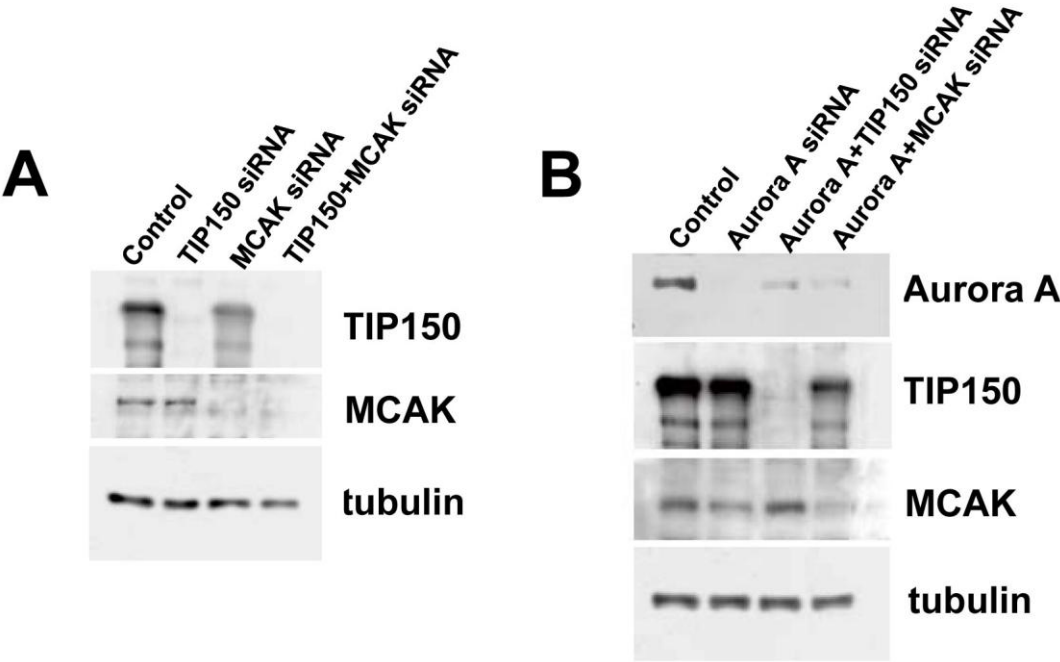


# Figure S1

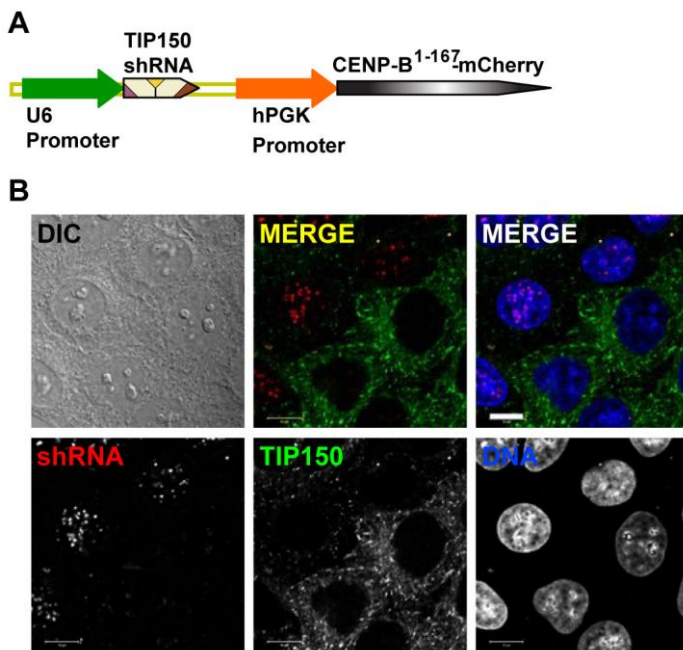


**Supplementary Figure S1. Characterization of siRNA knock-down efficiency.**

(A) Endogenous expression of TIP150 and MCAK in MCF7 cells were monitored by immunoblotting with anti-TIP150 and anti-MCAK antibodies, as indicated.

(B) Endogenous expression of Aurora A, TIP150 and MCAK in MCF7 cells were monitored by immunoblotting with anti-Aurora A, anti-TIP150 and anti-MCAK antibodies, respectively, as indicated. Surprisingly, a partial decrease of TIP150 level appeared in MCAK siRNA transfected cells, which made us presume that MCAK might take part in TIP150 folding or stabilization during translation from mRNA to proteins.

