Disponible en ligne sur
ScienceDirect
www.sciencedirect.com
Elsevier Masson France
EM|consulte
www.em-consulte.com
IRBM 36 (2015) 267–277
IRBM

Original article

Elastic bone-column buckling including bone density gradient effect within the context of adaptive elasticity

S. Ramtani [a,*], H. Bennaceur [b], T. Outtas [b]

[a] *Laboratoire CSPBAT – Biomatériaux pour la Santé, UMR 7244, CNRS, Université Paris 13, Institut Galilée, 99, avenue J.B. Clément, 93430 Villetaneuse, France*
[b] *Laboratoire Mécanique des Structures et Matériaux, Université de Batna, Département de Mécanique, Faculté des Sciences de l'Ingénieur, Avenue Chahid Boukhlouf, 05000, Batna, Algeria*

Received 3 March 2015; received in revised form 1 July 2015; accepted 15 July 2015

Available online 31 July 2015

Abstract

Objectives: Our main goal is to improve, from theoretical point of view, the mechanistic understanding of bone buckling failure which is known as at the core of important clinical problems such as osteoporosis.
Material and methods: What is well argued is that in older bone, stability-initiated failure dominates because of the instability of the individual trabeculae which is prone to inelastic buckling at stresses far less than expected for strength-based failure. Taking advantage of our previous work, an improved original Euler's adaptive-beam buckling equation incorporating density gradient effect is investigated.
Results: For one, we indicate that resorption can leads to new elastic instabilities that can conduct to bone-buckling mechanism of fracture. For another, we demonstrate that bone density gradient play a key role in the initiation of the bone-column elastic buckling instability.
Conclusion: As a result, it is clearly stated here that firstly, the number of these elastic instabilities which are potentially implied in the mechanisms of bone fracture, localized at the trabecular element scale, depends strongly upon the material parameter η and secondly; the bone density gradient affect notably the stability of the bone-column deflection.

1. Introduction

Throughout life, bone is continuously turning over by the well-regulated processes of bone remodeling which involves changes in material properties where the bone may change its internal structure through reorientation of trabeculae. During normal aging and menopause, cancellous bone is lost at all skeletal sites due to remodeling-related factors: negative formation balance; temporarily increased remodeling space; and osteoclastic perforations. Osteoporosis, characterized as loss of bone mass with deterioration of microstructure and material properties, is known as a growing public health problem [1,2]. One can appreciate, for example, three-dimensional synchrotron computed-tomography image of a 4-millimeter-cube of trabecular bone from the vertebra interior, previously published by Parker [3]. The case of a 30-year-old female bone of normal density and architecture shows a significant number of trabeculae and plate-like structures are aligned in the load-bearing direction (Fig. 1a). A similar image of 63 year old male exhibits markedly different bone architecture, with fewer trabeculae and plate-like structures and more of the trabeculae shaped like slender rods (Fig. 1b).

What is well argued is that [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. A loss of trabecular connectivity such that vertical weight-bearing bars lose their cross attachments with each other, becoming susceptible to buckling, have been described as

* Corresponding author.
E-mail address: ramtani@univ-paris13.fr (S. Ramtani).

