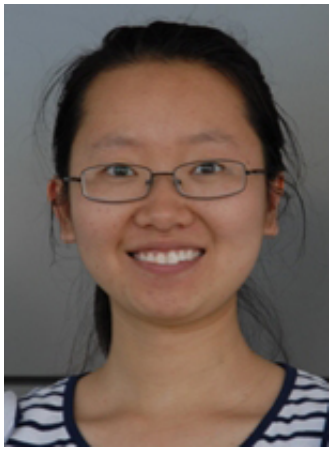


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Complement regulation in autoimmune disease

The complement system is a crucial part of innate immunity. It protects host cells from microbial infection, eliminates modified self cells such as apoptotic cells and comprises a significant link between the innate and adaptive immune system. However, this process needs to be strictly controlled. Any imbalance in complement regulation can result in tissue injury and autoimmune diseases such as hemolytic uremic syndrome (HUS), membranoproliferative glomerulonephritis (MPGN) or age related macular degeneration (AMD). Complement regulators control the complement cascade and defective regulation can result in autoimmune diseases. Thus study on complement regulators are important for both autoimmune disease pathology research and therapies and to understand the response to infections.

My project focus on complement regulators CFHRs and in particular I want to investigate how genetic and sequence as well as posttranslational modification and corresponding gene aberrations cause defective recognition of infectious microbes and autoimmune diseases. My PhD project therefore aims to provide update information for autoimmune diseases and complement regulation mechanism.

Publications

Zhao F, Afonso S, Lindner S, Hartmann A, Löschmann I, Nilsson B, Ekdahl KN, Weber LT, Habbig S, Schalk G, Kirschfink M, Zipfel PF, Skerka C (2019) C3-Glomerulopathy Autoantibodies Mediate Distinct Effects on Complement C3- and C5-Convertases. *Front Immunol* 10, 1030. Details PubMed

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Doctoral Disputation

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