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PDF Revu eXtreme 12.5.0 + Patch MPT.exe 24.57MB., including
those of the NKT and T cells, have been shown to suppress the
proliferation of virus-specific CD4⁺ and CD8⁺ T cells during
an acute viral infection ([@B1]). Although it has been suggested
that a similar immune evasion mechanism exists in HIV infection,
these experiments were performed using recombinant vaccinia
virus, which infects antigen-presenting cells (APCs) and many
other cell types. It is likely that the role of B cells in immune
evasion is unique to HIV infection and that the effector function of
B cells is not as important as in other infections. B Lymphocyte
Activation in the Absence of T Cell Help {#S4} =====
=====

== T follicular helper (T_{FH}) cells, a subset of CD4⁺ T cells
that are developmentally programmed to support B cell
responses, have been shown to be the primary source of
CCR7⁺IL-21⁺ T_{FH} cells during an acute HIV infection
([@B56], [@B57], [@B66]). However, not all B cells are generated

in the T~FH~ pathway ([@B56], [@B57]), and in the absence of a functioning T~FH~ cell response, there is a decrease in plasmablast differentiation into long-lived plasma cells, and a decrease in antigen-specific IgG and IgM plasma cell production ([@B56]). There also appears to be a shift away from isotype switching

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