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Evolution of vaccine resistant herpesviruses

Initiative: Lichtenberg - Professuren

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Projekt-Website: http://www.vetmed.fu-berlin.de/en/einrichtungen/institute/we05/02_mitarbeitende/

aktuelle_mitarbeitende/kaufer_benedikt/

Vaccines are the centerpiece of protection of humans and animals against viral diseases. Although most antiviral vaccines elicit robust immune responses, many viruses are still able to infect and replicate in the host, which contributes to continuous pathogen evolution. One virus that steadily evolves and undermines vaccine protection is the highly oncogenic herpesvirus Marek's disease virus (MDV). MDV not only causes a devastating disease that severely impairs the health of chickens, but is also an excellent model for virus-induced cancer and viral evolution. In this project, the hypothesis will be tested that mutations in the major oncogene meq drive vaccine resistance and that imperfect vaccines force evolution of MDV towards increased virulence. Recombinant viruses will be generated harboring representative meq isoforms to investigate the contributions of these changes in meq to virus replication, virulence, oncogenic potential and vaccine resistance. To determine if vaccination results in selection of more virulent viruses, a genetically highly diverse MDV population will be generated and the selective pressure on the virus in the natural target cells will be analyzed. The selection and evolution of the viruses in vaccinated and unvaccinated animals will be investigated by characterizing newly evolving viruses genetically and phenotypically.

Projektbeteiligte

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Unraveling the role of B cells in the pathogenesis of an oncogenic avian herpesvirus
The Transcriptional Landscape of Marek's Disease Virus in Primary Chicken B Cells Reveals Novel Splice Variants and Genes
Overexpression of cellular telomerase RNA enhances virus-induced cancer formation.
Identification of the receptor and cellular orthologue of the Marek s disease virus (MDV) CXC chemokine
Epstein-Barr virus-encoded RNAs (EBERs) complement the loss of the herpesvirus telomerase RNA (vTR) in virus-induced tumor formation