**Figure S2**

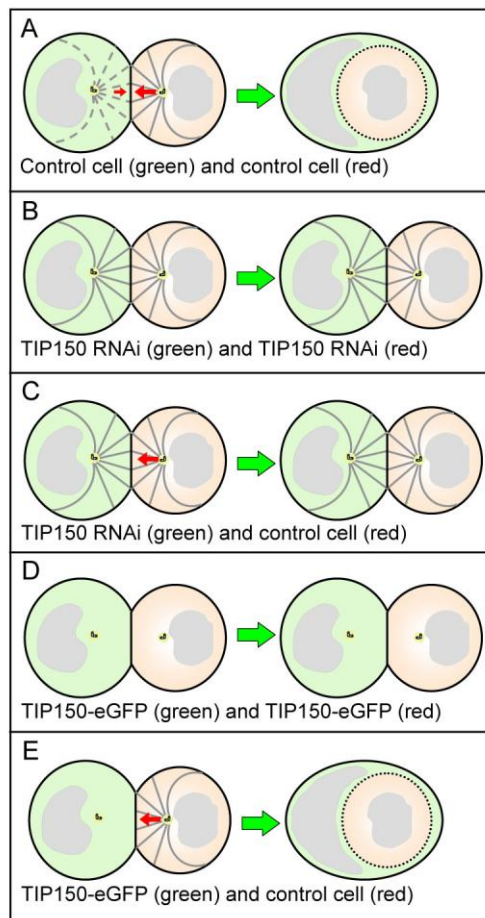


**Supplementary Figure S2. Characterization of a unique fluorescence-marker TIP150 shRNA system.**

(A) Schematic illustration of the system. PLKO.1 cloning vector was used as a backbone to build TIP150 shRNA. Sequence coding the shRNA targeted to TIP150 was inserted after U6 promoter. Coding sequence of puromycin resistance marker after hPGK promoter was replaced by CENP-B<sup>1-167</sup>-mCherry cDNA.

(B) Characterization of TIP150 shRNA knockdown efficiency. TIP150 stain in mCherry-CENP-B-marked cells is significantly decreased comparing with unsuccessfully transfected cells. The scale bar is 10  $\mu$ m.

