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Relative Roles of Cortical and Trabecular Thinning in Reducing Osteoporotic Vertebral Body Stiffness: A Modeling Study

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Abstract— While the effects of reduced bone density on osteoporotic vertebral strength are well known, the relative roles of cortical shell and trabecular architecture thinning in determining vertebral stiffness and strength are less clear. These are important parameters in investigating the changing biomechanics of the ageing spine, and in assessing the effect of stiffening procedures such as vertebroplasty on neighbouring spinal segments. This work presents the development of a microstructural computer model of the osteoporotic lumbar vertebral body, allowing detailed prediction of the effects of bone micro-architecture on vertebral stiffness and strength.

Microstructural finite element models of an L3 human vertebral body were created. The cortex geometry was represented with shell elements and the trabecular network with a lattice of beam elements. Trabecular architecture was varied according to age. Each beam network model was validated against experimental data. Models were generated to represent vertebral bodies of age <50 years, age 50-75y and age >75y respectively. For all models, an initial cortical shell thickness of 0.5mm was used, followed by reductions in the age >75y models to 0.35mm and 0.2mm to represent cortical thinning in late stage osteoporosis. Loads were applied to simulate in vitro biomechanical testing, compressing the vertebra by 20% of its height.

Predicted vertebral stiffness and strength reduced with progressive age changes in microarchitecture, demonstrating a 44% reduction in stiffness and a 43% reduction in strength, between the age <50 and age >75 models. Reducing cortical thickness in the age >75 models demonstrated a substantial reduction in stiffness and strength, resulting in a 48% reduction in stiffness and a 62% reduction in strength between the 0.5mm and 0.2mm cortical thickness models. Cortical thinning in late stage osteoporosis may therefore play an even greater role in reducing vertebral stiffness and strength than earlier reductions due to trabecular thinning.

Keywords— Vertebra mechanics, FE modeling, trabecular architecture, osteoporosis, cortical shell

I. INTRODUCTION

Osteoporosis is a disease which affects more than 75 million people in Europe, Japan and the USA, and is the cause of more than 2.3 million fractures annually in Europe and America alone [1]. Osteoporosis is characterized by

low bone density and micro-architectural deterioration of bone tissue, resulting in increased susceptibility to fracture [2]. The micro-architectural deterioration includes thinning of the trabeculae, increased spacing between trabeculae, and in the later stages, thinning of the cortical shell. These changes transform the structure from dense and plate-like to a sparse, rod-like structure. It is believed there is an associated change in the failure mechanisms from plastic collapse of the trabeculae to inelastic, or possibly even elastic, buckling of the trabeculae. Due to the experimental difficulty in investigating this complex structure, the effect of bone loss on trabecular failure mechanisms and whole vertebra mechanics is still poorly understood.

To overcome experimental difficulties, previous studies have employed computational modeling methodologies. These studies utilize two distinct approaches. Firstly, the macro scale approach, whereby a solid, whole vertebra is simulated and the structure of the trabecular bone approximated as a continuum. The material properties of the vertebral core are varied to represent the changes in trabecular structure and the effect of this change on whole vertebra mechanics is observed. Secondly, micro scale approaches, whereby an isolated section of the trabecular structure is simulated using either continuum or beam elements. Continuum elements are computationally expensive and therefore only small regions of bone can be modeled. Conversely, computationally efficient beam elements have been used to simulate larger regions of trabecular structures. The results for these micro scale models are then extrapolated for a whole vertebra.

This study aimed to create a multi-scale finite element model of an L3 human vertebral body. The architectural changes observed in osteoporosis (trabecular thinning, increased trabecular spacing and cortical shell thinning) were modeled and the effect of these changes on whole vertebra mechanics and trabecular mechanics explored.

II. METHODS

A finite element model of an L3 human vertebra was analysed under compression to assess the relative affect of cortical and trabecular microstructure on vertebral mechanics. The trabecular structure was modeled using threedimensional beam elements and the vertebral cortex simulated with shell elements. Model development involved ascertaining the efficacy of the beam elements in simulating trabecular biomechanics, validation of the beam element trabecular lattice and simulation of an intact vertebra.

A. Modeling the trabecular core

Three dimensional beam elements were used to represent individual struts within the trabecular lattice. As buckling is an important failure mode of longitudinally oriented trabeculae [3], it was important that the model accurately predicted buckling behavior. Therefore, analyses were performed on various beam element configurations to determine an appropriate trabeculum model. The effect of element type, initial beam curvature, mesh density and solution time increment were investigated.

