

Published online September 18, 2006. doi:10.1083/jcb.200605022

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JCB, Volume 174, Number 7, 1087-1096

## Article

# p120 catenin is essential for mesenchymal cadherin-mediated regulation of cell motility and invasiveness

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During epithelial tumor progression, the loss of E-cadherin expression and inappropriate expression of mesenchymal cadherins coincide with increased invasiveness. Reexpression experiments have established E-cadherin as an invasion suppressor. However, the mechanism by which E-cadherin suppresses invasiveness and the role of mesenchymal cadherins are poorly understood. We show that both p120 catenin and mesenchymal cadherins are required for the invasiveness of E-cadherin-deficient cells. p120 binding promotes the up-regulation of mesenchymal cadherins and the activation of Rac1, which are essential for cell migration and invasiveness. p120 also promotes invasiveness by inhibiting RhoA activity, independently of cadherin association. Furthermore, association of endogenous p120 with E-cadherin is required for E-cadherin-mediated suppression of invasiveness and is accompanied by a reduction in mesenchymal cadherin levels. The data indicate that p120 acts as a rheostat, promoting a sessile cellular phenotype when associated with E-cadherin or a motile phenotype when associated with mesenchymal cadherins.

Abbreviations used in this paper: HGF, hepatocyte growth factor; shRNA, short hairpin RNA; wt, wild-type.

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