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Characterization of an *in vitro* model to study the role of human Polyomavirus BK in prostate cancer

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Prostate cancer (PCa) is one of the most common male neoplasm in the western world, being the most commonly diagnosed non-skin cancer and the second leading cause of cancer death. Various potential risk factors exist for the initial triggering events, including exposure to infectious agents, such as the human Polyomavirus BK (BKV). BKV is a good candidate as risk factor of PCa because it naturally infects the human reno-urinary tract, it establishes latency, and encodes oncoproteins that interfere with tumor suppressors pathways, thus altering the normal progression of cell cycle.

Previous studies suggested a potential association between BKV and PCa, revealing that the prevalence of BKV was significantly higher in cancer than in control tissues, with a significant association between viral expression and cancer. However, this hypothesis is controversial because BKV is not restricted to tumor tissues but also infects healthy individuals in a high percentage. Moreover, an in vitro model of BKV infection in prostate cells is not available to understand the role for BKV in pathogenesis of PCa.

Our aims were to determine whether BKV a) could infect normal epithelial prostate cells, b) affects cell phenotype and c) affects the phenotype of human prostate tumor cell line PC3.

For this purpose normal epithelial prostate cell line RWPE-1 and prostate cancer cells PC3 were infected with BKV for 21 days. Cell proliferation, epithelial-to-mesenchymal markers (EMT) and invasion potential were analyzed by, respectively, MTT, immunofluorescence and SDS-zymography.

Our results show that cell proliferation was increased or decreased by BKV, respectively, in RWPE-1 and PC3 cells. BKV induced E-cadherin downregulation and vimentin expression in both control and BKV-infected cells RWPE-1, suggesting that uninfected cells underwent EMT. Matrix metalloproteinase-2 and 9 activity was increased in RWPE-1 cells after BKV infection. By contrast, BKV did not significantly modified the phenotype of PC3 cells.

These preliminary results suggest that normal epithelial prostate cells RWPE-1 and PC3 are susceptible and permissive to BKV infection. However, RWPE-1 cells exhibit some phenotype modifications related to EMT, possibly induced by the papilloma virus used to obtain their immortalization, thus suggesting that further experiments will be necessary to define if they represent a good experimental model to study prostate cancer.

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