



Damaged elastic bone-column buckling theory within the context of adaptive elasticity

Salah Ramtani

Laboratoire CSPBAT – LBPS, UMR 7244 CNRS, Université Paris-Nord, Institut Galilée, 99, avenue J.B. Clément, Villetaneuse 93430, France

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ABSTRACT

Bone column-buckling is known as due to the loss of trabecular connectivity such that vertical weight-bearing trabeculae lose their cross attachments with each other. It has been considered as a predominant mechanism accounting for skeletal fragility disproportional to the degree of bone loss. Here, we focus our attention upon the improvement of our understanding of the relationships between bone degradation and fracture risk assessment (i.e., buckling load) which is both timely and at the core of important clinical problems such as osteoporosis and age-related bone fragility. With respect to well-known existing literature, there is a great need for a theoretical framework that provides new insights into the bending-buckling process when coupled to continuum damaged-bone adaptation. Within the limitations of our assumptions, it is clearly shown that damage occurs preferentially in regions where bone resorption activity dominates and the existence of a bone density gradient helps to maintain the stability of the bone-column deflection. For another, predicted buckling stress as a function of slenderness ratio is satisfactorily appreciated through experimental results. In our knowledge, this contribution is an original first attempt, at the scale of the single trabecula, which used adaptive elasticity in order to state an original but simplified adaptive-damage bone column buckling theory.

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1. Introduction

Bone-column buckling, at the trabecula scale, remains a subject of interest especially when applied to spongy bone structure and perhaps, the only area of structural mechanics in which failure is not directly related to the material strength. Trabecular or spongy porous type bone is known as playing crucial role in load transmission in major joints such as the hip, knee and spine [1]; and exhibits different bone architecture, with both interconnected trabeculae shaped plates and rods-like microstructures [2–12] (Fig. 1). Osteoporosis is known as a metabolic bone disorder, which causes bone loss and result directly in an increased risk of bone failure with respect to decreased bone strength [13–15]. Previously published by Parker [3], a three-dimensional synchrotron computed-tomography images of a 4 mm -trabecular bone cube extracted from the vertebra interior are depicted Fig. 1. The image case of a 30-year-old female bone (Fig. 1(a)) is compared with a similar image of 63 year old male (Fig. 1(b)). One can note that the first case which is concerned with a normal bone density and architecture; shows a significant number of trabeculae and plate-like structures aligned in the load-bearing direction (Fig. 1(a)).

The second case, which is considered as osteoporotic, exhibits notably different bone microarchitecture, with fewer trabeculae plate-like structures and more of the trabeculae shaped like slender rods (Fig. 1(b)). Inspired by previous works conducted by Kinney and coworkers [7–11], Parker [3] has developed an interesting analysis giving research highlights at how aging bones fracture. In particular, interesting mechanisms have been identified [3]: (a) “bone loss makes the trabeculae longer and more slender”, (b) “bone turnover introduces physical defects, in terms of resorption pits”, (c) “in younger trabecular bone, the stress overcomes the strength of the bone tissue and thus strength-initiated failure dominates”, (d) “in older bone, stability-initiated failure dominates because of the instability of the individual trabeculae known as prone to inelastic buckling at stresses far less than expected for strength-based failure”

With respect to eighteen wet and dry trabeculae obtained from three medial tibia end sections (knee joint), Townsend et al. [4] carried out controlled buckling tests of single trabeculae. As a main result, the plot of buckling stress as a function of slenderness ratio, shown in Fig. 2, suggests that the dried samples behave as a brittle material and the wet trabeculae as a very ductile material. The extrapolated curves from Fig. 2 estimate the elastic modulus, predicted by the Euler equation, to be 14.13 GPa and 11.38 GPa for dry and wet trabeculae respectively. As a main con-

E-mail address: ramtani@univ-paris13.fr

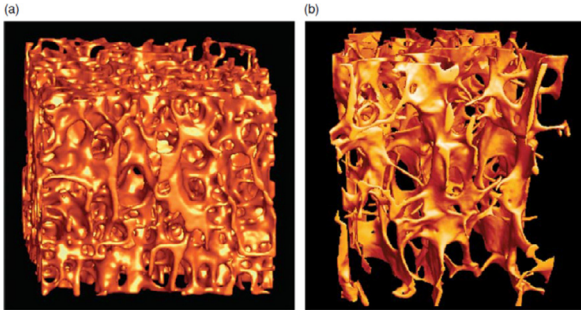


Fig. 1. Three-dimensional synchrotron computed-tomography image from [3].

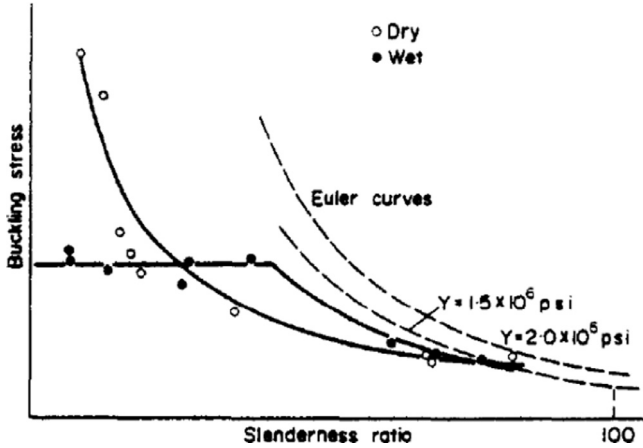


Fig. 2. Buckling stress versus slenderness ratio for wet and dry trabeculae [4].

clusion, Townsend et al. [4] stated that the buckling in vivo of individual trabeculae must necessarily be inelastic.

