Autoimmune hemolytic anemia (AIHA) is an immune-mediated disorder characterized by the reduced lifespan of red blood cells (RBCs) due to enhanced intravascular and extravascular destruction. Traditionally, the immunopathogenesis of AIHA has been considered in the context of the immunological tolerance breakdown of B cells, since the autoantibodies are the main disease mediators. However, more recent data suggest that the production of anti-RBC antibodies by B cells is only an epiphenomenon and that the tolerance breakdown in the CD4+ T cell compartment is a key point in early AIHA development. In AIHA, there are numerical and functional alterations of the essential CD4+ T cell subpopulations, including Th1, Th2, Th17, regulatory T cells and follicular helper T cells. In this review, the main characteristics of the cellular immune response during the development of AIHA, as well as the potential mechanisms by which CD4+ T cells promote the initiation and maintenance of the autoimmune process, are summarized. Identification of these characteristics and mechanisms would be of practical importance in the therapeutic sense because it opens up the possibility of designing more specific immunotherapy that is still not available for AIHA patients.

**Key words**: autoimmune hemolytic anemia, red blood cells, Th1 cells, Th2 cells, Th17 cells, Treg cells, Tfh cells