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The Role of Il-6-Mediated NS5ATP9 Expression in Autophagy of Liver Cancer Cells

Authors : Hongping Lu, Kelbinur Tursun, Yaru Li, Yu Zhang, Shunai Liu, Ming Han

Abstract : Objective: To investigate whether NS5ATP9 is involved in IL-6 mediated autophagy and the relationship between IL-6 and NS5ATP9 in liver cancer cells. Methods: 1. Detect the mRNA and protein levels of Beclin 1 after HepG2 cells were treated with or without recombinant human IL-6 protein. 2. Measure and compare of the changes of autophagy-related genes with their respective control, after IL-6 was silenced or neutralized with monoclonal antibody against human IL-6. 3. HepG2 cells were incubated with 50 ng/ml of IL-6 in the presence or absence of PDTC. The expression of NS5ATP9 was analyzed by Western blot after 48 h. 4. After NS5ATP9-silenced HepG2 cells had been treated with 50 ng/ml recombinant IL-6 protein, we detected the Beclin 1 and LC3B (LC3Ⅱ/I) expression. 5. HepG2 cells were transfected with pNS5ATP9, si-NS5ATP9, and their respective control. Total RNA was isolated from cells and analyzed for IL-6. 6. Silence or neutralization of IL-6 in HepG2 cells which has been transfected with NS5ATP9. Beclin 1 and LC3 protein levels were analyzed by Western blot. Result: 1. After HepG2 were treated with recombinant human IL-6 protein, the expression of endogenous Beclin 1 was up-regulated at mRNA and protein level, and the conversion of endogenous LC3-I to LC3-II was also increased. These results indicated that IL-6 could induce autophagy. 2. When HepG2 cells were treated with IL-6 siRNA or monoclonal antibody against human IL-6, the expression of autophagy-related genes were decreased. 3. Exogenous human IL-6 recombinant protein up-regulated NS5ATP9 via NF-κB activation. 4. The expression of Beclin 1 and LC3B was down-regulated after IL-6 treated NS5ATP9-silenced HepG2 cells. 5. NS5ATP9 could reverse regulates IL-6 expression in HepG2 cells. 6. Silence or neutralization of IL-6 attenuates NS5ATP9-induced autophagy slightly. Conclusion: Our results implied that in HCC patients, maybe the higher level of IL-6 in the serum promoted the expression of NS5ATP9 and induced autophagy in cancer cells. And the over-expression of NS5ATP9 which induced by IL-6, in turn, increased IL-6 expression, further, promotes the IL-6/NS5ATP9-mediated autophagy and affects the progression of tumor. Therefore, NS5ATP9 silence might be a potential target for HCC therapy.

Keywords : autophagy, Hepatocellular carcinoma, IL-6, microenvironment, NS5ATP9
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