MICROFRACTURE OF HUMAN THORACOLUMBAR VERTEBRAL BODY UNDER FATIGUE LOADING

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The purpose of this study was to investigate the relationship between lumbar vertebral microfracture and fatigue loading on young human spine under physiological cyclic compression loads. Thirty-three thoracolumbar vertebrae (T12 to L4) were obtained from 7 adult Chinese male cadavers. They were randomly divided into 5 groups. Cyclical compression was performed for 20,000 cycles with 2 Hz. Load magnitude was determined respectively as 10%, 20% and 30% of the ultimate compressive load. Four cylindrical sections were obtained from each vertebra and the cross-sectional slides were made. The histomorphometry was used to determine microfracture densitiy and distribution. No fracture was detected in the radiographs of groups III, IV and V after fatigue load. Microfracture density in the cyclic compression group increased from 0.46 #/mm^2 in Group III to 0.66 #/mm^2 (Group IV) and 0.94 #/mm^2 (Group V) under different loading levels ($p < 0.05$). These results provide evidence for the existence of microfractures caused by fatigue loads that are undetectable by X-ray.

KEY WORDS: microfracture, lumbar spine, biomechanics

INTRODUCTION: Stress fractures or fatigue fractures of the long bone with chronic pain are a well-known occurrences among athletes and military recruits(5). Epidemiological studies on the incidence of low back disorders associated with transportation workers, however, indicate that there may be a connection between cyclic loading and chronic low back pain (2,6). Hence, it is hypothesized that chronic low back pain may be related to fatigue microdamage during normal daily repetitive stress (7). No study has documented the distribution of micro-fracture within a single vertebral body under fatigue loads. Thus, the purpose of this study was to investigate the relationship between lumbar vertebral microfracture distribution and fatigue loading on a young human spine under cyclic compression at physiological levels.

METHODS: Seven human thoracolumbar spine specimens consisting of T12 to L4 were obtained from adult male cadavers (25 - 32 years old). The 7 specimens were cut into 33 separate vertebrae (two vertebrae were excluded because of the injury during preparation). The 33 vertebrae were randomly divided into 5 groups: I) control group (without loading) (n=6); II) compression group (loading to failure with axial compression) (n=6); and III to V) loading with cyclic compression but with different strengths in each group respectively (each n=7). The bone mineral density (BMD) of all the specimens were determined using the Fan Beam X-ray Bone Densitometer (Hologic Inc., USA). The estimated vertebral area and estimated bone mineral content were also obtained.

Axial loads were applied to group II specimens using an MTS Bionix 858 testing machine (MTS, Minneapolis, MN, USA). A steel plate, 10mm in thickness and 200mm in diameter covering surface of the specimen, was attached to the piston to ensure uniform axial loading on the vertebral body. The inferior endplate of specimens were embedded in a steel cup by Epoxy. The loading speed was 5mm/min. The axial load and displacement data were acquired per 0.1 second. The ultimate loads of all the specimens were then obtained and the failure stress was calculated by dividing the loads by the estimated vertebral body area .

Minimum initial load of cyclic compression was 350N (which is equal to the weight of upper body) in groups III, IV and V. Maximum cyclic compressions were defined as the 10%, 20% and 30% of the average ultimate compression load from group II. Mechanical loading was applied on the vertebral body for 20,000 cycles with a sine wave at a frequency of 2 Hz. Specimens in group I (Control) were not loaded. The load and deformation were recorded for 5 cycles in every 1000 cycles, and stiffness was determined as the slope of load-deformation curve using linear regression of the 5th cycle.

After loading, anteroposterior and lateral radiographs were taken to examine if the specimen has fractured. Four cylindrical sections were then taken from the center of the vertebral body using a core drill (diameter= 8 mm). The bone marrow of the cylindrical bone specimens was cleaned by water jet, then by ultrasound bath (Branson Ultrasonics Corporation, USA). After staining, two 200μm thick sections were made from the middle of the cylindrical specimens by using a steel saw. Histomorphometry was used to determine trabecular bone area and microcrack density in the histological sections using an Opton B-9901 microscope (Opton, Germany) equipped with an indexed grid (Graticules Ltd., Tonbridge Kent, England). Each section was divided into 3 equal regions along the longitudinal axis under the microscope: superior, middle and inferior (Figure 1). At ×100 magnification, 10 fields of 1 mm^2 were examined in each region. The microcrack density of each region was calculated by dividing number of microcracks by the trabecular bone area. BMD, stiffness and microcrack density were averaged in each vertebra and compared among groups using two-way ANOVA, followed by Student-Newman-Keuls (SNK) test using statistical software SPSS 8.0. with significance graded at $p < 0.05$.

