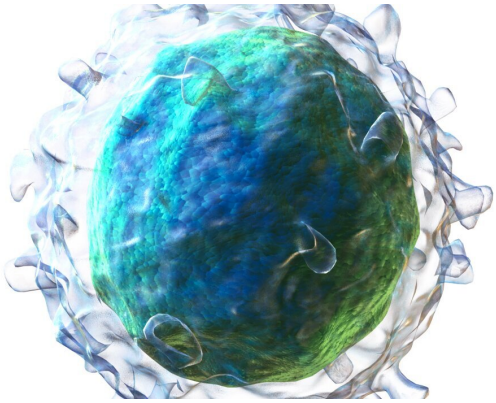
A new regulator of B cell development

11 October 2019, by Leigh MacMillan



3D rendering of a B cell. Credit: Blausen.com staff (2014). "Medical gallery of Blausen Medical 2014". WikiJournal of Medicine 1 (2). DOI:10.15347/wjm/2014.010. ISSN 2002-4436. CC BY-SA 4.0

Interleukin-33 (IL-33) drives inflammatory responses in allergic and nonallergic disease. Epithelial cells in the lungs, gastrointestinal tract and elsewhere release IL-33, which activates the ST2 receptor on immune cell targets.

In addition to the ST2-binding domain, IL-33 contains a nuclear chromatin-binding domain, suggesting that it may act inside cells that produce it. However, a cell-intrinsic role for IL-33 has not been established.

Matthew Stier, MD, Ph.D., Stokes Peebles, MD, and colleagues have now identified IL-33 expression, but not ST2 receptor expression, in developing B cells in the bone marrow (mature B cells produce antibodies). They demonstrated that IL-33 deficiency resulted in increased numbers of developing B cells.

The researchers detected IL-33 during early B cell development in humans and found reduced IL-33 expression in B cell chronic lymphocytic leukemia samples compared to healthy controls.

The findings reported in the Sept. 15 *Journal of Immunology* establish a cell-intrinsic role for IL-33 in early B cell development that is independent of the ST2 receptor.

More information: Matthew T. Stier et al. IL-33 Is a Cell-Intrinsic Regulator of Fitness during Early B Cell Development, *The Journal of Immunology* (2019). DOI: 10.4049/jimmunol.1900408

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