Epithelial cells communicate amongst themselves using finger-like projections, according to Cyrille de Joussineau, Daniel Alexandre, and colleagues (Université Montpellier II, Montpellier, France). The filopodia help individual fly neural precursors to create an island of nonneural cells around themselves, thus allowing the formation of discrete structures such as bristles at regular intervals.

The filopodia only become visible when single cells are labeled. Their inhibitory action is mediated by Delta attached to the filopodia, which contacts membrane-bound Notch on the surrounding, inhibited cells. The extent of visible filopodia roughly matched the range of inhibition. And overexpression of Delta increased the range of the filopodia, so Delta actually promotes formation of its own means of transport.

Inhibition of filopodial outgrowth did not prevent local Delta–Notch signaling, but did shut down longer range inhibition. The result was an increased density of neural cells and more of the associated structures such as microchaetes. Thus, epithelial cells can communicate at long distances without resorting to either diffusible mediators or cell relay mechanisms.

Reference: de Joussineau, C., et al. 2003. Nature. 426:555–559.

Filopodia allow epithelia to communicate over long distances.

Epithelial cells zipper together thanks to linear actin cables—cables that assemble at newly formed adherens junctions to stabilize them, thereby counteracting retreactive forces at sites of cell–cell contact. Now, Agnieszka Kobielak, Amalia Pasolli, and Elaine Fuchs (Rockefeller University, New York, NY) have found that formin-1 drives the actin polymerization that creates the cables.

Formin-1 entered the story as a binding partner of α-catenin, a component of cadherin adhesion complexes. Cells lacking α-catenin fail to form actin cables at adherens junctions, and the Rockefeller group found that the same was true when the formin-1–α-catenin interaction was disrupted in vivo. In vitro, formin-1 was shown to polymerize actin into linear filaments. Finally, fusion of a β-catenin-binding domain to the actin-polymerization domains of formin-1 restored adhesion ability to cells lacking α-catenin.

Actin polymerization is important in two steps of adhesion. First, branched polymerization of actin by Arp2/3 pushes out both filopodia and broad areas of membrane as lamellipodia. Many of the resultant contacts are not productive, and the processes retract. But any surviving contact prompts the formation of an actin cable, which stabilizes the contact. It also pushes on a specific area of membrane so that more adherens junctions form nearby, thus zipping cells together. Just how formin-1 is regulated during this process remains to be determined.

Reference: Kobielak, A., et al., 2003. Nat. Cell Bio. 10.1038/ncb1075.

Formin’ adherens junctions

Epithelial cells zipper together thanks to linear actin cables—cables that assemble at newly formed adherens junctions to stabilize them, thereby counteracting retreactive forces at sites of cell–cell contact. Now, Agnieszka Kobielak, Amalia Pasolli, and Elaine Fuchs (Rockefeller University, New York, NY) have found that formin-1 drives the actin polymerization that creates the cables.

Formin-1 (green) localization at cell junctions (left) is lost in an α-catenin knockout (right).

Epithelial cells zipper together thanks to linear actin cables—cables that assemble at newly formed adherens junctions to stabilize them, thereby counteracting retreactive forces at sites of cell–cell contact. Now, Agnieszka Kobielak, Amalia Pasolli, and Elaine Fuchs (Rockefeller University, New York, NY) have found that formin-1 drives the actin polymerization that creates the cables.

Formin-1 (green) localization at cell junctions (left) is lost in an α-catenin knockout (right).

Food and sex go hand in hand, according to Yukimasa Shibata, Siegfried Hekimi, and colleagues (McGill University, Montreal, Canada). They find that worms that have less oxidation of certain lipoprotein particles—possibly an indicator of a slowed metabolism—have slowed development of their germline. Only when food is abundant and metabolism active would the germline get the stimulus to develop to maturity.

The proteins in question are vitellogenins: analogues of vertebrate apoB, a component of low-density lipoprotein (LDL). In the clk-1 worm mutant, an increase in the levels of an antioxidant results in less oxidation of lipoprotein. The result is a slowing in germline development.

The slowed development is reversed by blocking the production of lipoproteins, or by reestablishing a more normal level of oxidation by reactive oxygen species (ROS). “The degree of oxidation is a measure of general metabolism,” says Hekimi. “The germline may want to know that the worm is running fast. It could be sensing the general quality of metabolism.” ROS effects on signaling have been seen before in vitro, but the new results are the most dramatic to be detected in vivo.

The effects of lipoproteins on germline development go through a receptor-associated kinase called ARK-1. Although ARK-1 is known to work downstream of an EGF-like receptor, it is not clear whether this or another type of receptor is a mediator for the lipoprotein signal, or which lipoprotein species (oxidized or nonoxidized) is doing the signaling.

Reference: Shibata, Y., et al. 2003. Science. 302:1779–1782.

Oxidation makes a germline