Y oung rat hippocampal neurons are a ball of undifferentiated processes, one of which will become the axon, whereas the rest become dendrites. On page 499, Lamoureux et al. show that any of the processes can become the axon when given the right stimulus. But this cue is not a hormone or growth factor. The stimulus that tells an axon to form is more physical than chemical.

What the neurite needs to become an axon is a good tug, which the authors applied by attaching a needle to the end of a process. In most cases, the processes that were pulled extended rapidly and expressed axonal markers. Thus, every process appears ready to form an axon, perhaps with stores of unassembled tubulin and vesicles ready to be secreted. Tension somehow elicits the rapid biochemical reactions needed to make an axon, including microtubule assembly and plasma membrane expansion, although how tension is transduced is unclear.

The experiments dispel theories suggesting that the first process to reach a critical length become the axon, as processes pulled and then released when still shorter than its neighbors continued to grow and mature into axons. Tension even elicited a second axon in cells that already had one. Thus, whatever factor prevents a second axon from forming is apparently overcome by tension. Perhaps tension, which in vivo is provided by the growth cone’s tugging on the extracellular matrix, is tightly regulated in the developing organism by precisely placed growth factors.

**E-Cadherin in a stabilizing relationship**

O n page 465, Ireton et al. report an unexpected mechanism by which cells can increase E-cadherin levels to boost their stickiness. Their findings stem from an unusual cell line that allows a direct analysis of the function of a cadherin modulator.

E-cadherin is the main cell–cell adhesion molecule in epithelial tissues. Down-regulation of E-cadherin occurs in many carcinoma types and correlates with the transition to metastasis. Recently, several tumor types have also been reported to have low levels of p120, a catenin thought to regulate the linkage of E-cadherin to the actin cytoskeleton. Now, Ireton et al. describe the first example of a carcinoma cell line—a colon cancer line, SW48—with mutations in p120 that cause low levels of the protein. This cell line reveals that p120 has a surprising effect on cadherin stability.

As expected, restoring expression of p120 rescued the adhesive phenotype of SW48 cells. Unexpectedly, the effect appears to be mediated by stabilization of the E-cadherin protein. Though it is still unclear how E-cadherin is stabilized, only forms of p120 that bound to the cadherin rescued the SW48 phenotype. One speculation is that p120 binding might displace cadherin destabilizers, such as presenilin or Hakai. Whatever the mechanism, the down-regulation of E-cadherin in some tumors could be a result of prior p120 loss.

**Ribosomes pile up**

E xtensive piles of internal membranes accumulate when protein targeting to membranes is interrupted in E. coli, as seen in an article by Herskovits et al. on page 465. The membranes contain ribosomes and a receptor with an early function in ribosome targeting.

The receptor is FtsY, a homologue of the eukaryotic signal recognition particle (SRP) receptor. Recent evidence suggests that, unlike eukaryotes, E. coli do not need SRP for ribosome targeting to the membrane, where membrane proteins will be inserted. However, the process does depend on the receptor FtsY. In the new paper, FtsY function is highlighted.

The result is a visual confirmation that FtsY brings ribosomes to the membrane even in the absence of one of its ligands, the SRP. Herskovits et al. disrupted steps downstream of this event—the transfer of ribosomes to the translocon, which inserts newly translated membrane proteins into the membrane—by depleting cells of SRP or translocon components. Thus, the normally transient association of FtsY with ribosomes was stalled, and novel membrane-bound FtsY–ribosome complexes accumulated in a membrane network. The networks lie close to and might be derived from the cytoplasmic membrane. The fact that SRP is not necessary for ribosome targeting to the membrane begs a new question—how do mRNAs encoding membrane proteins find their way to membrane-bound ribosomes?