Investigation of the Effect of Spinal Defects on Spondylolysis & Stress Fracture of Vertebral Bodies

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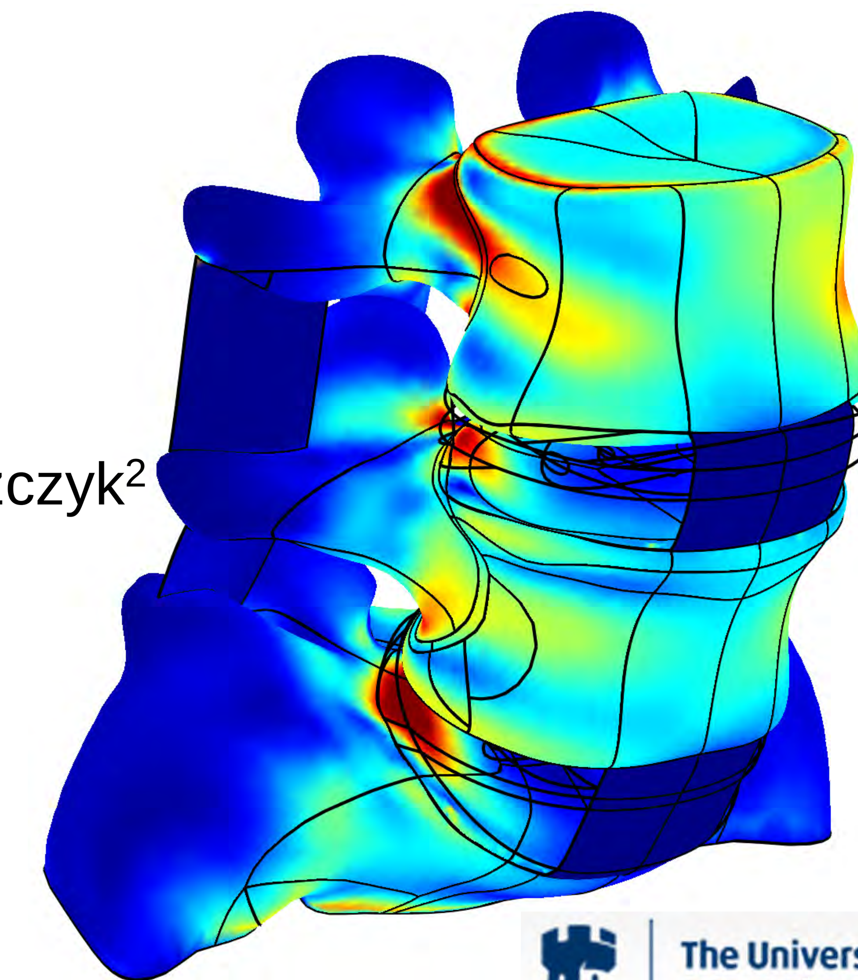


INTRODUCTION

Spondylolysis (SL) of the lower lumbar spine is frequently associated with spina bifida occulta (SBO). A recent investigation revealed a 3.7 fold increase in the presence of SL in individuals with SBO. However, it is unclear if SBO is a predisposition for the development of SL. SL is recognised as being a fatigue fracture with an increased incidence among athletes participating in disciplines requiring repetitive forceful hyperextension, axial loading & rotation of the spine.

AIM

To model both a SBO defect & an intact spine under combined axial load & rotation, assessing the stresses observed on the vertebral bodies, and their distribution on the ipsilateral & contralateral inferior isthmus lines, where fatigue fracture is likely more likely to occur.

