The Role of B Cell Lipidome in Immune Homeostasis

Dr. Andy Tan
Bioprocessing Technology Institute (BTI),
Agency for Science, Technology and Research (A*STAR)

Abstract
The immune system protects the host from infection by pathogens. It does this through the action of multiple immune cell types, for example, invariant natural killer T (iNKT) cells that specifically recognize lipid antigens and B cells. iNKT cells provide help to B cells to promote production of antibodies that neutralize the pathogen. When the immune response is directed against molecules expressed by self, iNKT cells also modulate production by B cells of autoantibodies that result in damage to host tissues. What is not as clearly understood is how B cells reciprocally regulate iNKT cell responses, which can occur via presentation of self-lipid antigens by B cells to activate iNKT cells. To address this question, we studied mice which harbour a B cell-specific loss-of-function mutation in the FAS receptor, expression of which is important for immune, including B cell, homeostasis. One striking aspect of the progressive autoimmunity which mutant mice developed with age was severe deficiency of iNKT cells. I will discuss how and why this happens and describe alterations in the lipid metabolism of autoimmune B cells that possibly contribute to the iNKT phenotype. Taken together, findings from our study unveil a critical link between the B cell lipidome and the maintenance of iNKT cells, and are consistent with the emerging notion that B cells have essential immunomodulatory functions beyond antibody production. In conclusion, I will also review novel therapeutic strategies that target dysregulated lipid biosynthesis pathways in the treatment of autoimmune diseases.

Selected Publications for Reference


* Equal first author; ** Co-senior author; *** Corresponding author.

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