INTRODUCTION:
Recurrent disc herniation and progression of disc degeneration were the major concern of surgical failure of discectomy. Numerous annular injury models showed that interruption of the annular fibers would lead to failure of biomechanical environment of the intervertebral disc and eventual marked progressive degeneration of nucleus pulposus with time. Although many authors indicated that primary healing of annulus fibrosis was incapable, Cauthen demonstrated that annular repair after discectomy would diminish the risk of recurrent disc herniation. Ahlgren et al. and Yoon et al. showed that minimizing injury to the annulus fibrosis may minimize the severity of disc degeneration after discectomy. The purpose of this study was to investigate whether annular repair can slow down the degenerative process in the porcine annular injury model of disc degeneration.

MATERIALS AND METHODS:
Twelve miniature pigs were used in this study. All methods and procedures were peer reviewed and approved by the animal care committee of Yang-Ming University. After anesthesia, a full thickness transverse incision, deep into nucleus pulposus (4mm length, 12mm deep), was made in the anterior annulus fibrosis of the cervical disc. Then the annular repair was randomized performed as (1) Group 1: leave the incision open (2) Group 2: securely seal the annular defect by a modified purse-string suture with crossing over compression. The intact intervertebral discs were used as controls group. Magnetic resonance imaging grading were recorded preoperatively, and at 2, 4 and 6 months postoperatively, before sacrificed. The isolated annulus fibrosis of lesioned disc was evaluated histologically by a polarizing light microscope for examining healing status. Using a reverse transcription-polymerase chain reaction, mRNA expression of aggrecan, collagen type I, II and matrix metalloproteinase-13 (MMP-13) were investigated in the isolated nucleus pulposus specimens.

RESULTS:
On MR imaging evaluation, the grading of the repaired disc was slightly worse than the control group at each time point. Sequential and progressive signal intensity decrease was observed in the non-repaired disc. All gene expression of experimented disc indicated disc degeneration from the time of injury to the final time point. Collagen type II was much higher in repaired disc than in non-repaired one. This indicated that more nucleus pulposus material was kept within the disc and the degenerative process was slow down. The inflammatory mediator of experimented discs was extremely higher post-operatively, especially in sutured group. When the time went by, the value of inflammatory mediator was decreased, but still high in non-repaired disc. Histologic examination of annular repair region indicated matrix reorganization in outer annular lamellae at 2 months after annular injury. By 6 months, the annular defect was healed.

DISCUSSION:
Based on MR imaging, morphologic findings and gene expression results, this study showed that suture sealing of the annular defect could preserve the integrity of the intervertebral disc and then decelerate the progression of disc degeneration. Diminish of the annular defect area by suture may promote active repair process in the annular injury area. Inflammatory reaction after annular injury may persist for a long time if no annular repair was done. This finding was promising that if securely annular repair after discectomy could be effective to prevent early disc degeneration.

REFERENCES:
3. Masuda K et al. Spine 2005; 30:5-14