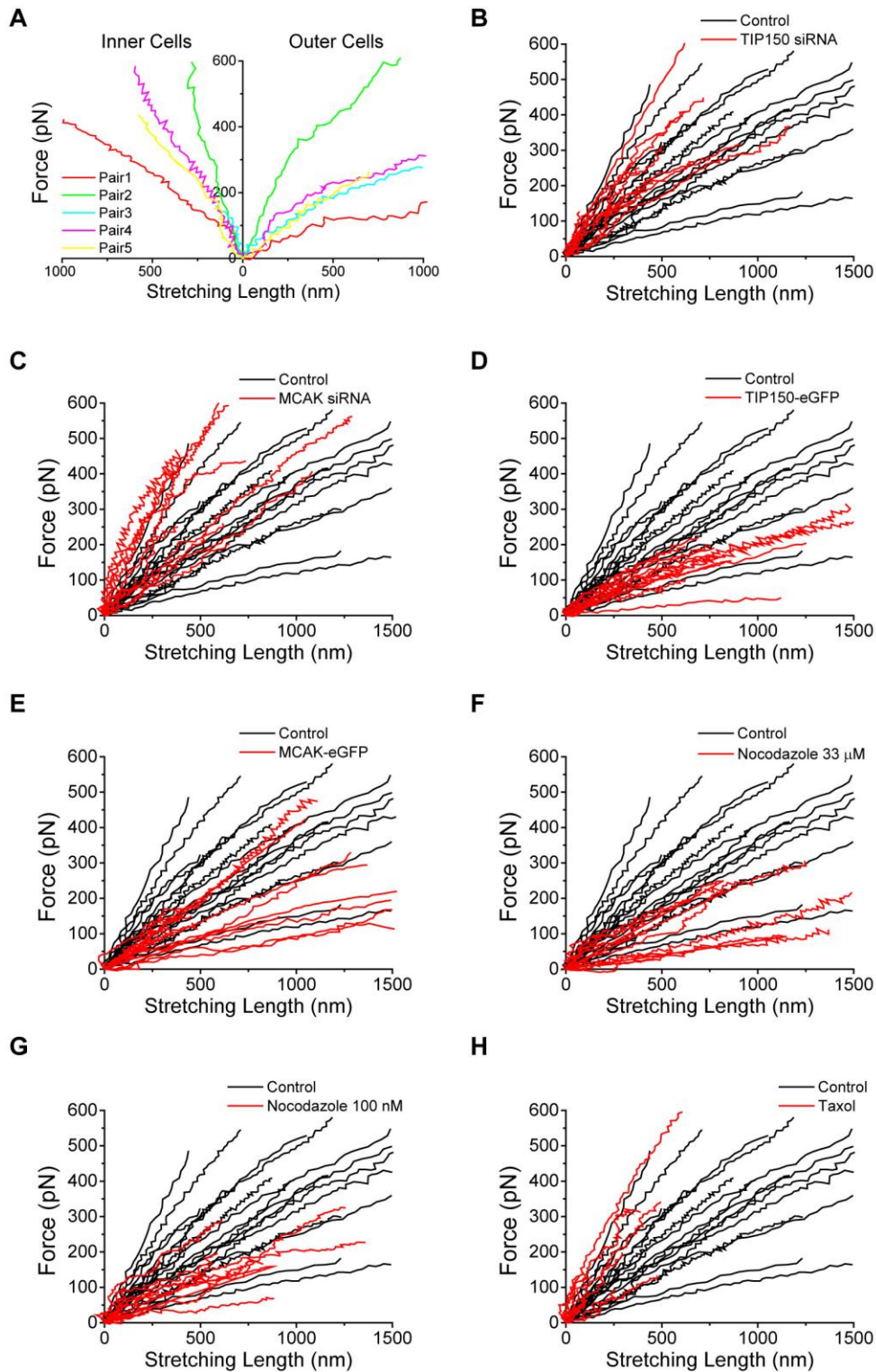
**Figure S3**



**Supplementary Figure S3. Working model accounting for the relationship between entosis and microtubule dynamics.**

- (A) One cell invades another driving by unbalanced cell contractile force (red arrows).
- (B) TIP150/MCAK knock-down cells can not invade each other by decreasing of microtubule dynamics.
- (C) TIP150/MCAK over-expression cells can not invade each other by depression of cell contractile force.
- (D) A TIP150/MCAK knock-down cell can not invade a control cell as lacking of microtubule dynamics, while it can also not be invaded by a control cell as it has extremely high cortex tension and difficult to change morphology.
- (E) A TIP150/MCAK over-expression cell tends to be invaded by a control cell as it lacks microtubule dynamics and has little cortex tension.

