Passing a strong message: the IgA Fc receptor in health and disease

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2020

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In this thesis I focused on the interactions between IgA and FcαRI. Although IgA has been mainly regarded as a non- or anti-inflammatory antibody, our previous work has shown that IgA can also potently activate immune cells. While pro-inflammatory immune responses are essential for (mucosal) protection, in IgA-autoantibody mediated diseases these pro-inflammatory immune responses can contribute to tissue damage. The severity of these IgA-mediated diseases start to unravel how powerful the inflammatory antibody IgA can be under certain circumstances, and how much injury this can cause. Therefore, it is of utmost importance to understand how IgA is capable of initiating such potent immune responses, and to explore the possibility of blocking IgA-FcαRI interactions in IgA-mediated autoimmune diseases.