Title:	The strong propensity of Cadherin-23 for aggregation inhibits cell migration
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Keywords:	Cadherin-23 Non-classical cadherin Neuroepithelial
Issue Date:	2019
Publisher:	FEBS Press and John Wiley & Sons Ltd.
Citation:	Molecular Oncology, 13(5),pp. 1092-1109.
Abstract:	Cadherin-23 (Cdh23), a long-chain non-classical cadherin, exhibits strong homophilic and heterophilic binding. The physiological relevance of strong heterophilic binding with protocadherin-15 at neuroepithelial tip links is well-studied. However, the role of Cdh23 homodimers in physiology is less understood, despite its widespread expression at the cell boundaries of various human and mouse tissues, including kidney, muscle, testes, and heart. Here, we performed immunofluorescence studies that revealed that Cdh23 is present as distinct puncta at the cell–cell boundaries of cancer cells. Analysis of patient data and quantitative estimation of Cdh23 in human tissues (normal and tumor) also indicated that Cdh23 is down-regulated via promoter methylation in lung adenocarcinoma (AD) and esophageal squamous cell carcinoma (SCC) cells; we also observed a clear inverse correlation between Cdh23 expression and cancer metastasis. Using HEK293T cells and four types of cancer cells differentially expressing Cdh23, we observed that cell migration was faster in cells with reduced levels of Cdh23 expression. The cell migration rate in cancer cells is further accelerated by the presence of excretory isoforms of Cdh23, which loosen its cell-adhesion ability by competitive binding. Overall, our data indicate the role of Cdh23 as a suppressor of cell migration.
URI:	https://febs.onlinelibrary.wiley.com/doi/10.1002/1878-0261.12469 (https://febs.onlinelibrary.wiley.com/doi/10.1002/1878-0261.12469) http://hdl.handle.net/123456789/2071 (http://hdl.handle.net/123456789/2071)
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