**Figure S4**

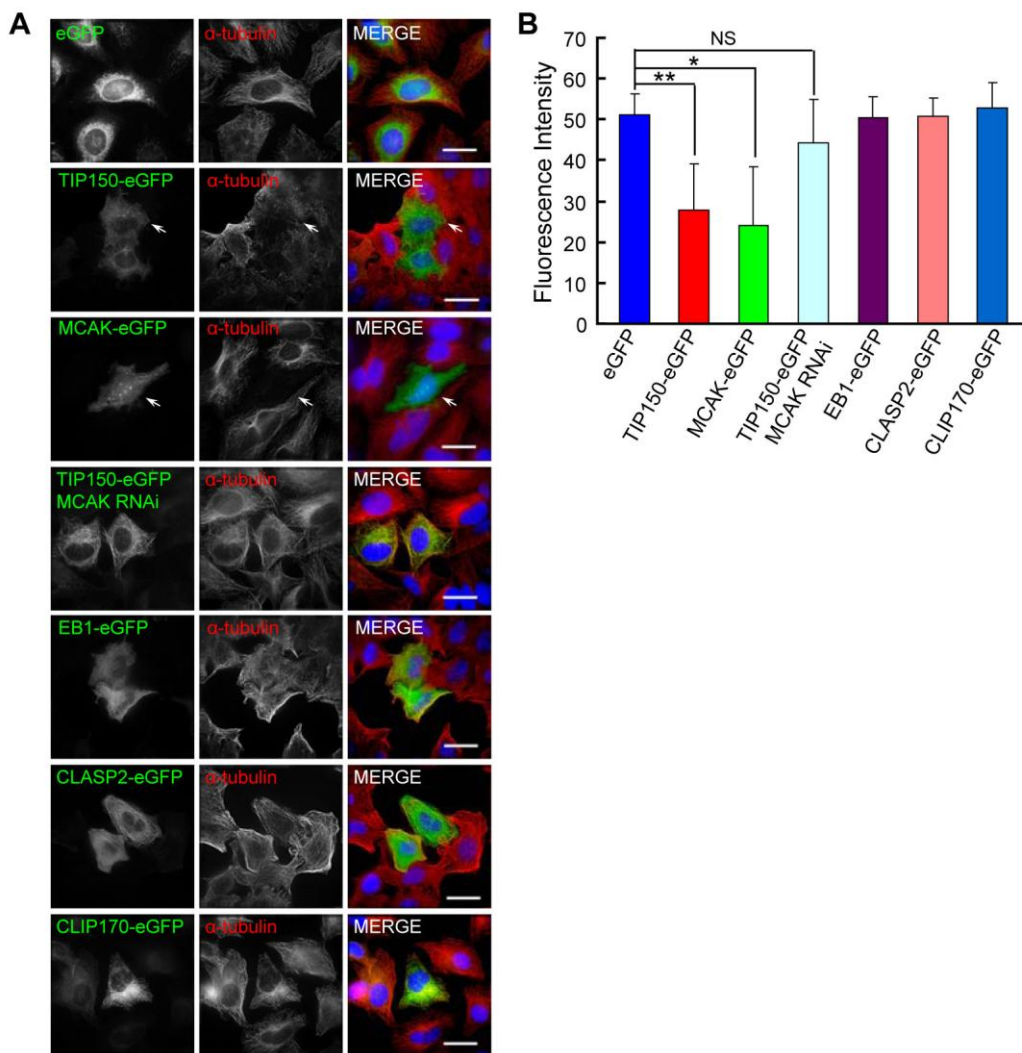


**Supplementary Figure S4. Analysis of cell rigidity by optical trap system.**

(A) Related to Figure 2J. Trapping force  $F$  versus stretching extension  $l$  of paired entosis cells.

(B-H) Related to Figure 2K and 2L. Trapping force  $F$  versus stretching extension  $l$  of cells in experiment or control groups.

**Figure S5**

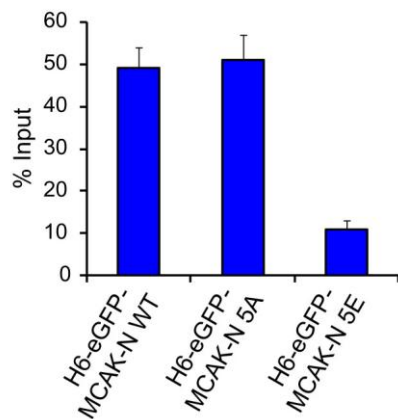


**Supplementary Figure S5. Interaction of MCAK and TIP150 is essential for MT depolymerization.**

(A) MCF7 cells were transfected with indicated GFP-tagged proteins and siRNA, followed by fixation and staining for  $\alpha$ -tubulin. Arrows indicate cells with MT loss. The scale bars are 25  $\mu$ m.

(B) Quantification of MT depolymerization experiments. Total tubulin polymer was measured by IF. MT depolymerization was enhanced in TIP150/MCAK over-expressing cells, but restored in cells over-expressing TIP150 but with knock-down of MCAK (for each condition, at least 200 cells from three independent experiments were analyzed). Data are means  $\pm$  SD. \*,  $p < 0.05$ ; \*\*,  $p < 0.01$ ; NS,  $p > 0.05$ .

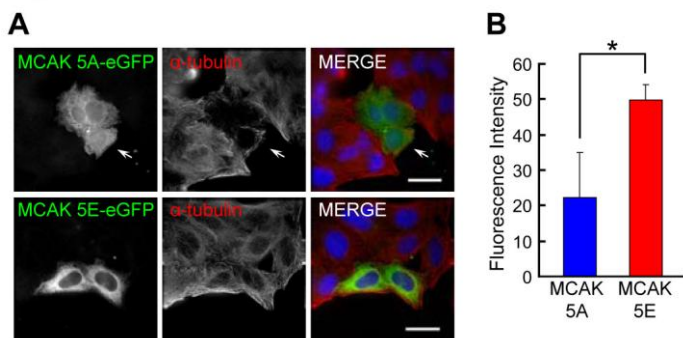
**Figure S6**



**Supplementary Figure S6. Related to Figure 3J.**

Quantitative analysis of H6-eGFP-MCAK-N WT/5A/5E binding to GST-MCAK-C. Data are means  $\pm$  SD of three independent experiments.