http://dx.doi.org/10.1016/j.irbm.2015.07.004

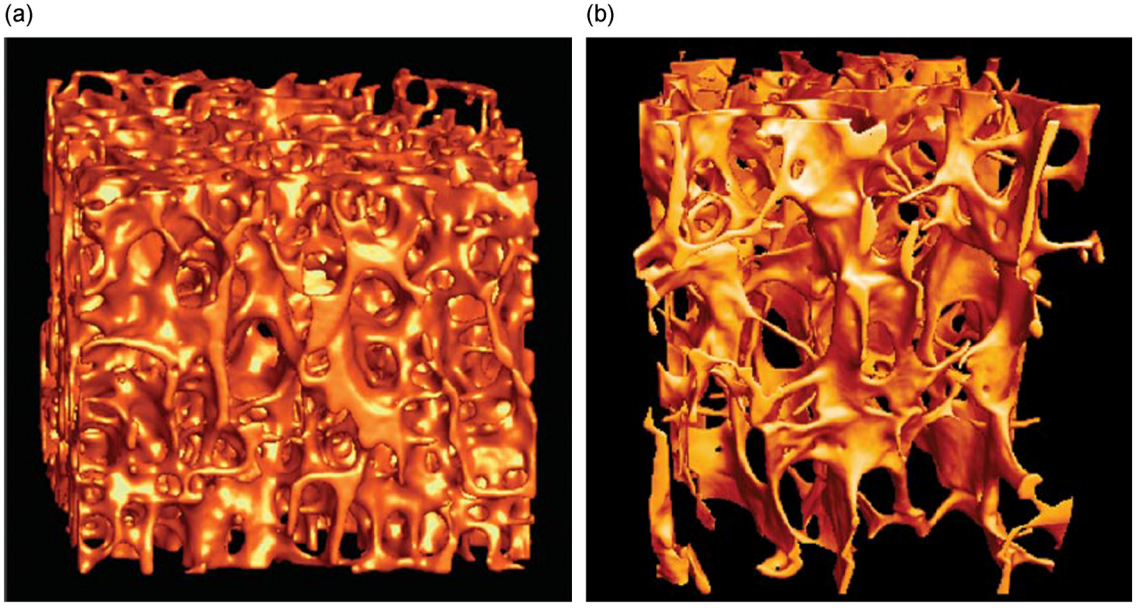

Fig. 1. (a) A three-dimensional synchrotron computed-tomography image of a 4-millimeter-cube of trabecular bone from the vertebra interior of a 30-year-old female shows bone of normal density and architecture. A significant number of trabeculae and platelike structures are aligned in the load-bearing direction. (b) A similar image of a 63-year-old male shows a markedly different bone architecture, with fewer trabeculae and platelike structures. In addition, more of the trabeculae are shaped like slender rods. (Credit from Lawrence Livermore National Laboratory, [3].)

an abnormality that can account for skeletal fragility disproportional to the degree of bone loss [3–5]. In order to investigate the influence of bone structure type on the failure mechanism, Müller et al. [6] measured samples of bovine tibiae and whale spine. They found that in the whale spine's rod like type of architecture, structural failure consisted of the initial buckling and bending of structural elements followed by the collapse of the overloaded trabeculae.

The hypothesis that scoliosis progressed with resorption of loaded bone has been adopted by Goto et al. [7] for computational analysis using the finite-element method. They investigated the influence of bone modeling on the buckling spine and compared changes in the coronal and the transverse planes of idiopathic thoracic scoliosis. As a result, they suggested that scoliotic changes in the spinal column triggered by the buckling phenomenon are counteracted by bone formation, but worsened by bone resorption.

The usual assumption that the increase of fractures in aging bone is due entirely to lower bone density is considered here with respect to the possibility that aging bone fractures result from a loss of its elastic stability, or buckling, in the structure of the bone lattice. With respect to known clinical investigations, there is a great need for a theoretical framework that provides insight into the bending–buckling process coupled to bone adaptation which accounts for many fracture mechanisms. As a first step in this direction, we have numerically investigated the particular case of adaptive Euler–Bernoulli beam model due to Ramtani et al. [8] by adding bone density gradient effect using a finite difference method (number of equidistant nodes $n = 35$) and the power method in order to evaluate the largest eigenvalue and corresponding eigenvector.

This study intends globally to establish, from mechanistic point of view, a better knowledge about bone-column buckling mechanism affected by both bone density and bone density gradient distributions. In this contribution, among other results, we indicate and confirm that the activation of bone loss process can lead to the apparition of new elastic instabilities that can conduct to local bone-buckling mechanism of fracture localized at the trabeculae scale. We also demonstrate that bone density gradient plays a key role in the initiation of the bone-column elastic buckling instability.

2. Materials and methods

The present work is conducted within the context of the well-known theory of adaptive elasticity originally stated by Cowin and coworkers [9,10]; and usually applied as a model for the physiological process of bone adaptation. Let us first define the bone volume fraction resultant over the bone-column's cross-section A as follows

$$\bar{e}(x,t) = \int_A e(x,y,z,t)dydz. \tag{1}$$

where $e(x,y,zt) = \xi(x,y,z,t) - \xi_0$ is the measure of the change in solid phase volume fraction from a reference volume fraction ξ_0, whereas ξ is the solid phase volume fraction in the reference unstrained state.

For the adaptive elastic bone material, local *Cauchy* stress has been previously expressed as a modified form of Hooke's law in that the proportionality between stress and strain is dependent upon the volume fraction of the material present $\bar{e}$ [9,10]

$$\sigma_{ij} = \left\{\xi_0 C^{(0)}_{ijkl} + \bar{e}C^{(1)}_{ijkl}\right\}\varepsilon_{kl} = \xi_0\sigma^{(0)}_{ij} + \bar{e}\sigma^{(1)}_{ij} \tag{2}$$