# Immunoregulation and Autoantibody Production in Primary Thrombotic Antiphospholipid Syndrome

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## Abstract

Antiphospholipid syndrome (APS) is an autoimmune disease characterized by recurrent thrombosis in the presence of antibodies targeting protein-binding phospholipids – antiphospholipid antibodies (aPL). T follicular helper (Tfh) cells are likely implicated in APS since most pathogenic autoantibodies are high-affinity isotype-switched IgG. APS offers the opportunity to address the relationship between Tfh subsets and autoantibody production in an autoimmune disease not subjected to immunosuppressants, as treatment is restricted to anticoagulation. We recruited a cohort of APS patients and age and sex-matched healthy donors to address a putative quantitative and/or qualitative dysregulation of circulating T cells subsets (Tfh, CD4+CXCR5+; activated Tfh, CD4+CXCR5+PD-1+ICOS+; Tfh1-like, CXCR3+CCR6-; Tfh2-like, CXCR3-CCR6-; Tfh17-like, CXCR3-CCR6+; and T follicular regulatory cells, CD4CXCR5+FoxP3+) leading to the production of aPL and establish a putative correlation between aPL titers and Tfh subsets within the APS group. Our results show a correlation between the aPL titters and the Tfh cells subsets within the APS group as well as a distinct patters of Tfh cells subsets distribution between APS patients and healthy donors.