**Figure S7**

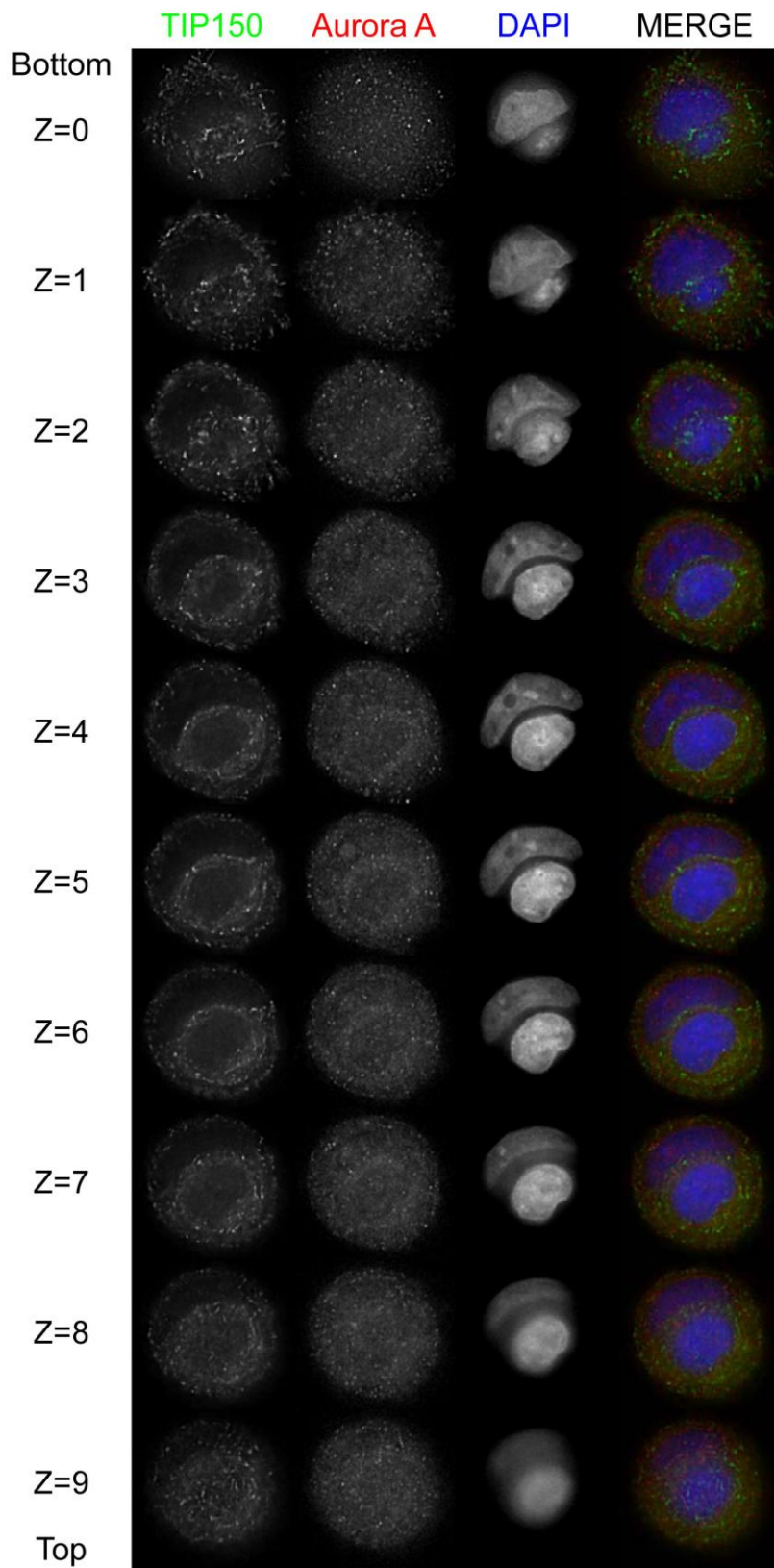


**Supplementary Figure S7. MCAK-5A but not MCAK-5E can depolymerize microtubule *in vivo*.**

(A) MCF7 cells were transfected with eGFP tagged MCAK-5A or 5E mutant and then fixed and stained for  $\alpha$ -tubulin. Arrows indicate cells with microtubule loss. The scale bars are 25  $\mu$ m.

(B) Quantification of *in vivo* microtubule depolymerization experiments. Total tubulin polymer was measured by immunofluorescence (for each condition, at least 200 cells from three independent experiments were analyzed). Microtubule depolymerization enhanced in MCAK-5A but not MCAK-5E over-expressing cells. Data are means  $\pm$  SD. \*,  $p < 0.05$ .

