During mitosis, the *Drosophila* protein Asp binds and focuses microtubule minus ends at the spindle poles, and maintains their attachment to the centrosomes. Two groups of researchers now provide insights into how the protein achieves these functions (1, 2).

*asp* was one of the first genes to be identified as being required for correct assembly of the *Drosophila* mitotic spindle (3), and mutations in its human homologue, *ASPM*, cause microcephaly. Despite this, Asp’s biochemical function has remained uncertain. “We wanted to know what the molecular activity of this protein was,” explains Gohta Goshima, from Nagoya University in Japan.

One possibility was that Asp works with Ncd, a minus end–directed kinesin motor protein that can cross-link microtubules and promote spindle pole focusing (4). However, when Goshima and his graduate student Ami Ito closely compared the phenotypes of *Drosophila* S2 cells lacking Asp and/or Ncd, they realized that the two proteins act independently of each other (1). Asp was required to keep microtubule minus ends focused at the spindle poles from the onset of prometaphase onwards. Ncd, in contrast, wasn’t required until mid-prometaphase, and it appeared to coalesce microtubules throughout the spindle, rather than just focusing their minus ends at the poles.

Ito and Goshima therefore wondered whether Asp itself was capable of cross-linking microtubules. Asp contains a microtubule-binding domain near its N terminus, but the researchers found that a central region of the protein was able to cross-link microtubules in vitro. This region was necessary, but not sufficient, for Asp’s pole focusing activity in vivo.

Although Asp mainly accumulates at microtubule minus ends near the spindle poles, Ito and Goshima noticed that, in live cells, small puncta of GFP-tagged Asp, and it appeared to coalesce microtubules throughout the spindle, rather than just focusing their minus ends at the poles.

In keeping with Asp’s links to microcephaly, flies carrying hypomorphic *asp* alleles develop abnormally small brains (6). To better understand Asp’s role in neural development, Schoborg et al. used CRISPR to generate *asp* null flies. These animals also developed small brains, and their neuroblasts, which give rise to the nervous system through a series of asymmetric cell divisions, formed abnormal mitotic spindles with unfocused poles and detached centrosomes. These spindle defects could be rescued by full-length Asp, but not by mutant versions unable to bind CaM. “So the Asp-CaM interaction is important for pole focusing and centrosome attachment,” Schoborg explains.

Surprisingly, however, these CaM-binding mutants were able to rescue the flies’ brain size, suggesting that Asp has an additional, CaM-independent function, and that microcephaly doesn’t arise from defects in the mitotic spindle. Schoborg and Rusan now want to investigate what this additional function might be and to determine how CaM stabilizes Asp’s role in spindle organization. Preliminary results suggest that CaM stabilizes Asp and might promote its oligomerization. Goshima and colleagues, meanwhile, plan to check whether Asp’s function is conserved in human *ASPM*.