuse, and hormonal influence.

There are two types of bone material: cortical bone, on the outside part of the bone and trabecular bone presents in the flat bone and in the metaphyseal region of long bones.

The cortical bone is the denser part of the bone. It provides the global resistance of the bone and protects the interior part [8]. The cortical tissue is composed of the repetition of elemental units called osteons. They are highly compacted collagen sheets [8] and made of three to eight concentric lamellas, organized around a central canal. The structure is called a Haversian system [8]. Its stiffness varies between 17 and 20 GPa [9, 10]. Its porosity is between 5% and 30% [11-13].

The trabecular bone is less dense. It plays the role of an energy absorber and redirects the load onto the cortical bone [14]. It is highly anisotropic [15]. The collagen fibers are packed into lamellae of 40 to 50 μm thickness. The lamellae are grouped together to form barres and plates called the trabecula. In the literature, its stiffness is often set to 5 GPa.

The trabecular bone is more active than the cortical bone. Its remodeling rate is five to six times higher than in the cortical case [16]. Between 20-25% of the trabecular bone is renewed each year versus 1% - 4%

of the cortical bone [16, 17]. Therefore, mechanistic models tend to focus mainly on trabecular bone.

2.1. Healing

After a traumatic fracture, the bone repairs itself through different steps. First, inflammation occurs around the fracture site. This lasts one to ten days and ensures a large blood supply (see **Figure 1(a)** and **Figure 1(b)**). The blood carries important concentrations of stem cells and growth factors [18] leading to the development of a callus, which is a formation of fibrous tissues stabilizing both ends of the fracture (**Figure 1(b)**). The tissues reduce the relative movement which allows for ossification. This process lasts around four weeks. Through a chemical process called osteoinduction, the stem cells present in the callus differentiate themselves into either immature bone or cartilage cells (**Figure 1(c)**). Osteogenesis can then begin, which is the creation of bone tissue.

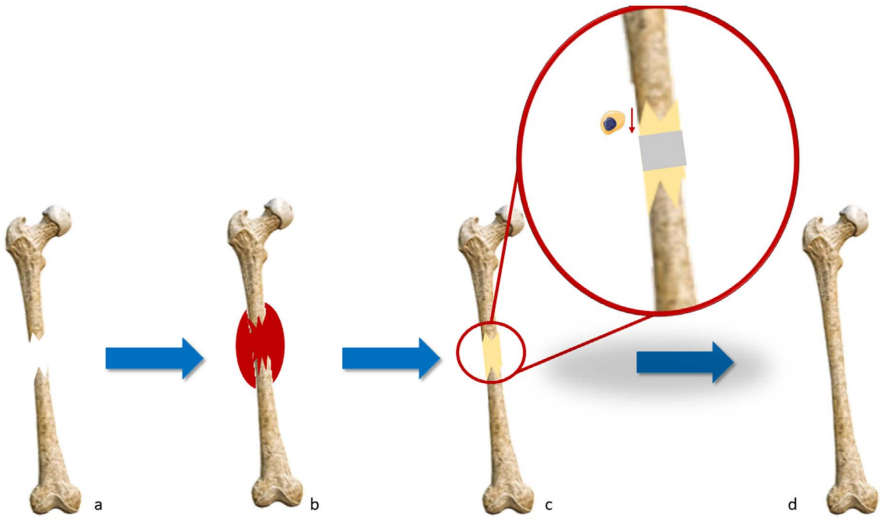

Figure 1. Healing process, a-fracture, b-formation of a hematoma around the fracture, c-ossification of the fibrous tissue, d- the mineralization of the immature bone tissue is completed and the bone returns to its previous state.

Ossification occurs through two distinct processes. Near the ends of the fracture, the fibrous tissue directly transforms into immature bone. This process is called membranous ossification. Further from the end of the bone, the connective tissue turns into cartilage and then into immature bone, which is called endochondral ossification. The different tissues spread through osteoconduction, a passive process. The cells reach the other end of the fracture by using the nearby tissue as scaffolding (**Figure 1(c)**). Generally, both processes occur conjointly, however, one of them can prevail depending on the fracture size. The immature trabecular bone forms during the ossification phase and allows a secondary ossification, stronger than the first, enabling the mineralization of the immature bone. The process can take up to a year (**Figure 1(d)**). After that, the bone starts the remodeling process like normal bone tissue [18-20].

However, following an arthroplasty, endochondral ossification is best avoided. It does not provide sufficient secondary stabilization due to remaining micromovements. Intramembranous ossification is mostly found around implants [6], mainly leading to the creation of trabecular bone at the interface between the bone and the implant [21]. The circulation of fluid within the bone is important because it increases the shear stress on the healing bone and the transportation of needed chemical components [22].

