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Role of Interleukin 6 (IL-6) on physical response and inflammatory disease

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Development of autoimmune disease can be divided into two phases, one is an immunological initial phase, and the other is a chronic inflammatory phase. IL-6 contributes to the pathogeneses at both phases. Since IL-6 induces the development of Th17 cells from naïve T cells and activates to the auto-reactive T cells in vitro. This phenomenon was demonstrated in autoimmune model mice. Treatment with an IL-6 specific inhibitor improved symptom with the reduction of Th17cells and inactivation of auto-reactive T cells. On the other, IL-6 develops inflammatory status which is late phase of autoimmune disease. IL-6 blocking therapy with an anti-IL-6 receptor antibody (Tocilizumab) for rheumatoid arthritis (RA) and juvenile idiopathic arthritis (JIA), improved the clinical symptoms and laboratory findings, especially CRP and SAA levels were normalized, even if another cytokines and chemokines were activated in the patients. Therefore it was analyzed this phenomenon in vitro utilizing hepatoma derived cell lines. In result, activation of STAT3 through IL-6 signal was essential for induction and augmentation of CRP and SAA expression via assembling the transcription factors complex with NF-kBp65 and with HNF-1α and c-fos, respectively. TNF-α or IL-1 complementally contributed the augmentation of CRP and SAA through the activation of NF-kBp65 and c-fos, respectively.

Biography

Kazuyuki Yoshizaki found BCDF (1981), former Interleukin 6 (IL-6) in 1986. He had analyzed IL-6 in the pathogenesis of immunological disorders, such as Castleman's disease (CD) in 1989 and rheumatoid arthritis (RA) in 1990. After humanized anti-IL-6 receptor antibody (named tocilizumab) in 1992, he engaged in the establishment of IL-6 blocking therapy with tocilizumab for CD, RA and juvenile idiopathic arthritis (sJIA) from 1993.

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