Abstract

Autoimmune and lymphoproliferative diseases share some etiologic mechanisms. The origin of the diseases is complicated process that involves an accumulation of hereditary and somatic mutations in a hematopoietic cell, which thanks to changed activity overcomes different growth and survival control checkpoints. Such mutations are for example those located in genes coding for transcription factors, apoptotic signaling molecules, costimulatory molecules and secreted extracellular molecules. All these molecules influence the balance between survival and programmed cell death. Their dysregulated expression enables the cell to overcome defensive mechanisms of the immune system. Therefore, autoimmune and malignant cells are able to survive though, under usual circumstances, they would be selected. The main aim of this work is to shed the light on the influence of the dysregulated expression of the particular molecules on the origin of autoimmune and lymphoproliferative diseases.

Key words: autoimmune illnesses, lymphoproliferative diseases, etiology, AIRE, c-MYC, TP53, FOXP3, Fas, PTEN, Bim, CTLA-4, CD5, CD30, CD40/CD40L, BAFF, α-taxilin, IL-10.