The healing of the bone will have a great impact on the remodeling process and the bone quality. Understanding the two biological process helps understand the models and their pertinence for each study.

Therefore, the next section will explore the remodeling process.

2.2. Remodeling

Bone remodeling is a biological process occurring throughout life and is presented in Figure 2. It has two primary purposes: to renew the composition of the bone to avoid fragility due to aging, and to adapt the microstructure to the mechanical environment. Bone mass decreases if the bone is disused and increases if an intense mechanical use occurs [23]. A cycle of remodeling lasts about four months and renews about 1% of the skeleton. It takes between 10 and 25 years to renew the entire skeleton [14]. The remodeling can be external or internal. The external case concerns surface remodeling. It changes the shape and geometry of the bone. Internal remodeling changes the bone property over time. In an adult, both occur simultaneously but internal remodeling is predominant [24]. After the implantation of a prosthesis, the stress is no longer bearded by the bone alone but by both the implant and the bone leading to disuse of the bone and bone resorption. Therefore, the internal remodeling leads to an increased porosity, while external leads to thinning of the bone [5].

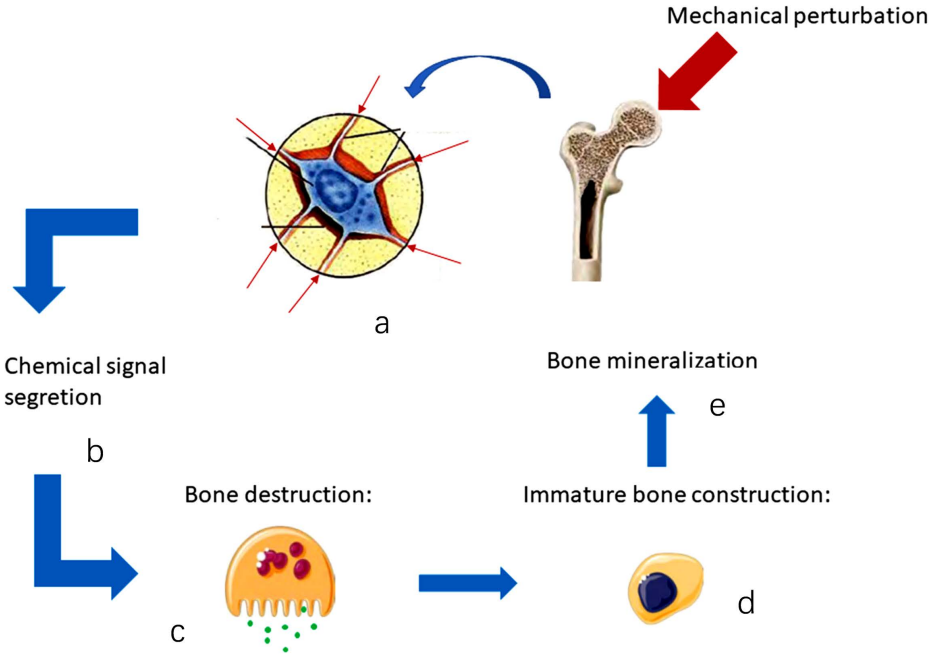

Figure 2. The remodeling process.

The remodeling cycle occurs in three steps. First, the bone is in a steady state, called quiescence in which nothing happens - neither resorption nor ingrowth. When a mechanical disturbance appears, which may be microdamage or the appearance of a prolongated cyclic loading, the remodeling cycle starts (see Figure 2(a)). Growth factor and stem cells are attracted to the remodeling zone, through chemical messages (see Figure 2(b)). Stem cells differentiate as osteoclasts (see Figure 2(c)). They are made for the destruction of the bone matrix. Their surface is striated to allow the production of enzymes. The damaged bone is resorbed in two steps: the enzymes decalcify the boney tissue. The debris particles are then ingested by the osteoclast which leads to their apoptosis. This bone decomposition takes approximately three weeks [16]. Afterwards, the concentration of growth factors and stem cells evolves to favor the differentiation of osteoblasts, responsible for the creation of an immature bone tissue, and osteoclasts (see Figure 2(d)). The volume of osteoclast decreases while that of the osteoblasts increases until the osteoblasts are more numerous. This is called inversion. It precedes the formation of new bone before the bone returns to a quiescent phase (see Figure 2(e)) [16].

For healthy bone, the ratio of formation and resorption of the bone is constant, which leads to the conservation of the bone mass [16]. Typically, after age 30, resorption is predominant over the formation of new bone, leading to loss of bone mass [16].

