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Functional characterization of the soluble complement receptor type II (CR2/CD21) in infection

Upon microbial infection the human immune system reacts immediately via innate immune reactions followed by the induction of the adaptive immune response. Complement is the first line of defense in innate immunity and plays a crucial role in recognition and elimination of infectious microbes. Complement activation leads to the opsonization of microbial surfaces by C3b, to inflammation due to the generation of anaphylatoxins C3a and C5a, and in particular to lysis of gram-negative bacteria via the formation of the terminal complement complex (TCC). Complement opsonized microbes further induce inflammatory reactions of immune cells such as B and T cells and thereby link the innate with the adaptive immune reactions. The C3 activation product C3b, as well as its further processed products iC3b and C3dg found on opsonized microbes are recognized by the complement receptor type II (CR2/CD21) which is part of the co-receptor complex on B cells. By binding of opsonized microbes through CR2, the co-receptor complex and the B cell receptor (BCR) can cross-link which substantially reduces antigenic threshold necessary for B cell activation. However, activated B cells also shed their CR2 receptors as a soluble form of the receptor (sCR2) in the infection process and the functions of these soluble receptors are unclear. As sCR2 maintains its ability to bind C3 activation products we hypothesize that sCR2 either further enhances immune reactions by recruiting inflammatory cells, or that the sCR2 terminates B cell activation which is of interest for evasion of pathogens. The aim of my PhD thesis is the functional characterization of sCR2 in context of infection processes.

Publications

Eberhardt HU, Buhlmann D, Hortschansky P, Chen Q, Böhm S, Kemper MJ, Wallich R, Hartmann A, Hallström T, Zipfel PF, Skerka C (2013) Human factor H-related protein 2 (CFHR2) regulates complement activation. *PLoS One* 8(11), e78617. [Details](#) [PubMed](#)

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