Figure 1. SBO model adapted from Intact case

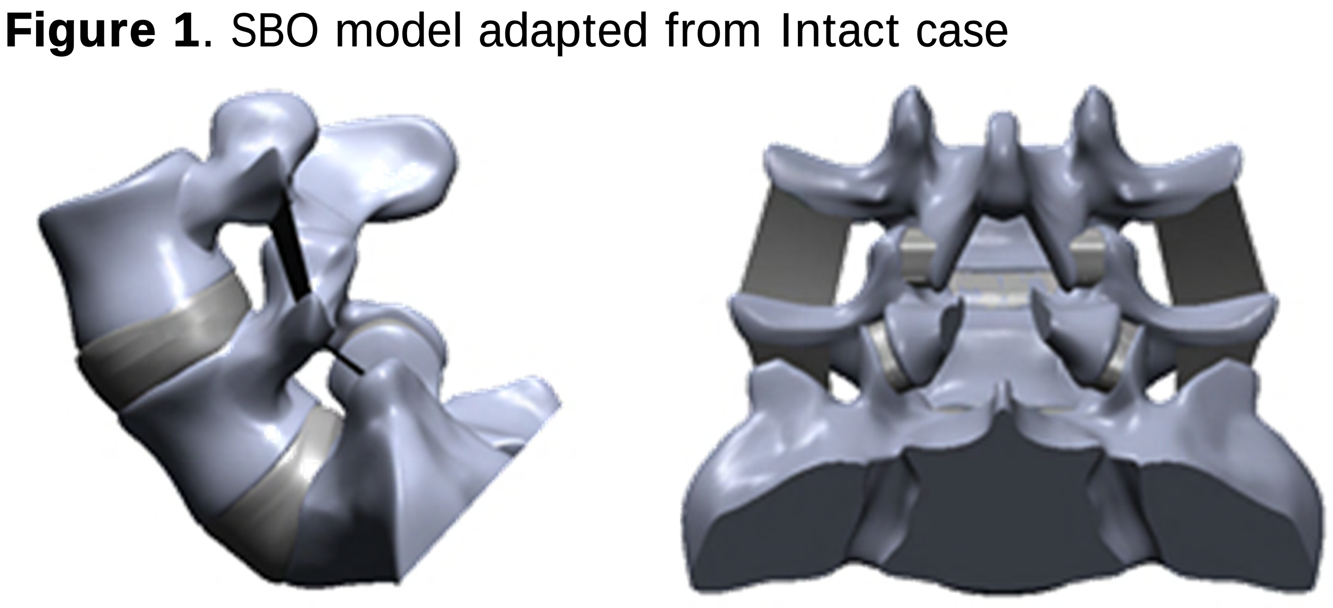
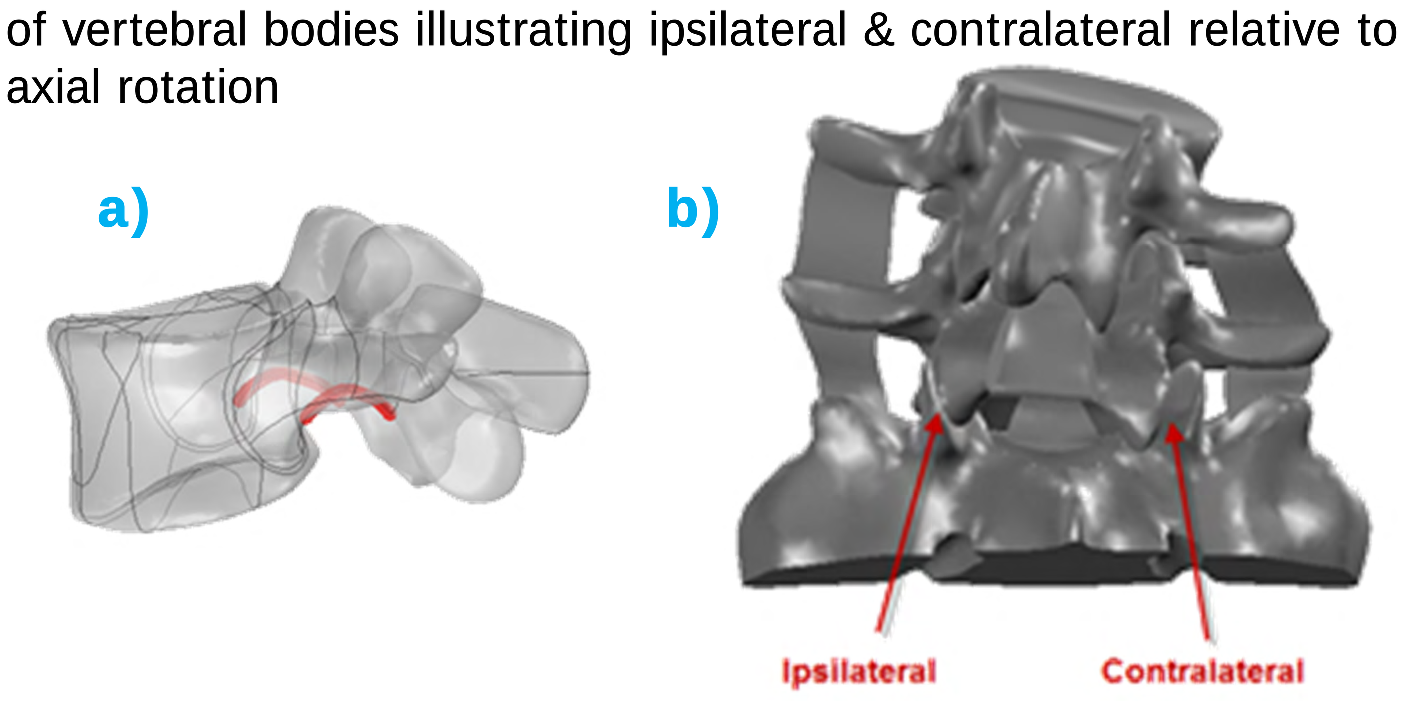


