The presentation focuses on the role of complement C1q, the first component of the classical pathway, and antibodies against C1q (anti-C1q) in Systemic Lupus Erythematosus (SLE). SLE is the archetype of an autoimmune disease and can involve any organ system eventually leading to comorbidities that can also be observed independently of underlying SLE. The complex pathogenic mechanisms leading to and being involved in this autoimmune-inflammatory syndrome are not well understood, but C1q seems to play a central role. Analysing the role of C1q as well as its interaction with autoantibodies (anti-C1q) in SLE helps elucidating mechanisms being involved in the development of the autoimmune reaction, leading to secondary acceleration of inflammation, and directing future targeted treatment strategies.