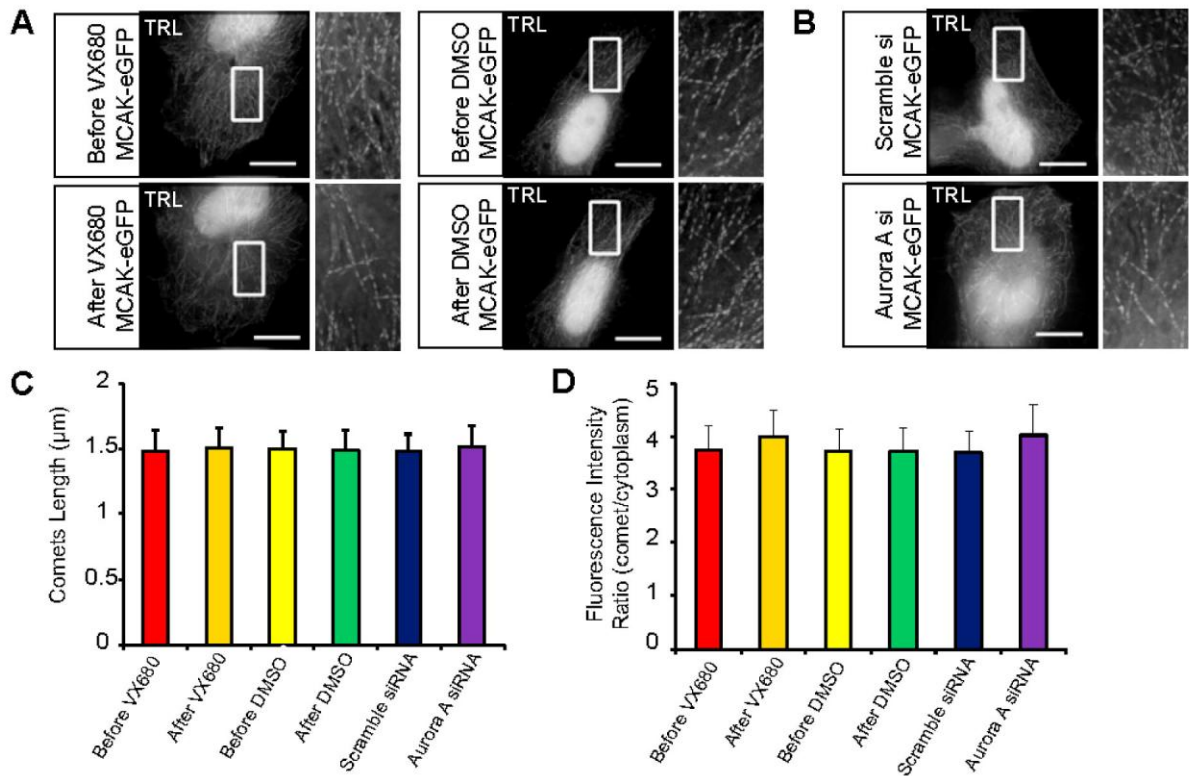
Figure S8



**Supplementary Figure S8. Related to Figure 5A.**

Tomography of entosis cells with immunofluorescence staining of TIP150 (FITC), Aurora A (TRITC) and DAPI. The interval of Z-stack is 1  $\mu\text{m}$ . The scale bar is 10  $\mu\text{m}$ .

# Figure S9



## Supplementary Figure S9. Aurora A orchestrates microtubule dynamics.

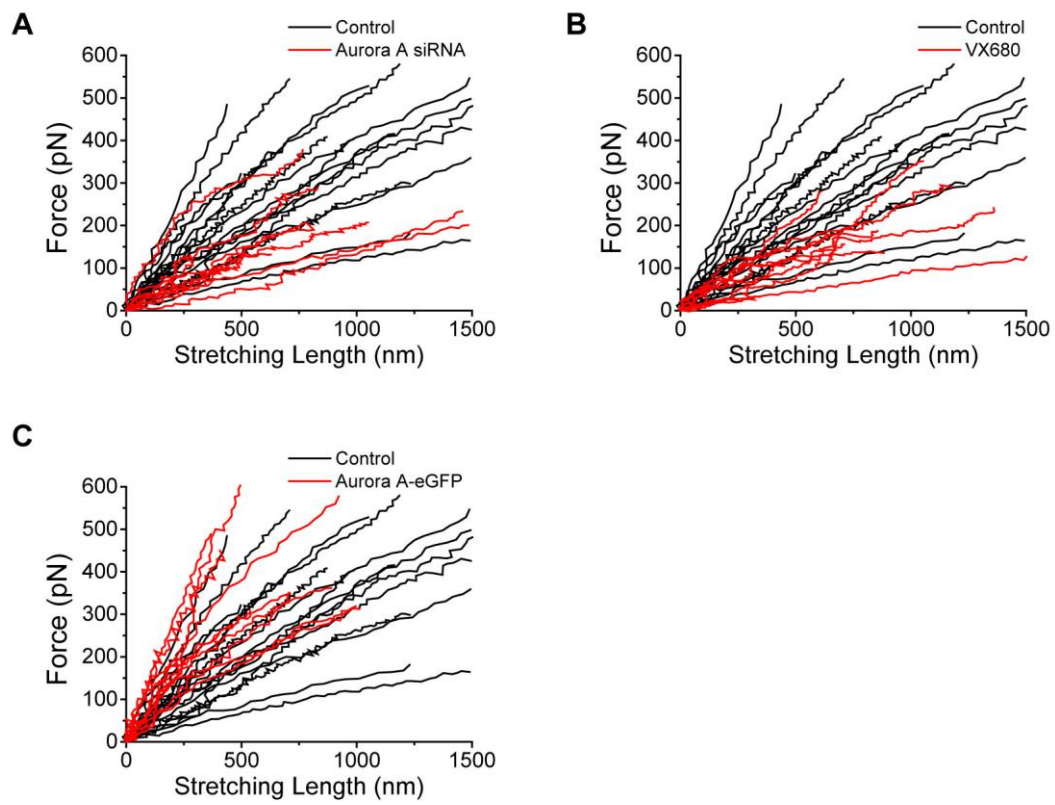
(A) Images of MCAK-GFP stable cells before and after treatment of VX680 or DMSO. Images are trails (TRL) of 31 frames with 3-sec intervals. The scale bars are 15  $\mu\text{m}$ . (See also Video 11-14).