Due to the paucity of data on trabeculum failure mechanisms at the microstructural level (specifically buckling), it was necessary to use a buckling mechanics study based on another material to verify the trabeculum model. Investigations performed by Rahman [4] gave critical buckling loads determined experimentally for solid columns of stainless steel (SUS304). This work covered a comprehensive range of slenderness ratios (14-184) and hence failure modes, from purely elastic to purely plastic. The material properties and slenderness ratios of the trabeculum model were altered to represent the steel columns, and Rahman's experimental results used to verify the prediction of the model. From these analyses it was concluded that two quadratic beam elements, with a slight initial curvature, were able to predict failure (be it elastic buckling, inelastic buckling, or plastic collapse) with a mean error of 20% for the whole range of slenderness ratios. For the trabeculum model, an initial offset of 0.001mm in the centre of the column was necessary to induce buckling. Once confidence was obtained in the ability of the FE techniques to predict buckling, they were applied to represent a trabecular core model.

A three dimensional trabecular core was created using a lattice of individual trabeculum beams. Three-node, quadratic beam elements were used to represent the longitudinal trabeculae, and two-node, linear beam elements represented the transverse trabeculae. Since the transverse trabeculae are primarily loaded in tension, the additional complexity of the quadratic beam representation was considered unnecessary. To provide a degree of irregularity to the lattice, as is seen in real trabecular bone, a perturbation factor of 0.3 was applied [5]. The perturbation factor defines the maximum distance each node may be perturbed by as a proportion of the trabecular spacing. For example, with a perturbation value of 0.3, each node point was perturbed $\pm 0-30\%$ of the spacing value.

The trabecular spacing and thickness values for the transverse and longitudinal trabeculae were derived from Mosekilde [6]. The values are shown in Table 1. Three trabecular structures were created; age < 50, age 50-75 and age > 75. A tissue modulus of 8GPa and a Poisson's ratio of 0.3 were applied [7, 8]. An elastic-perfectly plastic yield definition was included which had a yield strain of 0.85% and a yield stress of 68 MPa. The yield strain was determined as an average of the reported compressive and tensile yield strains for trabecular bone [9].

Table 1 Trabecular spacing and thickness values for female vertebral trabecular bone for age < 50, age 50-75 and age > 75 [6]

Model	Transverse Spacing (mm)	Longitudinal Spacing (mm)	Transverse Thickness (mm)	Longitudinal Thickness (mm)
Age <50	0.674	0.633	0.150	0.208
Age50-75	0.861	1.100	0.116	0.187
Age >75	1.145	1.668	0.107	0.201

The trabecular core model was verified against experimental results. As well as providing structural parameters for three age groups, the Mosekilde study also provided the compressive strength of the trabecular cores for these age groups. To provide a comparison, trabecular core models were created to replicate the in vitro cylindrical bone samples tested by Mosekilde (radius 3.5mm, length 5mm). To replicate the axial compression test performed in the study, the upper nodes of the cores were were free to move only in the axial direction and held in all other degrees of freedom. All bottom surface nodes were held in all degrees of freedom. The upper nodes were displaced in the axial direction until failure of the core occurred. The models were solved using ABAOUS/Standard (version 6.7, Abagus Inc, RI, USA) using a large displacement (non-linear geometry) quasi-static solution procedure. The maximum compressive strength and stiffness of the cores were determined and compared to the experimental results. Once confidence was gained in the trabecular core model, an intact vertebra was simulated.

B. Modeling the Vertebra

Using a similar methodology to that employed for the trabecular core, an intact vertebra was simulated whereby the inner trabecular lattice of the vertebra was enclosed by a thin vertebral cortex. Age < 50, age 50-75 and age >75 vertebra models were produced. The cortex was meshed using three dimensional, linear shell elements and the ge-

ometry was based on equations given by Mizrahi [10]. The mesh density gave shell elements 2mm in size. The material properties of the cortex were assumed to be the same as the trabecular bone [7, 8]. The thickness of the shell elements was 0.5mm, which represents a normal cortical shell [11]. In the age > 75 model, the shell thickness was reduced to 0.35mm, and then again to 0.2mm to represent shell thinning, as is observed in the later stages of osteoporosis [11]. This resulted in five vertebra models, all at different stages of osteoporosis.

Loads were applied to simulate in vitro biomechanical testing, compressing the vertebra by 20% of its height. The upper endplate was displaced by -6mm axially and held in the transverse plane. The lower endplate was held in all directions. A quasi static solution step (total length 1 sec) was used with a minimum time increment of 0.01 sec and a maximum time increment of 0.1 sec. As previously stated, the ABAQUS non linear geometry capability was used to include the effect of large deformations in the solution.

