Retrovirology

BioMed Central

Oral presentation

Effects of HPV-16 early proteins on trophoblastic cells

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from Fifth Dominique Dormont International Conference. Mother-to-child transmitted viral diseases: from transmission to children care Paris, France. 26–28 March 2009

Published: 22 July 2009

Retrovirology 2009, **6**(Suppl 1):O1 doi:10.1186/1742-4690-6-S1-O1

This abstract is available from: http://www.retrovirology.com/content/6/S1/O1

The trophoblastic cell represents the main functional unit of the placenta. It proliferates, migrates, and invades the maternal tissue in a way that is similar to malignant tumors. Nevertheless, these processes are tightly controlled by stringent spatial and temporal confines. Therefore, the trophoblastic cell, as 'a well-behaved tumor', represents an ideal model system to investigate several oncogenic processes. Several studies reported that HPV viruses could infect trophoblasts during pregnancies. Surprisingly, HPV can replicate *in vitro* in trophoblasts. Higher HPV infection frequency has been reported to be associated with some spontaneous abortion and gestational trophoblastic diseases.

In this study, we have studied the impacts of HPV-16 early proteins, mainly E5, E6 and E7, on the viability, adhesiveness, migration and invasion of trophoblastic cells.

Our results showed that the hydrophobic E5 protein is localized in many interne membranes compartments of the transfected trophoblast. E5 affects the viability of transiently and stably transfected trophoblastic cells. The viability seemed to be restored or even increased in the presence of E6 and E7. These observations were also confirmed by transfection in C33a cells, the HPV-negative human cervical carcinoma cell line. In addition, E5 decreased the adhesiveness of the trophoblastic cells to the support and to the endometrial cells. Cells expressing metastasis E6, E7 and in less extend E5 favour chemotaxic migration and matrigel invasion compared to the cells expressing the LacZ control. These effects are also observed when early proteins are expressed under the control of their own viral promoter (LCR). Our findings show that HPV-16 early proteins can affect the adhesiveness, the migration and the invasion of trophoblastic cells, key properties involved in placentation and metastasis.