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Replication competent viral vectors for vaccine development

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Viral infections account for 15 million deaths per year, one-third of all mortalities worldwide. The most effective medical approach to combat viral diseases and reduce deaths is vaccinations, which have less adverse side effects than drugs while inducing longer lasting protection from re-infection. Live attenuated, inactivated or subunit vaccine approaches have been successfully utilized to combat mortalities caused by infectious diseases such as yellow fever, varicella, measles, mumps, rubella, influenza, smallpox, polio, rabies, hepatitis A and B and human papillomavirus. Viruses themselves have also been used as vectors (either replication competent or replication deficient) for development of vaccines against both infectious and non-infectious diseases. The most important factor in the construction of effective viral vectors is finding the right balance between safety and immunogenicity. Although live viral vaccine vectors are highly efficacious, there is also a greater potential risk involved with their broader usage because they are replication-competent. Vaccines based on replication-incompetent viruses are perceived to be safer but there is not yet any vaccine on the market for human use. In this talk, characteristics of both replication-deficient and replication-competent viral vectors and barriers for their developments will be discussed. The talk will specifically focus on a few vector examples that have either generated marketed products or have successfully completed their phase 3 efficacy trials.

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Papillomaviruses: From mutation to metastasis

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Bovine papillomavirus (BPV) is considered a useful model for HPV oncogenic process study. Both BPV and HPV are recognized as able to promoter cancer initiation and progression. Our results have reinforced the BPV mutagenic potential, which contributes to genomic instability and cancer initiation. However, currently results also point out that BPV promotes metabolic changes in host cells which induce Warburg effect after malignant transformation. This action is discussed as a consequence of hyper proliferative action of viral oncoproteins (E5, E6 and E7). This effect reduces the reactive oxygen species (ROS) production which is associated to cell survival. Moreover, both papilloma and esophageal carcinoma cell lines co-infected by BPV-1, 2 and 4 showed a migratory phenotype which was verified by the presence of lamellipodia and filopodia using immunofluorescence (IF) and time-lapse microscope. These data are evidences that BPV induces epithelial-mesenchymal transition (EMT), process characterized by the apoptosis resistance, metabolic changes and migratory phenotype acquisition. In order to verify the presence of EMT in these cells, expression levels of epithelial and mesenchymal proteins were evaluated by IF and flow cytometry. Results pointed out a reduction of epithelial biomarker expression levels which were accompanied by an increase of mesenchymal proteins. Thus, the combinatory results bring strong evidences that BPV participates of cancer initiation, progression and metastasis. Due to the morphological, genomic and pathological similarities between BPV and HPV, these results have to be considered in human infections.

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