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Poster presentation

The trophoblast: a model to study HPV transcription, replication and host cell interactions

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Background

Hallmarks of HPV infection include a restricted tropism for human epithelial cells and a viral life cycle tightly linked to the differentiation program of the host cells. This has hampered the study of the HPV vegetative life cycle. Previous studies reported that the tissue and differentiation dependence seemed to be dictated by viral transcription rather than viral DNA replication.

Objectives

1. To compare HPV transcription in cervical and trophoblastic cells and to study in those models the regulation of the LCR activity by various hormones and the viral early proteins. 2. To study the impact of the early viral proteins, especially E5, E6 and E7, on cell adhesiveness, migration and invasiveness.

Methods

To study transcription, we analyzed the activation of a reporter gene under the control of the HPV-16 LCR. To study replication, we measured, in RT-PCR after DpnI/MboI digestions, the amount of replicated DNA. Cellular properties were studied using various biological assays.

Results

The LCR activity was similar in both cell types and could be regulated by various hormones. To analyze the effect of all early proteins expression on the LCR activity and on viral replication in both cell types, the reporter plasmid was cotransfected with a plasmid allowing the expression of the entire early coding region under the control of its own HPV-16 LCR. Viral early proteins activated viral transcription and replication. Using various plasmids harboring point mutation in E1 or E2 ORF, we were able to observe that neither E1 nor E2 did play a role in the increased viral transcription. Early proteins could also modify the adhesion, the migration and the invasion of trophoblastic cells.

Conclusion

We will discuss about the interest of this model to identify new cell host (trophoblast)/pathogen (HPV) interactions.