Figure 2. a) Inferior Isthmus Lines Assessed & b) Posterior view of vertebral bodies illustrating ipsilateral & contralateral relative to axial rotation



METHOD

A 3D model of a validated intact L4-S1 human lumbar motion segment including ligaments was utilised[1-2]. The intact model was adapted to mimic the SBO condition, by removing a section of the L5 vertebral arch & spinous process (Figure 1)[3]. The sacral slope of both intact & SBO models were orientated to 66°, mimicking the degree of sacral slope in athletes with a high pelvic incidence. Two load conditions were studied (Figure 2), an axial load of 1kN applied to the superior vertebral endplate of the L4 spine segment, & the same axial load combined with a 3° axial rotation. Stresses on both ispsilateral & contralateral inferior lines of the L5 vertebra were assessed & compared (Figure 3 & 4). Mean & alternating stress values were obtained & used in a Goodman diagram with the Soderberg relationship to find the stress amplitude & mean stress to an equivalent alternating shear stress. These values were then used to predict the number of cycles to failure (N_f), using the following relationship from Literature [4]:

$$S = S_o + S_r \log(N_f)$$

Where:

S = Fatigue strength
S_o = 36.6 MPa (Shear strength in a single fatigue cycle failure)
S_r = -2.9 MPa

RESULTS

Under static load conditions, the SBO ipsilateral pedicle experiences higher stresses in the mid-region of the isthmus line (ventral to dorsal) compared to the intact model (Figure 3), with a maximum observed at a position 37% of the length from the ventral side.

The maximum stress amplitude for the SBO & Intact models were 22.5 & 13.6 MPa, respectively (Figure 4). A stress amplitude above 16.3 MPa will result in a fracture in under 10 million cycles. In the intact case, the maximum shear stress amplitude remained below 14 MPa & is unlikely to failure. However, the SBO model will result in a fatigue fracture after 70,000 cycles, & will most likely be located along the red section as indicated in Figure 5.