After the cycle, some osteoblasts cells remain trapped inside the bone matrix and become osteocyte cells. They are linked by the canaliculus. They act as mechanoreceptors which are sensitive to bone quality. They perceive microdamage and trigger the remodeling cycle [25]. There are several hypotheses on how those cells sense the mechanical environment [26, 27]. The most widespread hypothesis in the literature is the one reported by Cowin *et al.* (1991) [28]. It supposes that osteocytes are responsive to fluid flux. When the flux changes, the deformation of the cells evolves. This is interpreted by the cells as a chemical signal which attracts growth factors and stem cells on the remodeling site.

Having drawn the principal mechanism responsible for the healing and the remodeling of the bone after the implantation helps better comprehend the principles factors to simulate. It was then possible to decide inclusion and exclusion criteria, explained in the following section.

3. NUMERICAL MODELS

3.1. Methodology

As stated before, the aim of this bibliographic work was to create a complete model of the bone reaction to the implantation, linking the healing and remodeling laws. This dictated several different criteria. First, there was an exclusion criterion: the mathematical and computational frame work had to remain simple enough to function inside an Abaqus subroutine UMAT. Moreover, our goal is to create a model as precise as possible, therefore, the representation of the bone material was a criterion too. Bone is mostly represented as a homogeneous material which is an approximation leading to a roughly 30% under-estimation of the remodeling process [29]. A more precise representation of the bone is a poro-elastic representation that we will try and integrate in the complete model. It is one of the criteria for the model's selection. Three cases were considered: if the model was already used on a poroelastic representation in the literature the criterion took a "yes" value. If the model was not already adapted to poroelasticity but it seems possible to do so without significant modification, the criteria were marked "possible". Finally, if it does not seem possible to easily adapt the model to poroelasticity, it was marked "no". The last criterion dictated by our study concern the constants and parameters of the model. They could not be experimentally determinate and have to be accessible through the literature.

Two main descriptions of healing and remodeling mechanisms exist in the literature. The first primarily considers the chemical response of the bone to stimuli. These descriptions are called phenomenological models. The healing and remodeling processes will be described through the diffusion and the volume ratio of cells such as the growth factors, and stem cells. Several hormones and proteins act as messengers to attract and help differentiate stem cells on the location. Such models tend to be very complex because of the intricacy of the biological functioning and of the chemical equations. Because of this, they often present computational complexity and requires important resources in terms of memory and computational time. Therefore, they rarely consider abnormal external mechanical perturbations - they mainly simulate "normal" remodeling. The changes in the mechanical environment tend to be little to no impacts in such models. In the context of arthroplasty, the mechanical environment changes are the main factors of remodeling. The phenological models were excluded from this bibliography.

Machine-learning based model were not included in this study as they would lead to a whole different study. Few patients' specific models were found. Those found were usually on the very begging of their development and needed experimental tissue characterization to function. As we were trying to create a surgeon-friendly model, the models requiring experimental characterization were excluded.

Models mainly focused on mechanical aspects are called mechanistic models. They model how the bone will react to a change in its mechanical environment but simplify or ignore the chemical part of the biological response. Mechanistic models are better suited to respond to both healing and remodeling and therefore,

were the only ones included in this study. The mechanical and chemical criteria are treated as simply "yes or no", based on whether the criteria impacted the bone evolution in **Appendix 1**. The Ambard model is described as having a weak mechanical aspect, since the mechanical environment only impacts the evolution of the friction coefficient between the bone and the prothesis [6].

Another studied criterion was the model scale. Three scales are differentiated. The microscopic scale is centered around the cells and their evolution without considering the global behavior of the bone. On the other hand, the macroscopic scale only considers the global evolution of the bone without modeling the response at the cell level. The mesoscopic scale is a compromise between the two: the cell response is considered, but the main focus remains on the global evolution of the bone. As our concern is the global change in the bone distribution and mechanical properties changes, while including the cellular reaction to the implant, the mesoscopic scale seemed the most appropriate.

3.2. First Stabilization: Healing of the Bone

The models studied and the criteria are summarized in **Appendix 1**. All the models presented are mecha-nistic models. Some models focus more on the diffusion equations than the others. For example, Ambard & Swider (2002), Askari Rizvi *et al.* (2022), Bailón-Plaza & Van Der Meulen (2001), Geris *et al.* (2010) and Gómez-Benito *et al.* (2006) describe more or less accurately the diffusion equations of different cells, such as stem cells or growth factors, and the evolution of their concentration [6, 19, 30-32]. The mechanical factor has little impact on those equations. They offer little detail on how the mechanical environment of the implant influences healing. Checa & Prendergast (2009) describe a precise description of cell migration and vascularization [33]. However, it is very complex from the mathematical point of view and will not be adaptable to a poroelastic representation of the bone. The model of Moreo *et al.* (2009) describes the cell concentration and bone ingrowth as a propagating wavelength [34]. This model is useful to model a traumatic facture healing but is not suited to arthroplasty healing. In these conditions, two models are examined more fully and will be detailed in the following sections.

