The MHC class I status of tumour cells during immunotherapy is often underestimated. It represents one of important tumour escape mechanisms and thus can contribute to the failure of most of the cancer clinical trials that are usually based on the induction of cytotoxic T cell responses. Epigenetic changes in the promoters of genes involved in the MHC class I Ag presentation can result in decreased expression of the cell surface MHC molecules on tumour cells. Thus, epigenetic modifiers can restore an expression of the MHC class I molecules and make tumours visible to the $CD8^+$ effector cells. Besides the epigenetic changes on the tumour cells, epigenetic modulators affect cells of the immune system such as dendritic cells (DC). Tumour cells can escape from the immune response not only by changes in the cancer cells, but also by influencing, expanding and/or activating immunoregulatory cell populations, such as regulatory T cells (Treg).

This thesis focuses on the potential of the DC-based vaccines against HPV-16-associated tumours with a different MHC class I expression, on the combination of cancer immunotherapy with the treatment using epigenetic modifiers, with special attention paid to their effects on DC, and, finally, on the impacts of the anti-CD25 antibody (used for Treg elimination) on Treg and NKT cells, as well as on tumour progression.

Results obtained from the projects involved in this dissertation are important for optimization of vaccination and immunotherapeutic strategies that take into the account the MHC class I status of neoplasia. Our findings suggest that the efficiency of peptide vaccines against MHC class I-deficient tumours can be increased by peptides harbouring CD4 epitopes or by longer peptides requiring DC processing. In addition, the treatment of MHC class I-deficient tumour by epigenetic agents sensitized neoplasia towards the immunotherapy using of CpG ODN (oligodeoxynucleotide containing CpG motif) or IL-12-producing cellular vaccine. We reported the modulation of immune responses in several experimental settings. Application of anti-CD25 mAb (PC61) impaired the NKT cell activation and treatment by epigenetic modifiers interfered with DC maturation. Our data provide evidence that besides the known targets of epigenetic modifiers or immunoregulatory antibodies, other unspecific or indirect activities should be considered during the therapy.