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Title:	Structural basis of the strong cell-cell junction formed by cadherin-23
Authors:	Singaraju, G.S. (/jspui/browse?type=author&value=Singaraju%2C+G.S.) Sagar, A. (/jspui/browse?type=author&value=Sagar%2C+A.) Kumar, Anuj (/jspui/browse?type=author&value=Kumar%2C+Anuj) Samuel, J.S. (/jspui/browse?type=author&value=Samuel%2C+J.S.) Hazra, J.P. (/jspui/browse?type=author&value=Hazra%2C+J.P.) Sannigrahi, M.K. (/jspui/browse?type=author&value=Sannigrahi%2C+M.K.) Rakshit, S. (/jspui/browse?type=author&value=Rakshit%2C+S.)
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Abstract:	Cadherin-23, a giant atypical cadherin, form homophilic interactions at the cell–cell junction of epithelial cells and heterophilic interactions with protocadherin-15 at the tip links of neuroepithelial cells. While the molecular structure of the heterodimer is solved, the homodimer structure is yet to be resolved. The homodimers play an essential role in cell–cell adhesion as the downregulation of cadherin-23 in cancers loosen the intercellular junction resulting in faster migration of cancer cells and a significant drop in patient survival. In vitro studies have measured a stronger aggregation propensity of cadherin-23 compared to typical E-cadherin. Here, we deciphered the unique trans-homodimer structure of cadherin-23 in solution and show that it consists of two electrostatic-based interfaces extended up to two terminal domains. The interface is robust, with a low off-rate of ~ 8 × 10−4 s−1 that supports its strong aggregation propensity. We identified a point mutation, E78K, that disrupts this binding. Interestingly, a mutation at the interface was reported in skin cancer. Overall, the structural basis of the strong cadherin-23 adhesion may have far-reaching applications in the fields of mechanobiology and cancer.
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