III. RESULTS

The apparent modulus of the trabecular cores and vertebral models were determined using the linear region of the stress strain graph, between 0-0.4% apparent strain. The maximum compressive strength was considered to be the maximum total vertical reaction force that was reached in the simulation.

A. Trabecular core

Table 2 shows the compressive strength of the various trabecular core models determined experimentally by Mose-kilde [2] and the corresponding computed compressive strengths from the trabecular core model. The table also shows the apparent stiffness of the cores determined computationally, however Mosekilde did not report the stiffness of the cores tested experimentally and hence no direct comparison can be made. Other studies have reported vertebral trabecular bone samples from the lumbar spine have an apparent modulus of 165 ± 110 MPa [12].

Table 2 Compressive strength of the trabecular core samples determined experimentally by Mosekilde and by FE trabecular core model and the stiffness of the cores predicted by the FE models

	Age < 50	Age50-75	Age >75
Mosekilde maximum	3.91	1.35	0.93
compressive strength (MPa)	± 1.61	± 0.64	± 0.4
FE trabecular core maximum compressive strength (MPa)	2.84	1.21	0.54
FE trabecular core apparent modulus (N/mm)	253	138	74

B. Vertebra Model

Figure 1 shows the stress strain curve for each of the vertebra models. The corresponding stiffness and compressive strengths are shown in Table 3.



Fig. 1 Stress versus strain for the vertebra models.

Table 3 FE predicted compressive strength and stiffness of the vertebra models

Model	Cortical thickness (mm)	Stiffness (N/mm)	Max. Compressive strength (kN)
Age <50	0.5	595	5.74
Age50-75	0.5	412	4.06
Age >75	0.5	336	3.28
Age >75	0.35	256	2.30
Age >75	0.2	176	1.25

IV. DISCUSSION

The trabecular core model was able to reproduce compressive strengths and apparent moduli determined experimentally. The predicted compressive strengths for the cores of various structures were within one standard deviation of Mosekilde's experimental results for the corresponding ages. The apparent moduli determined computationally were within the range of values (165 ± 110 MPa) in the literature [12]. With these results, confidence was achieved in the trabecular beam model.

The vertebra model confirms that changes in architecture have a large effect on overall vertebra stiffness and strength. A change in architecture from the age < 50 to the age > 75 cases resulted in a 44% decrease in stiffness and a 43% decrease in vertebral strength. With the age > 75model, a change in shell thickness from 0.5mm to 0.2 mm, without any change in trabecular structure, resulted in a 48% decrease in stiffness and a 62% decrease in compressive strength. These results not only highlight the importance of the trabecular architecture changes that occur with the osteoporosis process, but also the biomechanical importance of the cortical thinning that occurs in the later stages of the disease.

A current limitation of this model is that it has yet to be validated against experimental data for a full vertebral body (including the cortical shell). However validation of the trabecular core model and initial comparisons with the literature indicate the predictions of the vertebra model are reasonable. Reported vertebral body compressive strengths range from approximately 60MPa for a 20 year old vertebra to 2.6MPa for an 80 year old vertebra [13]. The predicted compressive strengths for the vertebral models of different ages are comparable with these values, although slightly lower.

The premise for this modeling approach was that buckling mechanisms dominate the response of rod-like osteoporotic bone. Hence, replicating the trabecular network using beam elements provides a sophisticated microstructural model capable of simulating plastic collapse, inelastic buckling, or elastic buckling in bone of various ages. Taking a closer look at the trabecular struts in the age > 75vertebra model shows the trabecular beams are undergoing large buckling deformation, but no plastic deformation is seen, indicating that the overall failure of the vertebra is due to elastic buckling of the trabeculae. In the age 50-75 model, the beams also experience large amounts of buckling; however there is also plastic deformation throughout the structure. This suggests inelastic failure of the trabeculae is playing a key role in the vertebra failure. Finally, the beams of the age < 50 model show almost no buckling, yet a high amount of plastic deformation, signifying plastic collapse of the structure. While further model investigation and validation needs to be done before any quantitative data on the trabeculae can be reported, these results highlight the distinctive insight into both the trabecular and vertebral mechanics this model allows.

In future work, this model will be validated against human vertebra specimens. Once validated, it will be used to investigate current drug therapies and their effects on the bone architecture and vertebral strength, as well the effect on trabecular and vertebral strength of surgical treatments such as vertebroplasty.

V. CONCLUSION

This paper has presented the development of a novel multi-scale, vertebra model produced with beam and shell

elements. The model predictions have been validated against experimental data in existing literature and show good agreement. The investigation into the effects of changes in architecture indicate that while the changes in trabecular architecture have a large effect on vertebral strength and stiffness in the early stages of osteoporosis, cortical thinning may have as great an effect (if not greater) in the later stages.

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