When measured at the whole specimen, micro damage was often associated with resorption cavities implicated in physiological phenomena including mechanical failure processes and adaptation of cancellous bone [16–18]. Thus, understanding its accumulation is of great importance in predicting, and providing early treatments for skeletal fragility. What is well established, when tiny micro cracks were observed in bone using microscopy techniques, is that they are dispersed throughout the strained bone volume, in close proximity but isolated from each other and apparently impervious to each other's presence [16–18]. When this micro-damage exhibits both an inefficient and prolonged micro damage repair, due to periods of pause in the formation phase of remodeling; skeletal fragility can be notably affected [16–18].

In our knowledge, a theoretical framework dealing with plate or beam column bending–buckling process coupled to bone adaptation has been previously investigated by [19,20] but current contribution is distinguished from the previous ones by the introduction of a continuum damage parameter d that accounts for many skeletal fragility mechanisms. The choice of beam column-like instead of plate-like theory was first motivated by the suggested and well admitted observation that a conversion or deterioration from plate-like to beam column-like trabecular bone, with fewer trabeculae plate-like structures and more of the trabeculae shaped like slender rods, constitutes an important etiologic factor in osteoporosis- and age-related bone fragility [3, 21,22]. As a first attempt, a simplified 1D adaptive damaged-bone beam buckling theory is first considered here with respect to Timoshenko hypothesis and then, as a result, we have numerically investigated previous case of adaptive Euler-Bernoulli beam model due to Ramtani et al. [20] by adding local continuum damage effect.

Among other results, predicted buckling stress as a function of slenderness ratio is satisfactorily appreciated through experimental results performed by Townsend et al. [4]. We indicate and confirm that the activation of local bone loss process favors the emergence of micro damage, and can leads to the apparition of new elastic instabilities which are likely to lead to local damage bone-buckling mechanism of fracture localized at the scale of a single trabecula. We also demonstrate that bone density gradient play a key role in both maintaining the mechanical stability of the single trabecula and micro damage repartition.

2. Theoretical formulation

The general theory of adaptive elasticity, originally formulated by Cowin and Hegedus [23,24] as a model for the physiological process of bone adaptation, is taken back as a framework for our theoretical developments. The modified Hooke's law which was dependent only upon the bone volume fraction of the material present e is extended here to damage variable d as follows

$$T_{ij}(e, d) = (1 - d)\xi_0 T_{ij}^{(0)} + (1 - d)^2 e T_{ij}^{(1)},$$

$$T_{ij}^{(n)} = C_{ijkl}^{(n)} \varepsilon_{kl}, n = 0, 1 \quad (1)$$

where $T_{ij}(e, d)$ and ε_{kl} represent respectively the Cauchy stress and linearized strain components. $C_{ijkl}^{(0)}$, $C_{ijkl}^{(1)}$ are constants tensors representing the elastic properties of the adaptive material, $e = \xi - \xi_0$ is the measure of the change in solid phase volume fraction from a reference volume fraction ξ_0 , ξ is the solid phase volume fraction of the reference unstrained state and γ is the density of the material that composes the matrix structure assumed to be always constant. The scalar parameter d represents the damage variable which is considered here classically as a reduction in elastic modulus. However, it can be defined in a various number of ways depending on the analysis involved. One can see that the elasticity tensor $C_{ijkl}(e, d)$ is not constant and evolves linearly and nonlinearly with respect to both bone density and damage respectively.

What is well admitted is that the bone tissue response exhibits changes both at microstructural (i.e., internal remodelling) and shape (i.e., external remodelling) level [25]. However, according to the biomechanical environment to which the tissue is subjected, one predominates over the other. It has been observed, for example, that the external remodelling becomes essential for specific disorders such as cortical hypertrophy at the diaphysary level, because the geometric alteration prevails over the microstructure degradation [25]. It is assumed here that internal bone remodelling probably predominates when osteoporosis is at an advanced stage, and the conversion from plate-like to trabeculae shaped like slender rods is completed. Thus, we adopt the equation that only governs the elastic damage internal bone adaptation, previously stated with respect to bone's mass balance considerations [26], and which exhibits an original relation between the rate of change of the bone volume fraction, strain, damage and damage rate

$$\dot{e} = \frac{1}{(1 - d)} [A(e) + A_{ij}(e)\varepsilon_{ij} + (\xi_0 + e)d], \quad (2)$$

where $A(e)$ and $A_{ij}(e)$ are material coefficients depending only upon the bone volume fraction e .

2.1. Timoshenko beam's hypothesis

According to the classical one dimensional *Timoshenko* beam theory, the incremental displacements (3) are,

$$u_x = z\psi(x), \quad u_z = w(x) \quad (3)$$

where $\psi(x)$ and $w(x)$ are respectively the bending rotation and the transverse deflection.

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