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Murine leukemia virus targets innate-like B1 B cells to establish infection in mice

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Background

Retroviruses are believed to efficiently spread via sites of cell-cell contact designated virological synapses. This model was developed based on *in vitro* evidence in which infected cells establish cell-cell contact with uninfected cells. Applying intravital microscopy, we were recently able to provide *in vivo* support for the existence of virological synapses within the lymph node of living mice. Visualizing cells infected with fluorescently labeled murine leukemia virus (MLV) we identified B cells that were able to form long-lived virological synapses with uninfected lymphocytes [1]. *In vivo* virological synapses were, like their *in vitro* counterpart, dependent on the expression of the viral envelope glycoprotein (Env) and characterized by a prolonged polarization of viral capsid to the cell-cell interface. B cells were among the first cells to become infected by incoming MLV. However, the specific subtype of B cells that is susceptible to MLV had remained unknown.

Results

Here we present evidence for a critical role of innate-like B1 B cells in the establishment of MLV infection in mice. Adoptively transferred B1 B cells are selectively targeted by MLV. Mice lacking B1 B cells are resistant to MLV infection. In addition, using knockout mice we provide evidence for the contribution of adhesion factors expressed by B1 B cells in spreading of retroviruses *in vivo.*

Conclusions

Our work reveals the critical importance of a distinct B cell subset in the susceptibility to retroviral infections under physiological conditions *in vivo.*

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Reference

1. Sewald X, Gonzalez DG, Haberman AM, Mothes W: ***In vivo*** **imaging of virological synapses.** *Nature Communications* 2012, **3**:1320.

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