3.2.1. The Model of Shefelbine *et al.*, 2005 [35]

This model was created to simulate fracture healing. Each element is represented by a blend of different tissue types with specific mechanical properties: soft tissue, bone and cartilage. The final properties of the element are calculated through a mixture rule. The elastic (Young's) modulus E is a cubic function of the volume fraction of the tissue (denoted c), and likewise Poisson's ratio ν is an average law.

$$E_{el} = E_{cart}c_{cart}^3 + E_{tiss}c_{tiss}^3 + E_{os}c_{os}^3 \quad (1)$$

$$\nu_{el} = \nu_{cart}c_{cart} + \nu_{tiss}c_{tiss} + \nu_{os}c_{os} \quad (2)$$

The volume fraction of each tissue component in an element is calculated through a fuzzy logic controller. The rules are formulated as 21 if/then statements. The term fuzzy logic refers to the possibility of activation of several rules for the same elements. They give the percentage change of bone, cartilage and vascularity for each element, following the diagram **Figure 3**. It is a diagram with the stress on the bone on the horizontal axe and the octahedral shear strain on the vertical axe. The values taken by the stress and the strain determine with tissue will grow, represented by the square.

Using the strain as a stimulus in this model has proven to improve the results, as it does not exhibit discontinuous behavior. No comparison between the model and clinical results were found. However, this model shows good results compared to other models of the literature. It especially offers a precise understanding of tissue differentiation.

This model has the advantages of considering a large number of parameters to agree with the experimental results, due to fuzzy logic. It describes very precisely the vascularization process which is essential for bone colonization. However, this model also presents counterparts. First of all, this model is not adapted to implantation and its computational complexity make it hard to use outside its original perimeter. It seems complicated to run with a complex Abaqus modeling and to input through a subroutine.

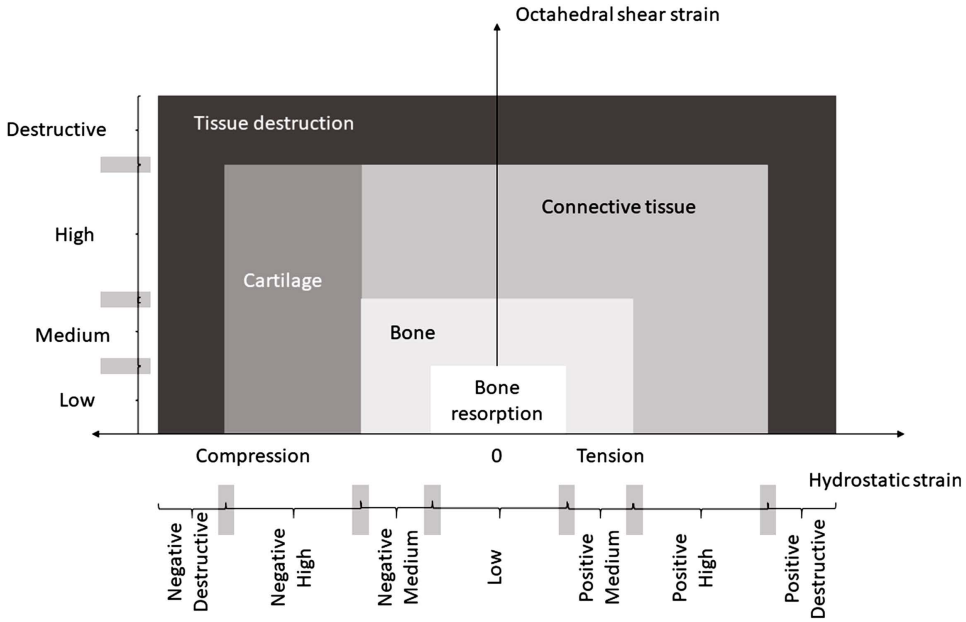

Figure 3. Fuzzy logic diagram from Shefelbine *et al.* (2005). Shaded areas are the limit of a domain. This instead of threshold makes it fuzzy.

