

Department of Molecular and Cellular Biology  
**Graduate Seminar MCB\*7500**

Friday, March 19, 2021 @12 p.m.

*presented by:*

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**“Regulation of SNAREs by phosphorylation during focal adhesion and invadopodium formation in tumour cells”**

Tumour cell invasion through the extracellular matrix (ECM) requires controlled localization of proteins that are necessary for ECM proteolysis and cell migration. Movement through the ECM requires cells and integrins to bind to ECM proteins and proteases to degrade the matrix. Adhesion to the ECM is mediated by integrin-based structures known as focal adhesions. In contrast, tumour cell invasion can occur through structures known as invadopodia, which are membrane protrusions that extend into the ECM and facilitate its localized degradation. Both invadopodia and focal adhesions have been described in several invasive cancer cell types. Soluble N-ethylmaleimide-sensitive factor-attachment protein receptors (SNAREs) play a fundamental role in vesicle trafficking, as they mediate the fusion of vesicles with target membranes. SNARE-mediated trafficking is known to have a significant role in cell-ECM interactions, invadopodium formation, as well as tumour cell invasion and migration; however, the regulation of SNAREs in these processes is not well understood. During this study, we will examine SNARE-mediated vesicle trafficking pathways that regulate the formation of focal adhesions and invadopodia. Although focal adhesions and invadopodia are related structures, it is unknown what regulatory and signalling mechanism they have in common. We propose that phosphorylation of SNAREs is an important mechanism for the regulation of their activity during focal adhesion dynamics and invadopodium formation and function. The phosphorylation of SNAREs will be assessed and how this contributes to cell-ECM interactions in cancer cells will be examined. These investigations will provide insight into the molecular mechanisms that control the coordinated cell-ECM adhesion and matrix remodelling that occurs during cell invasion of the ECM.