Concluding remarks Section 3

This section proposes a crucial role for DCs in the typical characteristics of MV infection, including the efficient transmission of the virus, the strong virus-specific immune responses and the induction of a transient, but profound immune suppression. The C-type lectin DC-SIGN is an attachment receptor for MV and mediates *cis*-infection of DCs through CD150 and *trans*-infection of T cells (Figure 3.5). These data provide a likely scenario for MV to infect a new host: DCs capture the virus, migrate to the lymphoid tissues and mediate transmission of the virus to CD4⁺ and CD8⁺ T cells. It is tempting to hypothesize that MV is internalized in similar compartments as HIV-1 (Section 2), before transmission and antigen presentation to T cells. However interaction with additional receptors, CD4/syndecan-3 for HIV-1, and CD150 for MV, might influence the intracellular routing. Indeed, CD150 inhibits *trans*-infection and is involved in antigen presentation (Figure 3.5). A role for DCs is further supported by the detection of MV-infected DCs in macaques with measles. DCs induce T cell responses as illustrated by our *in vitro* data that DCs efficiently present antigens to CD4⁺ T cells. Furthermore, chapter 5.3 will demonstrate that DCs cross-present MV-infected cells to CD8⁺ T cells. Moreover, interaction of the virus with DC-SIGN might contribute to immune suppression, by a prolonged and increased production of IL-10 (Figure 3.5).