Results The dysfunction of apoptosis may be a direct consequence of alterations in genes such as Fas, Bcl-2 and C1q. Increased expression of Fas antigen could intensify the exposure of hidden antigens. The overexpression of Bcl-2 protein might inhibit the removal of auto-reactive cells, and the lack of C1q could impair the clearance of self-antigens. Increased apoptosis of lymphocytes especially regulatory T cells is also an important reason to lead to breakdown of immunotolerance.

Conclusions The complete knowledge of the role of apoptosis components in the etiopathogenesis of lupus could lead to the development of new therapies targeting the apoptotic threshold, which could result in a more specific and effective disease response compared to global immunosuppression.