

# Metastatic movements in 3-D

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Caswell et al. report in the *Journal of Cell Biology* how the altered behavior of integrins can prompt metastatic movement in tumor cells.

On 2D surfaces, cells may migrate randomly, or be strongly unidirectional. Integrins, which link the cell to the extracellular matrix, are known to influence the mode of migration, but exactly how has been unclear.

Recent work has suggested that an integrin called  $\alpha 5 \beta 1$  drives random movement, while an integrin called  $\alpha v \beta 3$  has been associated with unidirectional migration—the balance of activity between the two determining the type of movement. To further explore the contribution of  $\alpha 5 \beta 1$  to random migration, the authors thus blocked  $\alpha v \beta 3$ .

The treated cells changed their mode of migration from unidirectional to random, and their ability to invade 3D gels increased. The changed behavior correlated with an increase in trafficking of  $\alpha 5 \beta 1$  from intracellular compartments to anterior membrane protrusions. But this increase in trafficking did not significantly alter  $\alpha 5 \beta 1$ 's contribution to cell adhesion—the ease with which cells were dislodged from a spinning disk increased as the amount of  $\alpha v \beta 3$  was reduced, but was not correlated with any change in  $\alpha 5 \beta 1$ . This suggested that the cells' increased invasive ability was due to alteration in some other property. That property turned out to be activation of a proinvasive pathway headed by a kinase called Akt.

In  $\alpha v \beta 3$ -blocked cells,  $\alpha 5 \beta 1$  became associated with epidermal growth

factor receptor 1 (EGFR1), which increased EGFR1's abundance at the membrane protrusions, as well as its autophosphorylation. Because EGFR1 is an activator of the Akt pathway, hey presto, the cells took on some new moves.

Source: Rockefeller University Press

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