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Effect of IL-17 monoclonal antibody Secukinumab combined with IL-35 blockade of Notch signaling pathway on the invasive capability of hepatoma cells

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ABSTRACT. We investigated the effect of the IL-17 monoclonal antibody Secukinumab combined with IL-35 in the blockade of the Notch signaling pathway on the invasive capability of hepatoma cells. We examined the effects of IL-17 antibody or IL-35 treatment alone or in combination on cell invasion and migration capabilities with Transwell chambers. The mRNA levels of Hes1, Hes5, and Hey1 were tested using quantitative polymerase chain reaction. The protein expression of N1ICD, Snail, and E-cadherin protein expressions were measured with western blot. The expression of Hes1, Hes5, Hey1 and N1ICD were all very high in hepatoma cell lines, and were positively correlated with the invasive migration capabilities of the cells. The combination of IL-17 monoclonal antibody Secukinumab with IL-35 could effectively inhibit the Notch signaling pathway, as well as the invasive migration of the cells. Snail and E-cadherin are involved in the migration of hepatoma

cells, and it has been established that Snail can regulate the expression of E-cadherin. IL-17 monoclonal antibody Secukinumab combined with IL-35 can increase E-cadherin and decrease Snail expression, which are positively correlated with cell invasive migration capabilities. Overall, treatment with both IL-17 antibody and IL-35 is more effective than each treatment alone. Notch signaling is activated in hepatoma cell lines and increases with the enhancement of cell invasive migration capabilities. IL-17 monoclonal antibody Secukinumab combined with IL-35 can block the Notch signaling pathway, simultaneously reducing the invasive migration capability of hepatoma cells.

Key words: IL-17 monoclonal antibody; Secukinumab; IL-35 gene; Hepatoma; Invasive migration; Notch signaling pathway