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Mysm1 epigenetically regulates B1 cell proliferation via targeting miR150

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B1 cells are the dominant population of B cells in the pleural and peritoneal cavities. They are a significant source of serum antibody, and they make a dominant contribution to low-affinity IgM antibodies that are present in serum of unimmunized mice. B1 cells in the mouse are thought to be derived from precursors in fetal liver rather than from adult bone marrow. They are believed to maintain their cell numbers in adult mice by longevity and homeostatic proliferation. Unlike conventional B cells (B2 cells) and despite the importance of B1 cells in protection from infections and their association with autoimmunity, the mechanism of B1 cell proliferation and function remained poorly understood. Mysm1 is a histone de-ubiquitinase and has been shown to play an essential role in hematopoiesis and lymphocyte development. Our previous study has demonstrated that in Mysm1 deficient mice, B2 cell development is blocked and B2 cell number is significantly lower compared with their counterpart. In this study we found that, in contrast to the dramatically decreased level of B2 cells, the percentage of B1 cells in the spleen and peritoneal cavity of Mysm1 deficient mice was increased compared with that in wild type mice. Mechanistic study has demonstrated that miR150 expression is compromised in B1 cells from Mysm1 deficient mice. Mysm1 controls the transcription of miR150 through regulating the chromatin state of miR150 locus. What's more, forced expression of miR150 in B1 cells from Mysm1 mice can partly rescue the phenotype. Overall, our study, for the first time, reveals the important role of histone H2A de-ubiquitinase in B1 cell proliferation and development.

Biography

Xiao-Xia Jiang has completed her PhD from Institute of Basic Medical Sciences. She is an Associate Professor in the Department of Advanced Interdisciplinary Studies, Institute of Basic Medical Sciences. She has published more than 40 papers in reputed journals.

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