## Supplemental material

## JCB



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Figure S1. **Overexpression of CEP120 induces the formation of extra long centrioles as well as atypical centriole amplification.** (A–C) CEP120-Myc-inducible cells (clone #5) were treated with (+Tet) or without (-Tet) Tet and analyzed by immunoblotting (A) or immunofluorescence staining (B) with the indicated antibodies. The percentage of cells with elongated centrioles or nonelongated centrioles in the presence or absence of Tet (48 h) are shown (C). Error bars represent means ± SD of 100 cells from three independent experiments. (D and E) CEP120-Myc-inducible cells were treated without (-Tet) or with (+Tet) Tet for 48 h and analyzed by confocal fluorescence microscopy using the indicated antibodies (D). DNA was counterstained with DAPI. (E) The percentage of amplified centrioles induced by excess CEP120-Myc. Error bars represent means ± SD of 100 cells from three independent experiments.



Figure S2. **CEP120-induced centriole elongation reveals a biphasic pattern with slow growth in S phase and fast growth in G2 phase.** (A–D) The timing of centriole elongation in CEP120-Myc-inducible cells during the cell cycle. CEP120-Myc-inducible cells were treated as described in C. At the indicated times after removal of aphidicolin, cells were analyzed by confocal immunofluorescence microscopy using the indicated antibodies (A) and by FACS (B). The lengths of centrioles (labeled with Ac-Tub) were quantified at the indicated times (D; n = 30 centrioles). Error bars represent means  $\pm$  SD from three independent experiments. The mother centrioles were marked by ODF2 staining. (E) CEP120 is a cell cycle–regulated protein. HeLa cells released from a double-thymidine block were analyzed at the indicated time points by immunoblotting using the indicated antibodies (top) and by FACS (bottom).



Figure S3. **Examining the centriolar localization of CPAP, CEP120, and hSAS-6 in siCEP120- or siCPAP-depleted cells.** (A) Protocol used to analyze siCEP120- or siCPAP-treated effects in U2OS cells. U2OS cells were transfected with siControl, siCEP120 (B–D), or siCPAP (E and F). 48 h after transfection, the cells were treated with aphidicolin for another day. 5 h after the removal of aphidicolin, the cells were analyzed by confocal fluorescence microscopy and immunoblotting using the indicated antibodies. Shown are quantifications of the centriolar signals of CPAP (C) and hSAS-6 (D) in siCEP120-treated cells, and the centriolar signals of CEP120 (E) and hSAS-6 (F) in siCPAP-treated cells. Error bars represent means ± SD of 100 cells from three independent experiments.