The effects of autophagy on neuronal damage can be positive or detrimental negative. By establishing a model of fetal rat cortical neuron hydraulic shock injury, dipotassium bisperoxo (picolinato) oxovanadate V [bpv(pic)] was used to inhibit PTEN at different time points post-injury and autophagy level after neuronal injury was assessed. Neurons were divided into several intervention groups according to the time point at which bpv(pic) was used to inhibit autophagy, normal neurons, and injured neurons were set as two control groups. The growth of neurons in each group was assessed through immunofluorescence staining. Expression of the autophagy-related proteins LC3-II and LC3-I was analyzed by western blot. Expression of PTEN, mTOR and Beclin-1 was detected by RT-PCR. The number of autophagosomes in the normal group, injury control group and 24 h, 36 h intervention groups were assessed by an electron microscope. We found that autophagy was enhanced after neuronal injury and that the levels of LC3-II was significantly reduced by bpv (pic) intervention. The growth of the injury control groups was worse than normal groups, while improved through bpv(pic) intervention at 24 h and 30 h after injured. Western blot analysis showed that the LC3-II and LC3-II/LC3-I ratios of cells increased post-injury, and autophagy induction was evident by electron microscopy. These effects were confirmed by RT-PCR analysis. Taken together, these data suggest that autophagy is activated after injury in neurons while can be inhibited by bpv(pic) administration and then promote the repair of injured neurons [1)].

1)
Yu H, Shao J, Huang R, Guan Y, Li G, Chen S, Zhou F, Yao Q, Shen J. Targeting PTEN to regulate autophagy and promote the repair of injured neurons. Brain Res Bull. 2020 Oct 10:S0361-9230(20)30665-1. doi: 10.1016/j.brainresbull.2020.10.008. Epub ahead of print. PMID: 33049350.