(B) Images of MCAK-GFP stable cells transiently transfected with scramble or Aurora A siRNAs. Images are trails (TRL) of 31 frames with 3-sec intervals. The scale bars are 15  $\mu\text{m}$ . (See also Video 15-16).

(C and D) Histograms of MT plus-ends comet lengths, or fluorescence intensity ratios at the growing MT tips and in surrounding cytoplasm of indicated cells (for each condition, at least 900 microtubule plus-ends in 30 cells from three independent experiments were analyzed); error bars indicate SD.



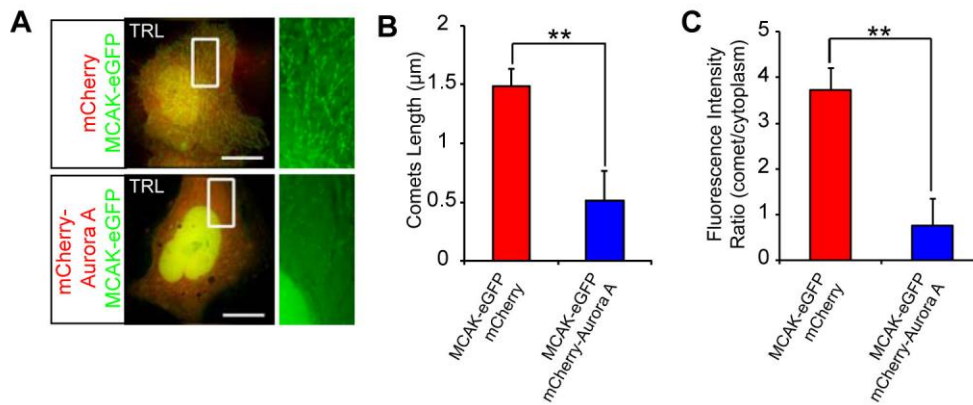
**Figure S10**



**Supplementary Figure S10. Cell rigidity analyses by optical trap system.**

(A-C) Related to Figure 5D. Trapping force  $F$  versus stretching extension  $l$  of cells in experiment or control groups.

