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### Changes of mitochondrial autophagy - related genes and autophagosome after skeletal muscle blunt trauma

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**Objective** Objective: To study the changes of mitochondrial autophagy-related genes and autophagosome after skeletal muscle blunt trauma, to reveal the changes of mitochondrial adaptive repair process after skeletal muscle blunt trauma, and to elucidate the mechanism of blunt trauma repair process.

**Methods** Methods: Sixty - four male Wistar rats were randomly divided into control group and blunt trauma group (divided into 12h group, 2d group, 5d group, 7d group, 10d group, 15d group and 30d group) according to the time of extraction. The expression of HIF-1 $\alpha$ , AMPK $\alpha$ 2, BNIP3 and NIX protein in skeletal muscle hypoxia and autophagy-related factors were measured by Western-Blot. QRT-PCR was performed to analyze the expression levels of HIF-1 $\alpha$ , AMPK $\alpha$ 2, BNIP3 and NIX. The ultrastructure and autophagic formation at different time points were observed by transmission electron microscopy (TEM).

**Results** Results: The expression of HIF-1 $\alpha$  and AMPK $\alpha$ 2 protein reached the peak at 12h and 2d, and the expression of HIF-1 $\alpha$  was significantly higher than that of the control group ( $P < 0.05$ ). The expression of AMPK $\alpha$ 2 was significantly higher at 5 days after injury ( $P < 0.05$ ), and reached the normal level at 10 days. BNIP3 began to decline after 5 days, but still higher than normal at 10 days after treatment. NIX expression peak appeared at 12h and 2d after injury, with high-express to 7d. The expression of HIF-1 $\alpha$  and AMPK $\alpha$ 2 mRNA was significantly higher than that of the control group ( $P < 0.01$ ), but decreased until 5d ( $P < 0.05$ ), then decreased to normal level. The mRNA expression of BNIP3 and NIX was basically the same as their protein performance. A number of autophagosomes were observed at 12 h after injury, and the number of autophagosomes increased gradually at 2-7 d. After 10 days, the number of autophagosomes decreased compared with that of 12 h-7 d after blunt. And after 15 days, the number of autophagosomes decreased gradually.

**Conclusions** Conclusion: The changes of early stage metabolic regulator AMPK $\alpha$ 2 and hypoxia-sensitive factor HIF-1 $\alpha$  after skeletal muscle blunt trauma indicated that an energy crisis occurred in the skeletal muscle after injury, and the hypoxic environment was formed. The mitochondrial autophagy, the expression of BNIP3 and NIX showed that mitochondrial autophagy was activated and hypoxia induced mitochondrial autophagy at early skeletal muscle contusion period. Hypoxia-induced mitochondrial autophagy could remove the damaged mitochondria, maintain mitochondrial quality and provide raw materials for new mitochondria generation, facilitate the rapid recovery of damaged skeletal muscle, which may be a compensatory mechanism of the body response to injury.