3.2.2. The Model of Chou & Müftü, 2013 [36]

This model simulates healing after a dental implant. The gap between the implant and the bone is considered filled with callus. The mechanical properties of this interstitial tissue are updated depending on the mechanical stimulus and the cell migration, following a mixture rule:

$$E_{nvx} = \frac{n_{cells}}{n_{max}} E_{tissue} + \frac{n_{max} - n_{cells}}{n_{max}} E_{callus} \tag{3}$$

Depending on the mechanical stimulus, different tissue will grow at each iteration. The symbol S is the same mechanical stimulus as introduced by Checa & Prendergast (2009) [33]. If S is high, fibrous tissue develops. If S is low, immature bone tissue occurs preferentially. Between these extremes, cartilage tissue grows. The tissue type determines the modulus E_{tissue} and simulates the differentiation of the cells.

$$S = \frac{\gamma}{a} + \frac{\nu}{b} \tag{4}$$

Next, n_{cells} is calculated through an equation, representing the cell diffusion during healing.

$$D\nabla^2 n_{cells} = \frac{\mathrm{d}n_{cells}}{\mathrm{d}t} \tag{5}$$

A smoothing equation ensures the convergence of the model.

$$E^{i+1} = \frac{1}{N}\left(E_i + \cdots + E_{i-(N-1)}\right) \tag{6}$$

This model seems to have good correspondence with clinical cases. It was successfully used to predict healing around dental implant.

This model presents several advantages. It focuses on mechanical perturbations and considers the fluid flow influence. It considers the cells diffusion and was made for implantation cases. However, in comparison with other studied models, it is relatively simple on a mathematical and computational point of view. The scale is also quite large. Therefore, the model is easily adaptable to specific studies but will not be suited for

the thorough study of one parameter of the healing process. The model is summarized in **Figure 4**.

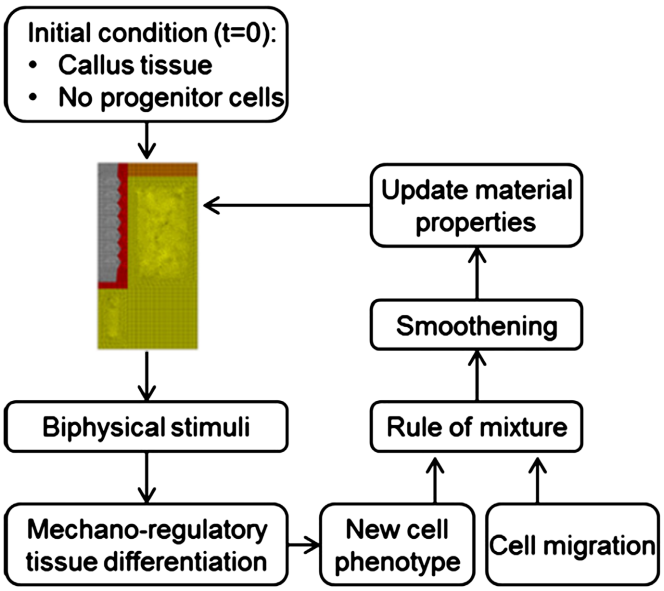

Figure 4. Flowchart of the model.

3.3. Second Stabilization: Remodeling

As for the healing models, several criteria were evaluated. The criteria and the models encountered are summarized in **Appendix 2**. The remodeling mathematical descriptions studied were isotropic and adapted to a non-cemented stem. The models of Weinans and Beaupré were the most frequently encountered in prior studies. They were used in 18 of the publications cited. The second most widely encountered model was that of Fernandes. These two models are presented in detail in the following sections. Because of their place in the literature, the models of Weinans-Beaupré and Fernandes will be considered as the points of departure. The unique features of the other models were evaluated for comparison. Some of the models studied propose a better description of the remodeling process [11, 37-39]. Most of these models are not adapted to arthroplasty and it is difficult to do so. They offer detailed representations of the influence of certain factors on remodeling, like the impact of the hormone rates [41] or the representation of a specific part of the remodeling process, like surface remodeling in [40]. However, little information is provided on the influence of the stem material over the osteointegration process. In the same way, a recent paper [42] creates an algorithm to construct a bone model faithful to the biologic process, but subsequently uses the Weinan remodeling law. The numerical process is time consuming and does not allow restarting from the healing model results. Tarala *et al.* (2013) create a model exploring the impact of micromovement over the osteointegration process [43]. It gives few details on the impact of the material properties of the implant but more on the size of the gap left between the implant and the bone. Finally, Hazelwood *et al.* (2018) present a model updating the pore rate in the bone [44]. This model is interesting but complex from a mathematical point of view. This makes it difficult to adapt to the patient and to coordinate with other models in the context of optimizing the time of calculation and studying the impact of a material on a specific patient.

3.3.1. The Model of Fernandes *et al.* (1999)

The bone is represented as a cellular material. The microstructure is a periodic repetition of cubic unit cells with rectangular holes. The matrix is considered as an isotropic, homogeneous, elastic linear material. The unit cells are characterized by three parameters: the dimension of the hole a, an angle θ, referring to the orientation of the element, and a control bone density μ [45].

