Introduction: Tumor size and location have been supposed as important factors that influence the vertebral body collapse from clinical evaluations. Concerning with the tumor size, some experimental studies have demonstrated its effect on the failure strength of the spine. However, the role of the tumor location has evaluated in few biomechanical reports. Recognition of probability of pathological fracture should be indispensable for decision of treatment to the spinal metastases. Therefore, to know the biomechanical effect of the tumor location on the load-bearing capacity of the vertebrae may provide invaluable information for the optimum treatment. We hypothesized that tumor location should affect the load-bearing capacity of the thoracic spine, in addition to the tumor size. The purpose of this study was to evaluate the biomechanical effects of the tumor size and location (costovertebral joints, pedicles and facet joints) on the failure load of the thoracic vertebrae by using a sheep model simulating osteolytic metastases.

Methods: A total of 111 specimens obtained from sheep thoracic spine were used. Each specimen consisted of three vertebrae (T7–9, T10–12) with intervertebral discs and ribs. The trabecular defects simulating the osteolytic metastases were created within vertebral bodies (T8 and T11). 51 specimens with various size of the vertebral defect were subjected to destructive testing, following determination of defect ratio (defect area / body area*100%, in cross sectional area). This study consisted of following two sections. Study 1: The relation between failure load and defect size within vertebral body. 51 specimens with various defect sizes within vertebral body (range of defect ratio = 0-74%) were subjected to destructive testing. In all specimens, any other parts of the vertebrae remained intact. Study 2: The biomechanical effects of the tumor locations. To evaluate the effect of tumor location, some patterns of metastatic involvement were simulated. 12 intact specimens were used as normal control (Group 1), 60 specimens involving the constant defect ratio (42.1±5.8%), and 6 specimens with various defect ratio (42.1±5.8%) were divided into five groups (n=12) according to the location of the additional destruction simulating metastatic lesion. No additional destruction except trabecular defect within vertebral body (Group 2). Additional destruction of the right costovertebral joint (Group 3), the additional right pedicle resection (Group 4), the additional right pedicle and costovertebral joint destruction (Group 5) and, the additional right facet joint, pedicle and costovertebral joint destruction (Group 6). In both studies, the upper and lower vertebrae were embedded in polyester resin, and load-to-failure test was performed on a hydraulic testing apparatus (MTS, Model 858 Bionix). The initial load was 50N, and an eccentric load was applied vertically to the anterior one-third of the vertebral body in the mid-sagittal plane at a loading rate of 50 N/sec, and the failure load was determined. One-way ANOVA and Fisher PLSD were used for statistical analysis.

Results: Study 1: A significant negative linear correlation between defect ratio and failure load was obtained (Figure 1). The equation for the regression line was as follows: Failure load (N) = 7367.6 – 93.623 * defect ratio (%); r2 = 0.782

Study 2: Group 1 showed a significantly higher failure load than the other groups (p < 0.05). Endplate fracture was observed in all specimens belonged to group 2 to 6 in radiographic evaluation. Although the defect size within vertebral body was constant, significant reduction of the failure load was observed by additional costovertebral joint destruction (Group 2 vs. 3, Group 4 vs. 5; p < 0.05). Destruction of the unilateral pedicle or facet joint did not affect the failure load significantly (Group 2 vs. 4, group 3 vs. 5, and Group 5 vs. 6) (Figure 2).

Discussion: Agreed with previous reports, defect size localized in the vertebral body was proportional to the vertebral failure load. And a significant effect of the tumor location on the load-bearing capacity of the thoracic vertebrae was proved. Result of this study revealed that the costovertebral joint share a considerable amount of a compressive load applied to the thoracic spine. The articulations between rib and vertebrae are independent from those between endplate and intervertebral disc. Therefore, destruction of the costovertebral joint may bring about a stress concentration on the vertebra endplate (Figure 3). It may lead endplate fracture that reported as the beginning of vertebral body collapse. In conclusion, the load-bearing capacity of the metastatic thoracic vertebrae is dependent on both defect sizes within vertebral body, and existence of the costovertebral joint destruction. An important role of the costovertebral joint in the metastatic collapse of the thoracic vertebra was demonstrated. If the costovertebral joint is invaded by metastatic tumor with involvement of vertebral body in moderate size, the vertebra is potentially unstable against the destructive force, therefore surgical stabilization should be indicated.