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Interleukin-1 (IL-1) is a major proinflammatory cytokine produced by mononuclear phagocytes. IL-1 is a key mediator of acute inflammation. Its effects are mediated by specific IL-1 receptors that can be either membrane bound (mIL-1R) or secreted (sIL-1R) forms. Both receptor forms signal through a common receptor-associated protein known as IL-1RAP and a member of the Toll-like receptor (TLR) family, MyD88. Two mIL-1R (IL-1R1 and IL-1R2) are known to exist, and these can occur as both homo- and heterodimers, as well as heterooligomeric complexes. The sIL-1R (IL-1R3) is thought to lack signal transduction capability and acts as a decoy receptor, competing for IL-1 binding with cell surface mIL-1R. IL-1R3 is primarily expressed in hematopoietic cells, particularly T cells. IL-1R1 is one of the first receptors to be identified and is a key mediator of IL-1-induced effects. Animal studies have demonstrated that disruption of the IL-1R1 gene is associated with the development of a spontaneous inflammatory disease in mice. In humans, several polymorphisms have been identified in the coding sequence of IL-1R1 that are associated with an increased risk of developing inflammatory bowel disease (IBD). Additional evidence supporting a role for IL-1R1 in human disease comes from the observation of a high prevalence of anti-IL-1R1 autoantib



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