NLRP6

NLRP6, a member of nod-like receptors (NLRs) family, has been reported to participate in inflammation and host-defence in multiple diseases. Distinct from the other NLR family members, NLRP6 regulates inflammation in an inflammasome-dependent as well as an inflammasome-independent pathway. However, the role of NLRP6 in regulating signalling pathways during ICH is poorly understood. In the present study, we demonstrated that NLRP6 expression was upregulated after ICH, both in humans and in rats. Subsequently, we developed a rat model of ICH and found that NLRP6 knockdown reduced brain injury, alleviated inflammation, and suppressed autophagy following ICH. Further, results indicated that autophagy involved in NLRP6 mediated inflammation after ICH. Moreover, we found that NLRP6 mediated regulation of autophagy and inflammation was inflammasome-dependent. This study revealed the underlying molecular mechanism of NLRP6 in inflammation and highlights the therapeutic potential of targeting NLRP6 in secondary brain injury after ICH. KEY MESSAGES: • NLRP6 was upregulated following ICH in humans and rats. • NLRP6 knockdown reduced brain injury, alleviated inflammation, and suppressed autophagy following ICH. • NLRP6 aggravated inflammation after ICH by activating autophagy. • NLRP6 regulated inflammation and autophagy after ICH by activating inflammasome pathway [1].

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Xiao H, Chen H, Jiang R, et al. NLRP6 contributes to inflammation and brain injury following intracerebral haemorrhage by activating autophagy [published online ahead of print, 2020 Aug 12]. J Mol Med (Berl). 2020;10.1007/s00109-020-01962-3. doi:10.1007/s00109-020-01962-3