Figure 3. Inferior L5 Isthmus Shear Stress Under 1kN Axial Load & 3° Rotation

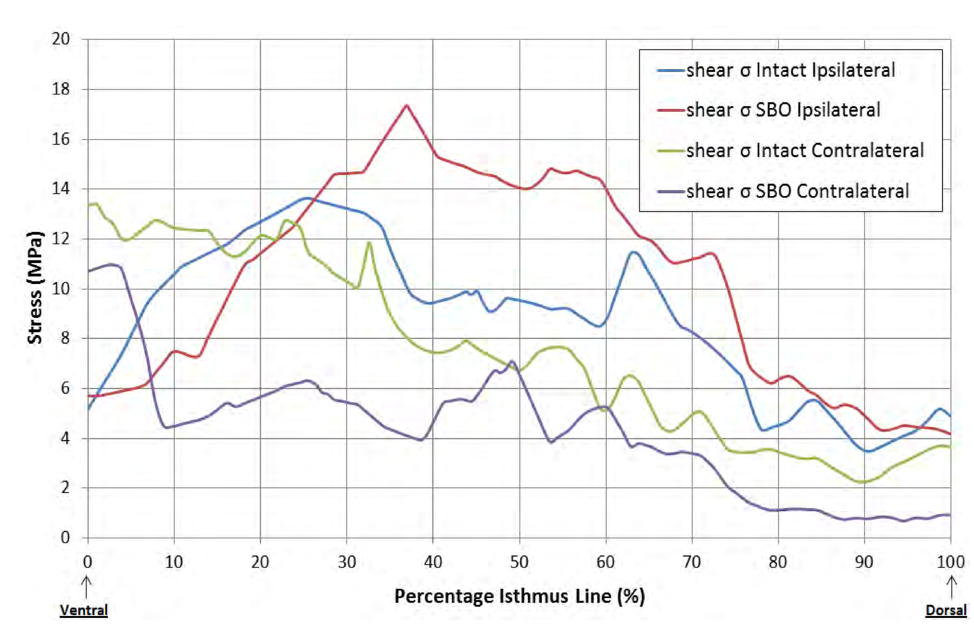


Figure 4. Inferior L5 Isthmus Stress Amplitude & Fatigue Load Under 1kN Axial Load & 3° Rotation

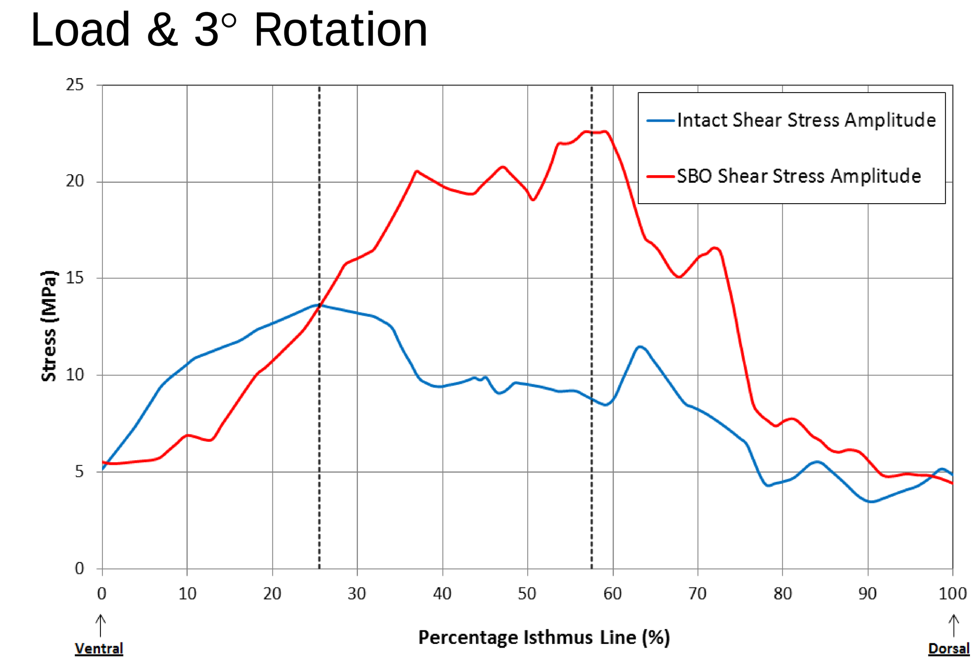


Figure 5. Intact & SBO Inferior L5 Isthmus Lines Illustrating Fatigue Failure Region (Red)