**Figure S11**

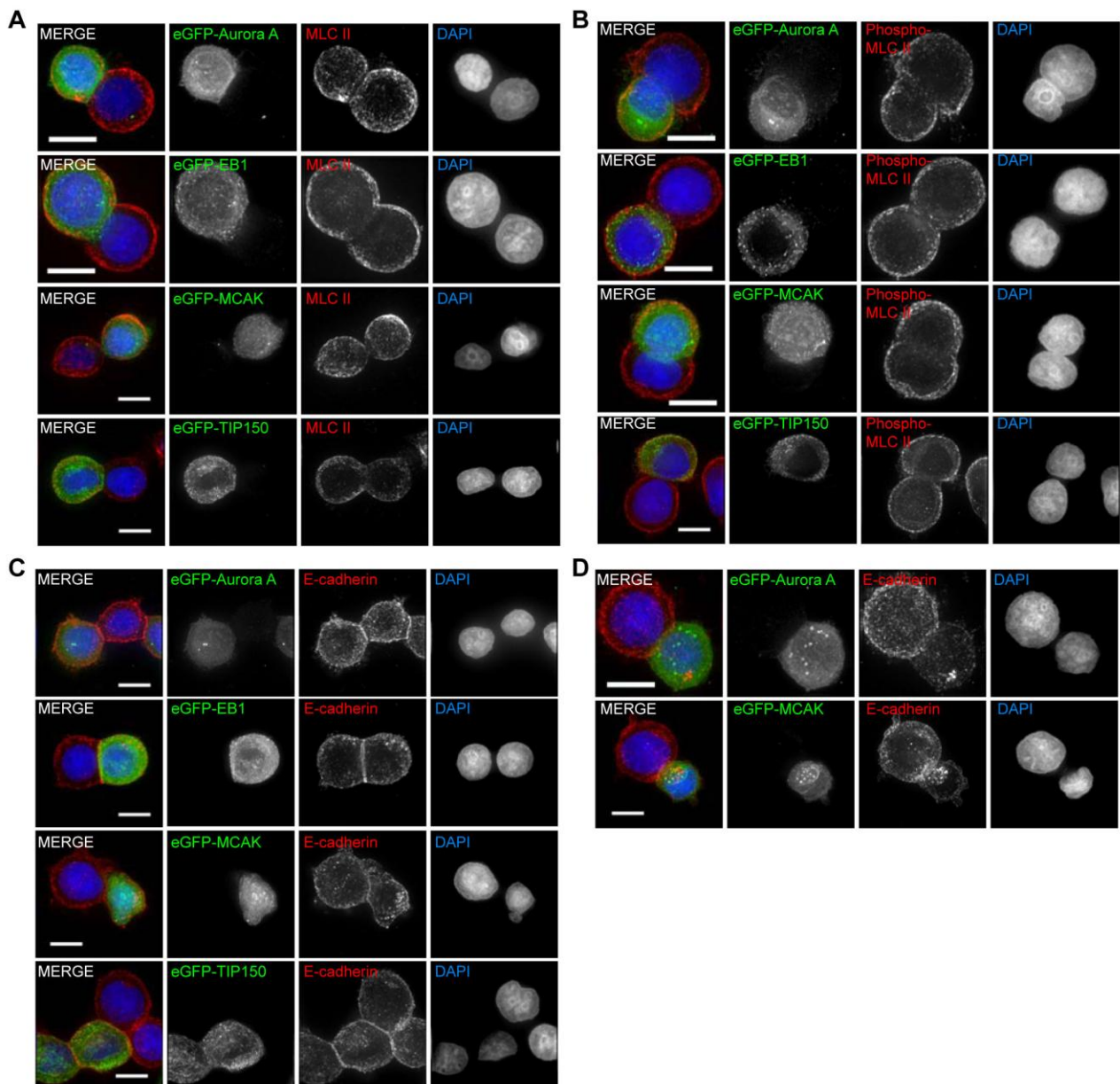


**Supplementary Figure S11. Over-expressing Aurora A disrupted the MT plus-end tracking of MCAK-eGFP.**

(A) Images of MCAK-GFP stable cells transiently transfected with mCherry or mCherry-Aurora A. Images are trails (TRL) of 31 frames with 3-sec intervals. The scale bars are 15 μm. (See also Video 17-18).

(B and C) Histograms of MT plus-ends comet lengths, or fluorescence intensity ratios at the growing MT tips and in surrounding cytoplasm of indicated cells (for each condition, at least 900 microtubule plus-ends in 30 cells from three independent experiments were analyzed); error bars indicate SD. \*\*,  $p < 0.01$ .

**Figure S12**



**Supplementary Figure S12. Examine of Myosin Light Chain 2, phosphor-Myosin Light Chain 2 and E-cadherin in cells over-expressing Aurora A, EB1, MCAK and TIP150.**

(A) Immunofluorescence staining of Myosin Light Chain 2 in cells over-expressing Aurora A, EB1, MCAK and TIP150 shows over-expressing of the proteins do not disturb Myosin Light Chain 2 localization obviously. The scale bars are 10  $\mu$ m.

(B) Immunofluorescence staining of phosphor-Myosin Light Chain 2 in cells over-expressing Aurora A, EB1, MCAK and TIP150 shows over-expressing of the proteins have no significant influence on Myosin activation. The scale bars are 10  $\mu$ m.

(C) Immunofluorescence staining of E-cadherin shows normally over-expression of Aurora A, EB1, MCAK and TIP150 in cells has no significant influence on E-cadherin distribution. The scale bars are 10  $\mu$ m.

(D) Immunofluorescence staining of E-cadherin shows extremely over-expression of Aurora A and MCAK in cells has obvious influence on E-cadherin distribution. The scale bars are 10  $\mu$ m.