

## IL-17 induces the proliferation and migration of glioma cells through the activation of PI3K/Akt1/NF- $\kappa$ B-p65

Wen Qiu

Nanjing Medical University, China

### Abstract

Interleukin 17 (IL-17), as a pro-inflammatory cytokine, is up-regulated in the sera and tumor tissues of glioma patients; however the effects of IL-17 on glioma proliferation and migration remain unclear. In this study, the roles of IL-17 in the proliferation and migration of glioma cells and their potential mechanisms were determined. The results showed that IL-17 could not only enhance the proliferation and migration of cultured glioma cells (in vitro), but also promote the tumor formation of glioma cells in BALB/c nude mice (in vivo). Mechanical exploration revealed that IL-17 stimulation could increase the phosphorylation levels of Akt1 and NF- $\kappa$ B-p65 in glioma cells, and knockdown or inhibition of PI3K, Akt1 and NF- $\kappa$ B-p65 could also reduce the IL-17-induced proliferation and migration of the glioma cells. Moreover, PI3K/Akt1 was the upstream regulator of NF- $\kappa$ B-p65 activation in IL-17-incubated glioma cells. Furthermore, the inhibition of PI3K, Akt1 and NF- $\kappa$ B-p65 markedly suppressed the tumor formation of glioma cells induced by IL-17. Together, these data indicate that IL-17 can promote the proliferation and migration of glioma cells via PI3K/Akt1/NF- $\kappa$ B-p65 activation, and these findings might provide a new insight into glioma pathogenesis.

### Biography

Wen Qiu is currently an associate professor at Department of Immunology of Nanjing Medical University. He is exploring roles and mechanisms of inflammatory factors in glioma cell proliferation and migration. These include signal transduction, microRNA regulation, transcriptional factor regulation. He is also exploring the effects of post-transcriptional regulation such as ubiquitination and acetylation on the activation of signaling molecules and transcription factors

**Citation:** Wen Qiu | IL-17 induces the proliferation and migration of glioma cells through the activation of PI3K/Akt1/NF- $\kappa$ B-p65  
| Journal of Molecular imaging and Dynamics(JMID)