Figure 6—of-Chapter 1. Mechanism of immune escape in HBV latency and replication. The essential immune escape feature in latent HBV infection is characterized by the limited expression of latent HBV proteins, while the Env envelope proteins are more than 80% antigenic during lytic replication. During latency infection, the copy number of the expressed Env antigen and of oncoprotein envelope proteins is low, but this is kept very low. It is not possible to produce Env or other HBV envelope proteins efficiently or to transduce by glycoprotein (GP) Ag repeat. The natural GP repeat domain inhibits processing of viral Env molecules. It is considered that the major reason that Env is down-regulated, but also protects HBV against antiviral resistance and immunomodulation. LMP1 expression provides SHV-infected cells with an antieat T cell help (T cell), which is normally neither expressed specifically (Lmp1-Cmdt) interaction present in the T cells. Immune-reactive regions (IMA) of LMP1 have been shown to be the expression hallmark of latent HBV and associate with the tumour necrosis factor (TNF) receptor-associated death domain (TRADD) and TRAFs (TNF receptor 1-associated death domain proteins). LMP1 contains a well-conserved immunosuppressive domain (LALLP; LMP1 sequence, which activity suppresses antigens, polyplosis, and LMP1 expression in SHV-infected cells in the form of enzymes, generate a function in immune suppression, free. LMP1/MA/MA-sequences may directly interact T cell receptor via MHC and co-receptor interaction, allowing immune response into the T cell and LMP1 (LALLP; LMP1) to physically disturb T cell signaling. Dying by death several mechanisms in immune escape are proposed. EGFR inhibits the LMP1-induced immunosuppressive pathway in HBV infection. EGFR2 may prevent the formation of immune complexes by blocking the binding of both peptides and ATP to the transporter complex ATP7. The products of the proteins EGFR2-1, genes, decreases. It's not clear whether the presence of EGFR and MHC-LMP1, a substrate of the immunosuppressive that shares a homologous promoter with the TRAF gene. EGFR downregulates MHC class I expression at the cell surface by targeting MHC class I molecules for lysosomal degradation. EGFR inhibits the synthesis of heat shock proteins, including MHC class I molecules, through its virus host-shutoff function. EGFR also blocks MKP3 class phosphorylation. Adapted from [13, 28, 29, 336, 406].
Figure 1 of Chapter 7: Antibody detection by indirect immunofluorescence of acetone-fixed SP cells infected with Bar-code-anti-RB (A, B), Bar-code-LMP1 (C, D), Bar-code-LMP3A (E, F), Bar-code-BABF3 (G, H, and I), and MTV HaCaT (J). Cells were incubated with anti-SPX1 (1:250), mouse anti-SPX1 (1:100), mouse anti-LMP1 (1:100), rabbit anti-LMP1 (1:100), rabbit anti-LMF1 (1:100), rabbit anti-BABF3 (1:100), and rabbit anti-SPX1 (1:100), rabbit anti-LMF1 (1:100), rabbit anti-BABF3 (1:100), and rabbit anti-SPX1 (1:100).}

Figure 4 of Chapter 7: Accessibility of extracellular loops of LMP1 on viable SA3 and R0 cells as determined by specific anti-loop antibodies. (A) In all experiments, both negative LMP1 cells produced a negative monocyte taining with the LMP1 and LMP3 loop-specific sera. (B) Positive control (RAI) and R0I cells showed more than 50M positive staining using anti-200 antibodies. (C) Negative control in the absence of RAI cells, with anti-LMP1 loop 1-3 specific antiserum produced a similar patch-like staining on stably transfected R0I cells induced for LMP1 expression during 24 hours from the in vivo regulated promoter. (D) Anti-LMP1 loop 2-3: produced a similar pattern staining all R0I cells, with somewhat larger patches than observed for LMP1. (E) Flow cytometry histograms comparing the levels of accessibility of anti loops LMP1 on R0I cells. Staining obtained with the indicated anti-loop 1 (blue line), anti-loop 2 (blue line) and anti-loop 3 (blue line) and using anti-LMP3 specific control (green line). Background staining with pre-serum shows an unknown specificity indicated by red line.