

# B cell maintenance of subcapsular sinus macrophages protects against a fatal viral infection independent of adaptive immunity

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## Abstract

Neutralizing antibodies have been thought to be required for protection against acutely cytopathic viruses, such as the neurotropic vesicular stomatitis virus (VSV). Utilizing mice that possess B cells but lack antibodies, we show here that survival upon subcutaneous (s.c.) VSV challenge was independent of neutralizing antibody production or cell-mediated adaptive immunity. However, B cells were absolutely required to provide lymphotoxin (LT)  $\alpha 1\beta 2$ , which maintained a protective subcapsular sinus (SCS) macrophage phenotype within virus draining lymph nodes (LNs). Macrophages within the SCS of B cell-deficient LNs, or of mice that lack LT $\alpha 1\beta 2$  selectively in B cells, displayed an aberrant phenotype, failed to replicate VSV, and therefore did not produce type I interferons, which were required to prevent fatal VSV invasion of intranodal nerves. Thus, although B cells are essential for survival during VSV infection, their contribution involves the provision of innate differentiation and maintenance signals to macrophages, rather than adaptive immune mechanisms.