RESULTS: The average bone mineral density of each group was 0.46 ± 0.03 g/cm^2, ranging between 0.45-0.47g/cm^2. There was no statistical significance between the BMD of each group. The average ultimate axial compression loading of group II was 4853.83 N and the average stress was 3.35 MPa.

The stiffness of specimens in group III was relatively constant during the cyclic compression test. However, the stiffness of specimens in groups IV and V significantly increased during the first 10,000 cycles (Figure 2). An increased stiffness indicated the less recovery of trabecular bone after deformation. The results showed that there were significant differences in stiffness between groups III (9.16%) and IV (25.26%) ($p<0.05$), as well as between groups III and V (25.42%) ($p<0.05$).

The microfractures in vertebral trabecular bone, which displayed as the cracks with increased basic fuchsin stain surrounding the border (Figure 3), ran parallel to the trabecular surface. There were significant differences in microfracture density among groups (Table 1). Microfracture density in Group I (control without loading) was 0.25 ± 0.05#/mm^2. Microfracture density significantly increased with fatigue loading between Groups III, IV and V ($p<0.05$). Density increased from 0.45 #/mm^2 in Group III to 0.64 #/mm^2 in Group IV (42.2% increase), and to 0.94 #/mm^2 in Group V (108.9% increase). It was observed that microfracture density in the fractured group II (0.67±0.21#/mm^2) was less than that in Group V ($p < 0.05$), higher than in Group III ($p<0.05$). Under microscopic observation, more broken trabecular bone rather than microfractures were found in the specimens of Group II. There was no statistical difference between different regions in each fatigue group.(Table 1).

Table 1 Average Stiffness Change Rate and Microfracture Density in Different Regions

Group	Stiffness (%)	Superior Region (#/ mm^2)	Middle Region (#/mm^2)	Inferior Region (#/ mm^2)	Total Area (#/mm^2)
III	9.15	0.40	0.46	0.50	0.45
S.D.	± 6.6	± 0.12	± 0.13	± 0.20	± 0.15
IV	25.26	0.67	0.66	0.59	0.64
S.D.	±18.2	± 0.21	± 0.23	± 0.12	± 0.19
V	25.42	0.87	1.02	0.94	0.94
S.D.	±18.3	± 0.27	± 0.21	± 0.17	± 0.22

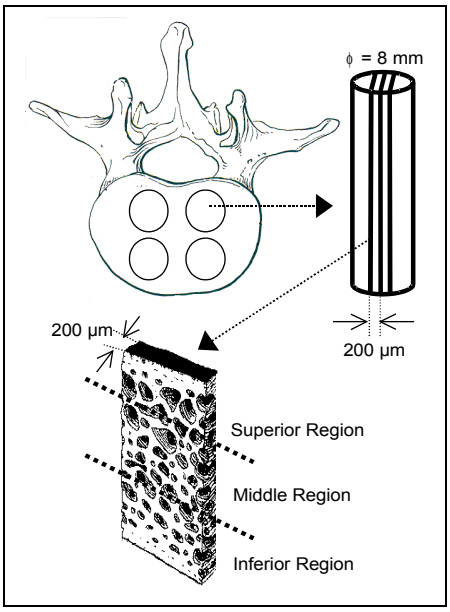

Figure 1 - Schematic diagram of experimental design for measuring microfracture after mechanical fatigue testing.

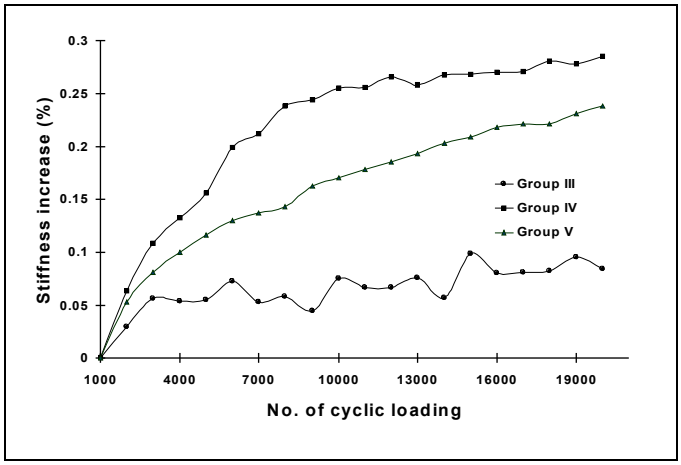

Figure 2 - Average stiffness change of specimens during cyclic loading in groups III, IV and V. Stiffness significantly increased during initial 5000 cycles.

