Cytokine receptor targeted therapies

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The cytokine profile of rheumatic diseases is a complex and interactive network. Novel and creative therapeutic strategies based on it is steadily expanding. This is especially true in rheumatoid arthritis (RA), where the cytokine milieu of the joint is well understood. Therapies intended to offset the destructive effects of proinflammatory cytokines including tumor necrosis factor TNF-α, interleukin-1 (IL-1)-β, or IL-6 in RA have proved to be effective recently both *in vitro* and *in vivo*. These include etanercept, infliximab, adalimumab, anakinra and atlizumab. Although such agents have met with success in the treatment of RA, antibodies or chimeric proteins targeting at small molecules on the cell surface or receptors that result in blocking cytokine production or apoptosis of the immunocompetent cells *per se* will likely have a competitive advantage in the coming years. Indeed, some of these molecules have emerged and have been proved equally effective as cytokine inhibitor. A representative one of this group of biologics is rituximab, which is designed to antagonize CD20 molecule on the surface of B cells. In the near future, a deluge of such novel “receptor antagonists” are expected to be invented. However, an important note of caution is the possibility that suppressing even a physiologic level of cytokines or immune cells could have serious harmful consequences on immune surveillance. This is the intrinsic and inevitable drawback of target therapies in the era of biologics.