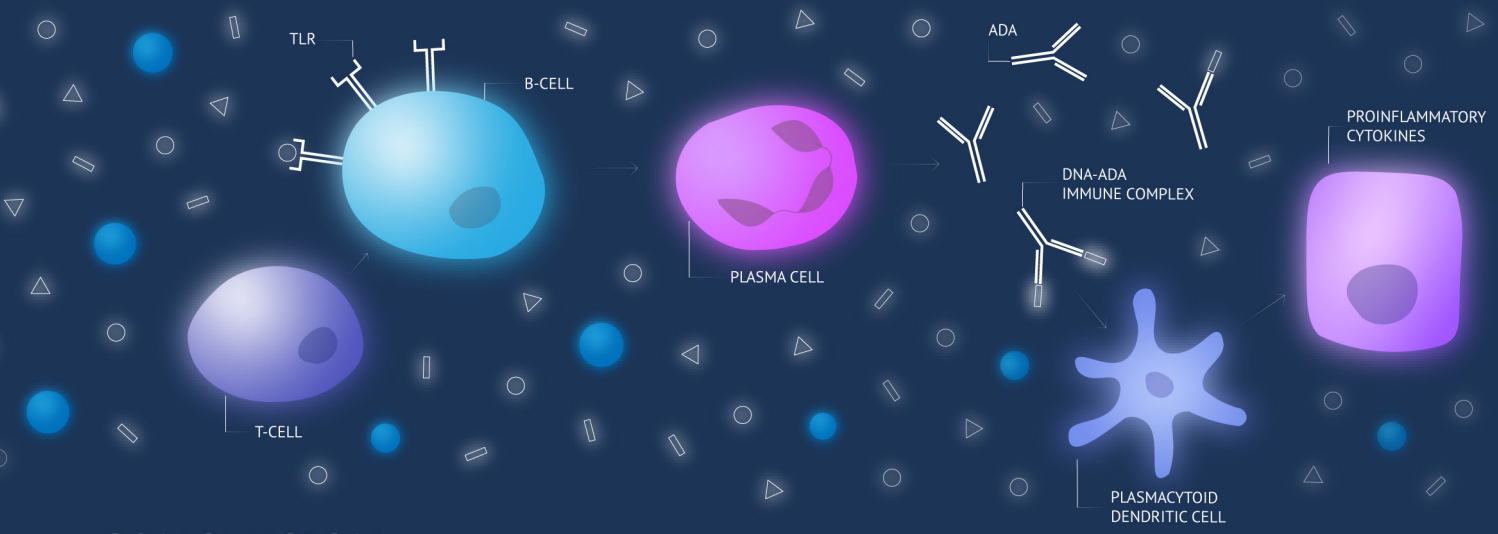
CLINICAL IMPLICATIONS

Due to the broad scope of SLE pathogenesis, including defective apoptotic clearance, extracellular nucleic acid recognition, and lymphocyte signalling, there are several possibilities for targeted immunotherapy.[6] Potential treatment options include the targeting of dendritic cells, inefficient macrophages, and deficiencies in components of the complement system.[6,16,17] Natural IgM is another attractive target to prevent the development of autoimmunity. Mouse models of lupus have demonstrated that a deficiency of serum IgM leads to a dual increase in the spontaneous development of IgG anti-DNA autoantibodies, as well as renal deposition of IgG and complement proteins.[18,19] In contrast, in mice lacking hypermutated IgG antibodies, elevation of IgM levels and subsequent complement activation increased clearance of apoptotic cell debris, enhancing survival rates relative to mice lacking B cells or secreted antibodies.[20] Further research may help to delineate the potential effects of natural IgM in reducing the severity of SLE pathophysiology.



CONCLUSION

Recent reviews demonstrate that mortality rates continue to increase in patients with SLE, with medical and productivity costs remaining high despite improvements made in available treatments.[8,21-23] Due to its clinical heterogeneity and unclear etiology, which involves complex gene-environment interactions, SLE requires the development of novel therapeutic treatments in order to improve the health and quality of life of affected individuals.[24]

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EDITED BY SARAH ASBURY & KEVIN ZHAO

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