DEPARTMENT OF BIOMEDICINE
HEALTH
AARHUS UNIVERISTY

I&I Seminar with Prof. Abel Viejo-Borbolla

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Varicella zoster virus modulates the activity of chemokines and interferon gamma to facilitate virus spread

Abstract:

During primary infection varicella zoster virus (VZV) employs lymphocytes to disseminate systemically in the human body. VZV infection of T cells remodels their receptor repertoire, inducing the expression of skin homing markers, facilitating their migration to the skin. Once in the skin, VZV infects keratinocytes causing the characteristic blisters of varicella or chickenpox. There is a second wave of T cell migration to the infected skin that contributes to resolution of infection in keratinocytes, but may also lead to further infection of these lymphocytes. Due to the relevance of T cell migration in VZV spread and thereby pathogenesis we asked whether this virus could modulate T cell migration to facilitate VZV spread.

Chemotaxis is a highly regulated process that requires the coordinated activity of chemokines, adhesion molecules and integrins. Binding of chemokines to their receptors on T cells triggers the expression and activation of integrins that bind to adhesion molecules, facilitating firm adhesion and extravasation. Other cytokines, like type II IFN, also increase the expression of adhesion molecules, playing a key role in T cell adhesion. In this talk I will present data showing that VZV modulates the activity of chemokines and type II interferon to increase T cell migration, adhesion and virus spread.

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