Huang et al., http://www.jcb.org/cgi/content/full/jcb.200806151/DC1

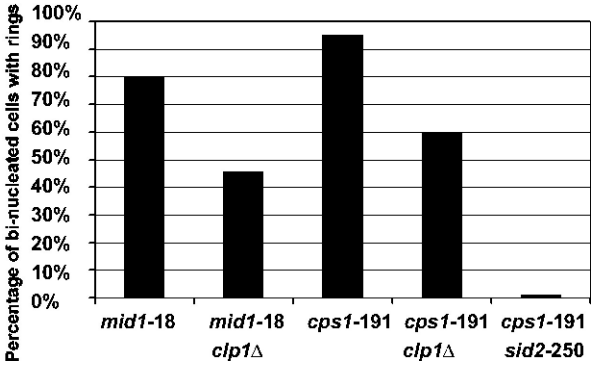

Figure S1. **Actomyosin ring/bundle assembly and maintenance in the absence of Mid1p and the cortical nodes depend on the SIN.** The fact that actomyosin bundle and ring assembly was significantly compromised in *mid1*Δ *clp1*Δ cells (Clp1p is a positive regulator of the SIN; Trautmann, S., B.A. Wolfe, P. Jorgensen, M. Tyers, K.L. Gould, and D. McCollum. 2001. *Curr. Biol.* 11:931–940) further established that ring assembly in the absence of Mid1p and the cortical nodes depended on the SIN. Additional genetic studies (Liu, J., H. Wang, D. McCollum, and M.K. Balasubramanian. 1999. *Genetics.* 153:1193–1203) showed the inability of *cps1*-191 *sid2*-250 (*sid2*-250 encodes a defective version of the SIN component Sid2p; Balasubramanian, M.K., D. McCollum, L. Chang, K.C. Wong, N.I. Naqvi, X. He, S. Sazer, and K.L. Gould. 1998. *Genetics.* 149:1265–1275; Sparks, C.A., M. Morphew, and D. McCollum. 1999. *J. Cell Biol.* 146:777–790) and a reduced ability of *cps1*-191 *clp1*Δ cells to assemble and maintain actomyosin rings. These genetic studies provided further evidence that the organization of actomyosin bundles in *mid1* mutants and their reorganization in the *cps1*-191 background depended on the function of the SIN. The figure shows quantitation of the presence of actomyosin rings/cables in binucleate cells of *mid1*-18, *mid1*-18 *clp1*Δ, *cps1*-191, *cps1*-191 *clp1*Δ, and *cps1*-191 *sid2*-250. Cells of the indicated genotypes were cultured at 25°C and were shifted to 36°C for 3.5 h, fixed, and stained with phalloidin and DAPI to visualize actomyosin rings/bundles and the nuclei, respectively. At least 200 cells were scored for each genotype.