Anti-citrullinated protein antibody B cells in rheumatoid arthritis: from disease-driving suspects to therapeutic targets

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Stellingen behorend bij het proefschrift getiteld:

Anti-citrullinated protein antibody B cells in rheumatoid arthritis: from disease-driving suspects to therapeutic targets

1 The formation of an anti-citrullinated protein antibody (ACPA)-response does not necessarily rely on somatic hypermutation and can result from germline-encoded autoreactive B cells. (this thesis)

2 EBV infection of B cells is by itself not involved in the rise of ACPA-expressing B cells and their escape from immune tolerance mechanisms. (this thesis)

3 Circulating ACPA-expressing memory B cells, compared to anti-tetanus toxoid-expressing memory B cells, exhibit elevated kinase phosphorylation, likely reflecting recent antigen encounter. (this thesis)

4 Refining therapeutic strategies to selectively target antigen-specific B cells is crucial for developing more effective and potentially curative treatments for chronic B cell-mediated autoimmune diseases while minimizing effects on non-target cells. (this thesis)

5 Anti-C1q-CCP4 bispecific complement engagers have the potential to eradicate autoreactive ACPA-expressing B cells in a specific manner without affecting non-autoreactive bystander B cells. (this thesis)

6 Benefit of antibody breadth for antigen reactivity comes at a cost of self-reactivity.

7 Tailoring therapy to disease-relevant B cell populations may shift the treatment of autoimmune diseases from broad immunosuppression to precise, selective targeting with minimal adverse effects.

8 Failure to detect is not proof of absence.

9 Science requires precision, but pragmatism makes it truly effective.

10 Interdisciplinary collaboration transforms isolated expertise into a platform for innovative solutions that no single discipline could achieve alone.

11 A thesis and a child both require love, patience, and perseverance, entail sleepless nights, yet make every effort worthwhile.