The bone remodeling is described through an update of the mechanical properties. The updates happen if they overweigh the cost of bone maintenance. Mathematically, this is described by the following equations [45-47]:

$$\sum_{P=1}^{NC}\left[\alpha^{P}\frac{\partial E_{ijkl}^{H}}{\partial a}\varepsilon_{ij}\varepsilon_{kl}\right]-k\frac{\partial \mu^{m}}{\partial a}=0 \tag{7}$$

$$\sum_{P=1}^{NC}\left[\alpha^{P}\frac{\partial E_{ijkl}^{H}}{\partial \theta}\varepsilon_{ij}\varepsilon_{kl}\right]=0 \tag{8}$$

with:

μ: bone density;

θ: angle of bone growth;

NC: number of applied loads;

α: weight function.

The preceding equations are based on the homogenization model of Guedes and Kikuchi. The parameters a and θ are updated by the equations. The symbols k and m define the bone maintenance cost. Iterations are repeated until the conditions of both equations are met, representing the bone equilibrium.

The weight function α^{p} satisfies the condition $\sum_{p=1}^{NC}\alpha^{p}=1$. The homogenization has the same hypotheses than the poroelastic model: the problem has two distinct scales. The macro scale is the bone whereas the micro scale is the trabecula. The microscale presents repetitive patterns unit cells with holes which shape varies depending on the representation chosen. This micro scale representation enables the homogenization, based on Guedes & Kikuchi [45, 47].

The basic model can be modified through several factors. For example, the model of Folgado *et al.* (2004) is based on orthotropic conditions [48]. They implement a prismatic hole and compare it to a cubic hole. The equations do not differ much from the first set. The results are strongly dependent on the cells used. The model is relatively simple from a mathematical point of view. It offers a macroscopic view of the remodeling process, centered on the mechanical aspect. The chemical process is not studied in this model. It has been designed for poroelastic representation of the bone.

At the time, this model was a real break-through as it is one of the first model to include poro-elastic behavior. It has proven to have good results with rather simple mathematical framework. However, the rigidity of the model makes it difficult to adapt, as the results strongly depends on the pores' shape. More recent models, centered around the poro-elastic behavior, may offer more precise results, they will however be more complex on a mathematical point of view.

3.3.2. Models of Weinans and Beaupré

These models are the most widely used in the literature. They are nearly identical and are based on the same concept and hypothesis. It is assumed that within the bone, cells act like mechanical sensors (i.e., the osteocytes). They capture a mechanical stimulus, whose amplitude will modulate the bone remodeling process [49]. The remodeling is modelled through the update of density and elastic modulus at each calculation point. Their evolution is based on the Wolf law: if the bone is sufficiently stimulated, it will grow. Thus, the density (ρ) and Young's modulus increase. If the bone is not stimulated, it resorbs; leading to a decrease of density and Young's modulus. There exists a range of loading where bone neither grows nor resorbs. This zone is called the dead zone and must be included to ensure the convergence of the model [50] (see **Figure 5**).

The models originally were used to describe isotropic, homogeneous, elastic linear bone [49]. They are based on three different equations: the mechanical stimulus equation, the equation modeling the evolution of the properties (either the density or Young's modulus) and the relation between the density and the modulus.

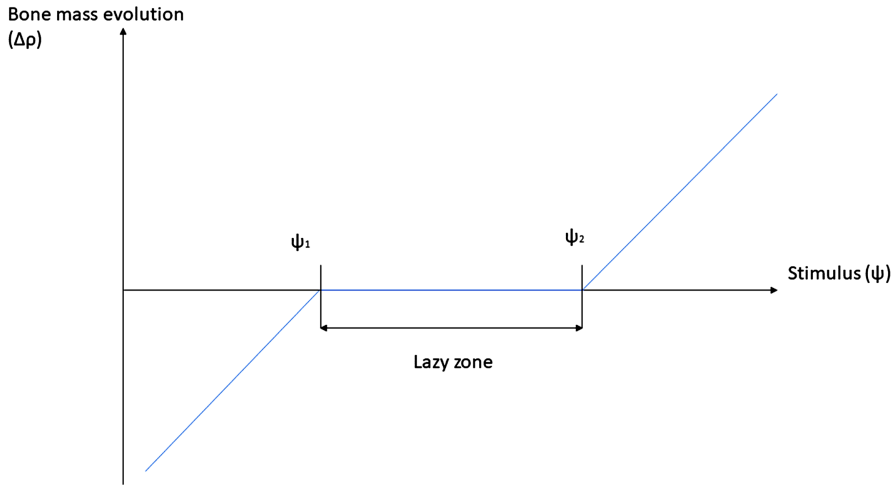

Figure 5. Schematic representation of the Wolf's lazy zone.

