The Phosphorylation and Distribution of Cortactin Downstream of Integrin α9β1 Affects Cancer Cell Behaviour

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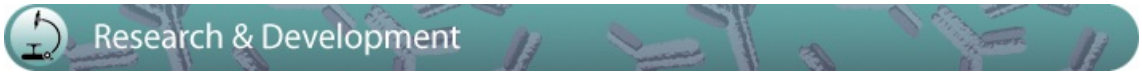

The Phosphorylation and Distribution of Cortactin Downstream of Integrin α9β1 Affects Cancer Cell Behaviour [1]

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Integrins, a family of heterodimeric adhesion receptors are implicated in cell migration, development and cancer progression. They can adopt conformations that reflect their activation states and thereby impact adhesion strength and migration. Integrins in an intermediate activation state may be optimal for migration and we have shown previously that fully activated integrin α9β1 corresponds with less migratory behaviour in melanoma cells. Here, we aimed to identify components associated with the activation status of α9β1. Using cancer cell lines with naturally occuring high levels of this integrin, activation by α9β1-specific ligands led to upregulation of fibronectin matrix assembly and tyrosine phosphorylation of cortactin on tyrosine 470 (Y470). Specifically, cortactin phosphorylated on Y470, but not Y421, redistributed together with α9β1 to focal adhesions where active β1 integrin also localises, upon integrin activation. This was commensurate with reduced migration. The localisation and phosphorylation of cortactin Y470 was regulated by Yes kinase and PTEN phosphatase. Cortactin levels influenced fibronectin matrix assembly and active β1 integrin on the cell surface, being inversely correlated with migratory behaviour. This study underlines the complex interplay between cortactin and α9β1 integrin that regulates cell-extracellular matrix interactions.

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