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West Nile virus infects B lymphocytes

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West Nile virus (WNV) is a flavivirus that is maintained in nature in an enzootic cycle between mosquitoes and birds. We have shown in mice that WNV is pantropic, except for the liver. However, the cellular tropism of WNV has yet to be completely defined. Our research objective is to understand the interaction between WNV and host immune cells. C3H/HeN (C3H) mice inoculated with WNV exhibited a B cell lymphopenia throughout the course of infection. Additionally, the B cell lymphopenia correlated with a delay in WNV-specific antibody compared to another mouse strain. We hypothesized that B cells are permissive to WNV infection, and direct infection alters these cells quantitatively and functionally. Viral growth curves were performed on WEHI cells, a cell line derived from mouse B cells, at a multiplicity of infection (MOI) equal to 1 or 10. WEHI cells produced up to 10^7 PFU WNV when inoculated at either MOI, indicative of a productive infection. Infected WEHI cells were analyzed for the presence of NS3 protein, a WNV non-structural protein, through intracellular staining and flow cytometry. At 72 hours post-infection, WEHIs were positive for NS3, confirming active viral replication. We repeated these experiments on mouse primary B cells isolated from spleens. Growth curve analysis showed that primary B cells produce up to $10^{4.5}$ and 10^6 PFU WNV when inoculated at an MOI of 1 and 10, respectively. Additionally, NS3 was detected in these cells. In summary, B cells, both primary and cell line derived, are permissive to WNV infection and productive replication. Future studies will focus on WNV infection of B cells in vivo, which may result in altered function, including the observed delay in antibody response.