The mechanical stimulus (ψ) choice is either Beaupré or Weinan. Weinans is based on the strain energy density U [51] whereas Beaupré is based on the overall stress in the bone [52]. N is the number of loading case.

$$\text{Beaupré:}\quad \psi = \left(\frac{\rho_c}{\rho}\right)^2 \left[\sum_{j=1}^{N} n(j)\sigma_k^m(i)\right]^{1/m} \tag{9}$$

$$\text{Weinans:}\quad \psi = \frac{1}{N}\sum_{j=1}^{N}\frac{U_j}{\rho} \tag{10}$$

Depending on the stimulus amplitude, the mechanical properties, and the density, are updated. Mainly three type of laws were encountered in the literature: linear laws, quadratic laws and plateau laws [53].

Finally, an equation in the form of $E = C\rho^{\gamma}$ links the evolution of the density to the elastic modulus, and updates the latest values [25]. The most common values for C and ϒ in the literature yield $E = 3790\rho^3$ [54].

Piccinini *et al.* (2016) compare the different stimulus and evolution laws most encountered in the literature [53]. They concluded by recommending the use of Weinans' mechanical stimulus with a linear or plateau evolution law. The Weinans stimulus will enable one to consider the effect of the fluid flow through the pores, including the deformation imposed on the solid matrix. It more accurately describes the biological process as the osteocytes capture their deformation, due to the change of the fluid flow. Concerning the evolution law, the plateau law is interesting, as it allows consideration of necrosis of the bone in the case of overloads. This could be a helpful parameter in the geometrical and material design of the stem.

These models provide a simplified, macroscopic view of the remodeling process. Centred on the mechanical side of the remodeling process, they can be adapted to a poroelastic model with an isotropic linear elastic solid matrix. As the mathematical framework is rather simple. Those models have good results compared to clinical case and are still widely use. However, they present limitation such as having positive feedback and discontinued behavior [35].

Ruimerman *et al.* (2001) propose an enhancement of this model [23]. The resorption is assumed to be happening at a random location. This version is more biologically accurate in the case of daily remodeling. However, in the case of an arthroscopy, it fails to account for the bone resorption due to disuse.

4. CONCLUSIONS

Currently, total hip arthroplasty is the only treatment available for numerous age-related hip pathologies.

However, the implant does not last for a lifetime, leading to revision surgeries. One of the main causes of the reintervention is aseptic loosening, meaning the resorption of bone near the implant. To tackle this issue, understanding and precise modeling of the bone remodeling after the implantation is essential. With this in mind, this paper draws on extensive bibliographic research on existing remodeling formulations.

As short- and long-term stability of the implant relies on two different biological processes, this article presents healing models, for short term stability and remodeling models, for long term stability. Because of the context of the study, the bibliography concentrates on mechanistic models, usable inside an Abaqus subroutine UMAT and in the context of arthroplasty. Several models are presented and compared inside **Appendix 1** and **Appendix 2**. Each has specificities and concentrates on different factors and/or bone material representations. The selection of the models most suited for each specific study is an essential step in having pertinent results. In the context of our study, we try to create a finite element Abaqus model with the constraint of working with poroelastic representation of the bone and UMAT subroutine. The model of Chou for the healing and the Weinans model for remodeling seem the most appropriate. They have the same framework, facilitating the linking. They both concentrate on mechanical factors and present a mesoscopic representation of the phenomena. The linking is made through a predefined field. The aim is to start the remodeling calculus at the last step of the healing calculus, to integrate stress, strain and mechanical changes history in bones. However, the Chou's model has limitations, as it does not account for vascularization. Concerning the Weinans model, more recent models include information on specific factors, such as pore evolution or hormonal factors. At this stage of the study, improvement does not seem essential. If the model evolves toward more subtle representation of the remodeling, the choice of the model might be reconsidered.

No paper currently published concentrates on linking short- and long-term stability modeling. By presenting both types of models, this article aims to facilitate this discussion and help create more complete bone reaction models.

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CONFLICTS OF INTEREST

The authors declare no conflicts of interest regarding the publication of this paper.

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Appendix 1: Summary of the Healing Model Encountered.