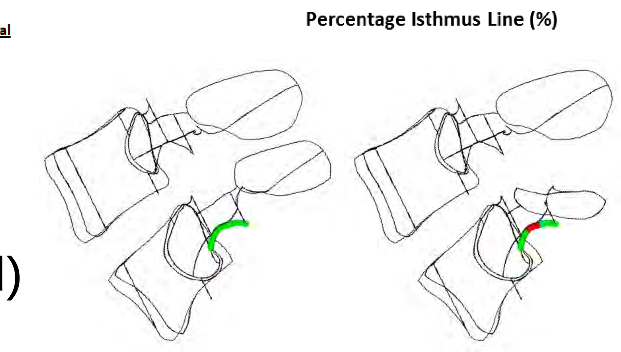
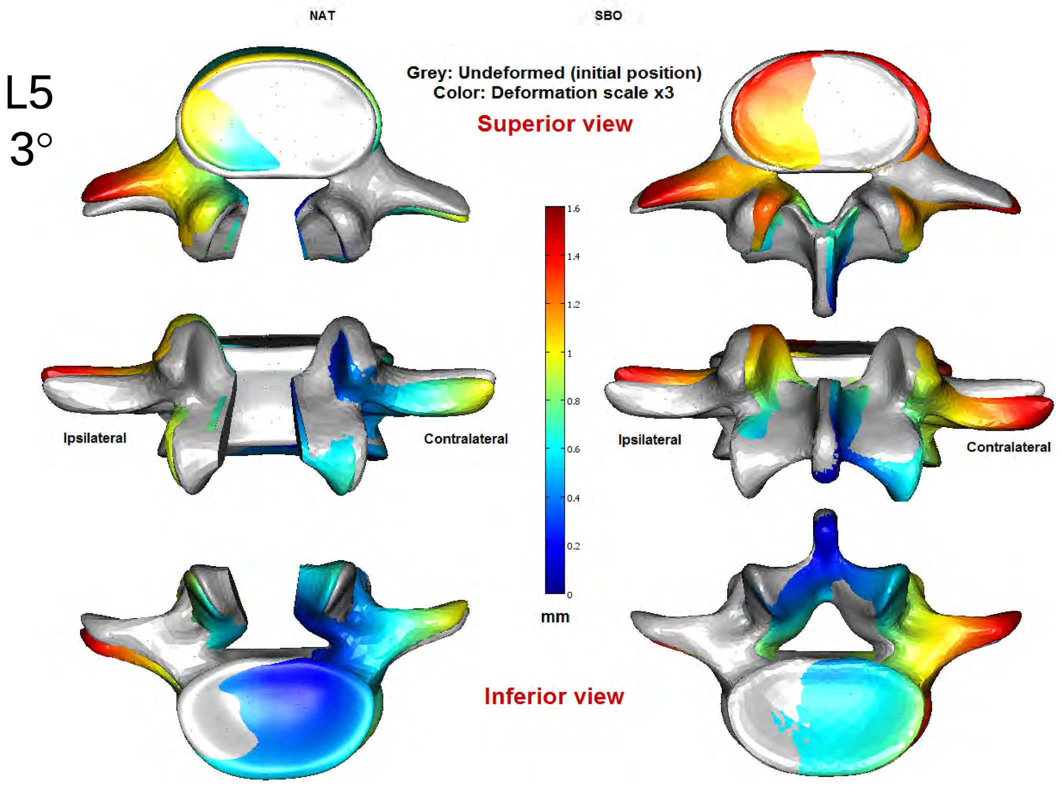


Figure 6. Displacement Plots of L5 Vertebra Under 1kN Axial Load & 3° Rotation for SBO & Intact Models



DISCUSSION

SBO predisposes SL, by generating increased stresses across the inferior isthmus of the inferior articular process, especially under combined axial load & rotation. Axial loading alone was not sufficient to generate stresses that would cause fracture or failure.

CONCLUSION

The study suggests that SBO increases load across the pars & predispose the vertebral body to early fatigue fracture, especially in athletes involved in activities requiring forceful rotational loading. This leads to the hypothesis that mechanical factors play a dominant role in the increased incidence of SL in patients with SBO than genetic predispositions.

References:

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