**Introduction:** Autophagy is a bulk degradation of subcellular constituents, and is activated in several neurodegenerative conditions [1]. Under stress conditions, the autophagic process can lead to non-apoptotic programmed cell death (type II programmed cell death) [2]. Beclin 1, a Bcl2 interacting protein, is known as a promoter of autophagy [3]. Previous research demonstrated that the expression of Beclin 1 is increased in lesions after traumatic brain injury [4]. However, to our knowledge, there has been no study that investigated the expression of Beclin 1 which indicates autophagy after spinal cord injury. In the present study, we used a spinal cord hemisection model in mice to study the alterations in Beclin 1 expression and the involvement of autophagy after spinal cord injury.

**Materials and Methods:** Animals Female C57BL/6j mice between 8 and 10 weeks of age were used. **Surgical procedures** The T10 vertebrae were laminectomized to expose the spinal cord. With a scalpel, the cord was transected on the right side only [5]. **Immunohistochemistry** At different time points (4, 24h, 3d, 7d, 21d) after hemisection, the spinal cords were removed, and the injured and the contralateral sides of the cords were homogenized in lysis buffer. Lysates were resolved by SDS-PAGE gels and transferred to a polyvinylidene difluoride membrane. To assess the level of Beclin 1 protein, immunoreactive bands were detected using rabbit anti-Beclin 1 antibody (1:300; Santa Cruz Biotechnology) and visualized by Alexa Fluor 555 goat anti-rabbit IgG antibody. **Counting of Beclin1 positive cells** The numbers of Beclin1 positive cells were counted in the injured and the contralateral sides of transverse sections at different time points (4, 24h, 3d, 7d, 21d) after hemisection, and compared with those of the sham control. **Western blot analysis** Mice were killed at 3 days after hemisection. Tissue sections were incubated with rabbit anti-Beclin1 antibody (1:300; Santa Cruz Biotechnology) and visualized by Alexa Fluor 555 goat anti-rabbit IgG antibody. **Double staining for Beclin1 and various cell type markers** To examine the expression of Beclin 1 in a specific population of cells, the transverse sections at 3 days after hemisection were co-stained for Beclin 1 and various cell type markers: NeuN for neurons, GFAP for astrocytes and Olig2 for oligodendrocytes.

**Results:** **Counting of Beclin1 positive cells** The numbers of Beclin1 positive cells on the injured side were significantly higher than those on the contralateral side and sham control at each time point (Fig. 1). The maximum number of Beclin1 positive cells in the injured side was observed at 3 days, and it decreased after 7 days (n = 3 at each time point) (Fig. 2).

**Western blot analysis** The level of Beclin 1 protein was significantly higher on the injured side (1.6 ± 0.31-fold increased, p = 0.009) than on the contralateral side (n = 3) (Fig. 2).

**Double staining for Beclin1 and various cell type markers** Double staining of Beclin 1 and various cell type markers demonstrated the expression of Beclin 1 in neurons, astrocytes and oligodendrocytes at 3 days after hemisection.

**Discussion:** In the present study, we have demonstrated that levels of Beclin 1 protein were dramatically increased after spinal cord hemisection. The expression of Beclin 1 was observed in neurons, astrocytes, and oligodendrocytes. These results showed that autophagy is activated in the spinal cord response to traumatic injury. Under stress conditions, the autophagic process can lead to non-apoptotic programmed cell death and also regulate other types of cell death [2]. In the present study, the expression of Beclin 1 was increased at the injured site and reached a peak at 3 days after hemisection. The time course of the expression of Beclin 1 was similar to that of apoptosis after spinal cord injury [6]. This finding suggests that the expression of Beclin 1 may be related to apoptosis after spinal cord injury.

**References:**