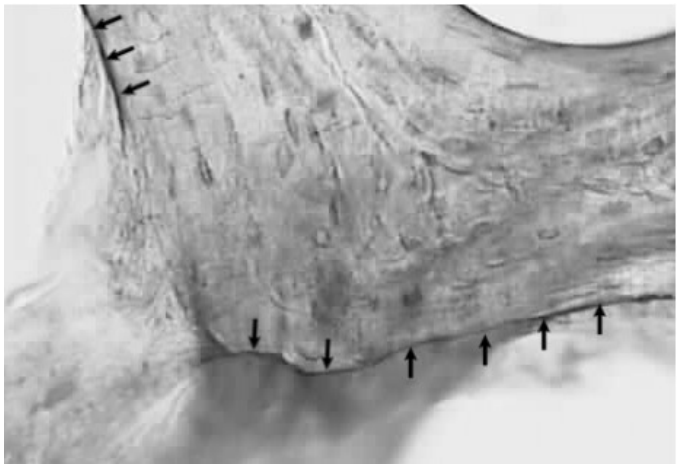

Figure 3 - Representative microfractures in vertebral trabecular bone. Arrow indicates cracks with a increased basic fuchsin stain surrounding the border. The microcracks ran parallel to the trabecular surface. (Magnification 400x).

DISCUSSION: This study investigated the physical nature of fatigue damage along with the relationship between microcrack density and cyclic loading in the lumbar spine of young Asian males. Results supported previous studies(1,4) that vertebral body fracture can occur with cyclic compressive loads within physiological loading ranges. In addition, results provided new insight into the physical nature of a single applied load and fatigue damage responsible for microfracture. Histological results revealed that microfracture density increased with cyclic loading strength, but the microfracture density in the single applied load group was significantly lower than that in fatigue loading. This study also hypothesized that when a load was added onto the vertebrae, microfracture appeared first. As the load was accumulated, trabecular fracture occurred. There was no previous report on microfracture and its density in the human vertebra after axial compression to failure.

Most previous studies focused on the fatigue failure of intervertebral discs during dynamic compressive loading (4). Some of the studies clearly indicated that cyclic loading during normal activity can result in fatigue failure. The type of fracture and fatigue strength appears

to be related to aging and/or degenerative changes associated with aging(3). However, those studies only present macroscopic evidence of failure. The microfracture density distribution results of current study suggested that there was no difference among different regions (superior, middle, inferior) of a vertebra. The results in this study are obtained from Asian adult lumbar specimen of a fairly young age range and should be able to contribute to the literature where most of the data were obtained from old human subjects or animal specimens. Furthermore, results in this study indicated that response due to stiffness increase in vitro may be parallel to the in vivo scenario, that a reduced height of spinal column is contributed not only by disc displacement, but also due to the deformation of each vertebral body. In the long-term, this may contribute to the increased chance of fracture occurrence.

In conclusion, microfractures were found within the vertebral body after fatigue loads and the fracture was not detected in the radiographs. After fatigue load, the axial stiffness or deformation increased after the applied load increase. Microfracture density was also increased with the load increase.

REFERENCES:

Adams, M.A., & Hutton, W.C. (1983). The effect of fatigue on the lumbar intervertebral disk. *Journal of Bone and Joint Surgery (Br),* **65**, 199-203.

Andersson, G.B. (1998). Epidemiology of low back pain. *Acta orthopaedica Scandinavica. Supplementum,* **281**, 28-31,1998.

Fazzalari, N.L., Forwood, M.R., Smith, K., Manthey, B.A., & Herreen, P. (1998). Assessment of cancellous bone quality in severe osteoarthrosis: bone mineral density mechanics and microdamage. *Bone,* **22**(4), 381-388.

Liu, Y.K., Njus, G., Buckwalter, J., & Wakano, K. (1983). Fatigue Response of lumbar intervertebral joints under axial cyclic loading. *Spine,* **8**, 857-865.

McBryde, A.M. (1976). Stress fracture in athletes. *Journal of Sports Medicine,* **3,** 212-217.

Sandover, J. (1983). Dynamic loading as a possible source of low-back disorders. *Spine,* **8**, 652-658.

Yoganandam, N., Maiman, D.J., Pintar, F., Ray, G., Myklebust, J.B., Sances, A. Jr., & Larson, S.J. (1988). Microtrauma in the lumbar spine:a cause of low back pain. *Neurosurgery,* **23**(2), 162-168.

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