Autors	Bailon-Plaza et al.	Van Der Meulen et al.	Gomez-Benito et al.	Shefelbine et al.	Ambard et al.	Checa et al.	Moreo et al.	Geris et al.	Chou et al.	Askari Rizvi et al.
Reference	[19]	[37]	[32]	[35]	[6]	[33]	[34]	[31]	[36]	[30]
Date	2001	2002	2005	2005	2006	2009	2009	2010	2013	2022
Mechacinal factor	No	Yes	Yes	Yes	Weak	Yes	Yes	No	Yes	Yes
Chemical factor	Yes	No	Yes	No	Yes	No	No	Yes	No	No
Poroelasticity	No	Yes	No	Yes	Yes	Yes	No	No	Yes	No
Scale	Micro	Meso	Meso	Meso	Micro	Meso	Micro	Meso	Meso	Meso
Parameter accessibility	No	Yes	Yes	Yes	No	Yes	No	No	Yes	Yes
Originality	Impact of growth factor on healing	Mechano-regulaiton	Cells behaviour description	Fuzzy logic	Fluid/bone interaction	Cellular migration	Wave	Connect biology and mechanics	Fluid flux	Fluid flux
Advantages	Precise description of the chemical process	Introduce the concept of osteogenic index Simple mathematical and computational france	Impact of gap size on healing Precise description of cellular behaviour	Vascularisation calculation Advance representation of the healing process Large number of parameters accounted for	Precise representation of cell concentration And fluid/bone interaction Adapted to implantation	Representation of cells migration and differenciation Adapted to implantation	Representation of the bone ingrowth as wave length Precise representation of cellular diffusion	Connecting chemical and mechanical factors	Considers cells diffusion Mechanical behavior Simple mathematical frame Adapted to implantation	Precise representation of the callus formation Precise representation of the cell diffusion
Incovenients	Not adapted to implantation No vascularisation No impact of the mechanical environment	Not adapted to implantation No vascularisation	Not adapted to implantation No vascularisation Complex mathematical frame.	Complicated computational frame Not adapted to implantation Little mechanical impact	Complicated mathematical frame No vascularisation	Hard to adapt to finite element modeling	Complex mathematical frame Seems hard to adapt to finite element analysis Not adapted to implantation No vascularisation	Not adapted to implantation No vascularisation Demand Matlab Complicated mathematical and computational frame	No vascularisation	Complex mathematical and computational frame Not adapted to implantation

Appendix 2: Summary of the Remodeling Model Encountered.

Models	Beaupré	Weinan	Fernandes	Hazelwood et al.	Tsubota et al.	Machalanne et al.	Tarala et al.	Yi et al.	Barkaoui et al.	Du et al.	Dicati et al.	Mathai et al.
Reference	[4] [50] [52] [54]	[4] [25-27] [51] [55] [56]	[46] [47-50]	[44]	[40]	[57]	[43]	[41]	[43]	[39]	[11]	[42]
Date	1987	1992	1999	2001	2005	2011	2012	2015	2016	2021	2022	2022
Mechacinal factor	Yes	Yes	Yes	No	Yes	Yes	Low	No	Yes	Yes	Yes	Yes
Chemical factor	No	No	No	Yes	No	No	No	Yes	No	No	No	No
Poroelasticity	Yes	Yes	Yes	Uncertain	Possible	Yes	Possible	Uncertain	Possible	Possible	Possible	Yes
Scale	Meso	Meso	Meso	Micro	Micro	Meso	Macro	Micro	Micro	Meso	Meso	Meso
Parameter accessibility	Yes	Yes	No	No	No	No	Yes	No	No	Yes	Yes	Yes
Originality	Reference	Reference	Consider fluid flux and pores size	Adaptation of the pore size	Surface activity	Impact of frequency factors on loading	Evolution of the friction coefficient	Hormonal factor	Hormonal factor	Microdamaging + cyclic loading	Surface remodeling	Orthotropy of the bone
Advantages	Simple mathematical frame Often used	Simple mathematical frame Often used	Simple mathematical frame Often used	Insight on the pores evolution	Precise remodeling model of trabeculae	Take into account the relaxation time of the bone and the marrow	Adapted to implantation Maturation of the bone	Insight on specific factor	Insight on specific factor	Adapted to implantation Good representation of the bone	Adapted to implantation Based on Beaupré stimulus	Adapted to implantation Precise representation of trabecular growrth
Incovenients	Stimuli used farer from biological functionning		Sensitive to cell shape	Not adapted to implantation Scale too small No global evolution Complex mathematical frame	Not adapted to the implantation Very small scale	Not adapted to implantation Complex mathematical frame	Few structural impact of mechanical environment	Not adapted to implantation Few insight on the general process Complex mathematical frame	Not adapted to implantation Few insight on the general process Complex mathematical frame	Demand access to precise tomography of the bone Mechanical model not as precise as other	Complex computational and mathematical frame	Complex bone representation Complex mathematical frame Demand access to precise tomography