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Titel des Beitrags: Primary B-cell infection with a deltaBALF4 Epstein-Barr virus comes to a halt in the endosomal compartment yet still elicits a potent CD4-positive cytotoxic T-cell response.

Abstract: Epstein-Barr virus (EBV) infection is mediated by several viral envelope glycoproteins. We have assessed gp110’s functions during the virus life cycle using a mutant that lacks BALF4 (DeltaBALF4). Exposure of various cell lines and primary cell samples of epithelial or lymphoid lineages to the DeltaBALF4 mutant failed to establish stable infections. The DeltaBALF4 virus, however, did not differ from wild-type EBV in its ability to bind and become internalized into primary B cells, in which it elicited a potent T-cell-specific immune reaction against virion constituents. These findings show that DeltaBALF4 viruses can reach the endosome-lysosome compartment and dovetail nicely with the previously identified contribution of gp110 to virus-cell fusion. Other essential steps of the virus life cycle were unaffected in the viral mutant; DNA lytic replication and viral titers were not altered in the absence of gp110, and DeltaBALF4 viruses complemented in trans transformed infected B cells with an efficiency indistinguishable from that observed with wild-type viruses. All of the steps of virus maturation could be observed in lytically induced 293/DeltaBALF4 cells. Induction of lymphoblastoid cells generated with transiently complemented DeltaBALF4 virus led to the production of rare mature virions. We therefore infer that gp110
is not required for virus maturation and egress in 293 cells or in B cells. The DeltaBALF4 virus's phenotypic traits, an inability to infect human cells coupled with potent antigenicity, potentially qualify this mutant as a live vaccine. It will provide a useful tool for the detailed study of EBV-cell interactions in a physiological context.

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