SRGP-1 drives cell junctions round the bend

A n F-BAR domain protein bends the plasma membrane to promote the formation of intercellular adhesions, Zaidel-Bar et al. report.

BAR (Bin1, Apmphiphysin, and RVS167) family proteins curve membranes inward or outward to facilitate endocytosis, filopodia formation, and other membrane movements. Zaidel-Bar et al. found that a C. elegans F-BAR protein, SRGP-1, localized to the junctions between neighboring worm cells, and that these adhesions formed more slowly in the protein’s absence. Nevertheless, SRGP-1 was dispensable for embryogenesis unless the worms also carried mutations in key junctional components like α-catenin: when both junctions and SRGP-1 were compromised, embryos died because neuroblasts and epidermal cells failed to seal up holes and enclose the embryos’ outer surface. A truncated version of SRGP-1, containing just the protein’s F-BAR domain and a junctional targeting sequence, was sufficient to rescue these embryonic defects.

In most cases, F-BAR domains are thought to pull membranes inward, but Zaidel-Bar et al. found that overexpressing SRGP-1 had the opposite effect, pushing out the plasma membranes and junctions of epidermal cells so that they protruded into their neighbors.

Lead author Ronen Zaidel-Bar thinks that this activity might facilitate the formation of intercellular adhesions by increasing the area of contact between adjacent cells. He now wants to investigate the four human homologues of SRGP-1 and their potential function in epithelial and endothelial cell adhesion.