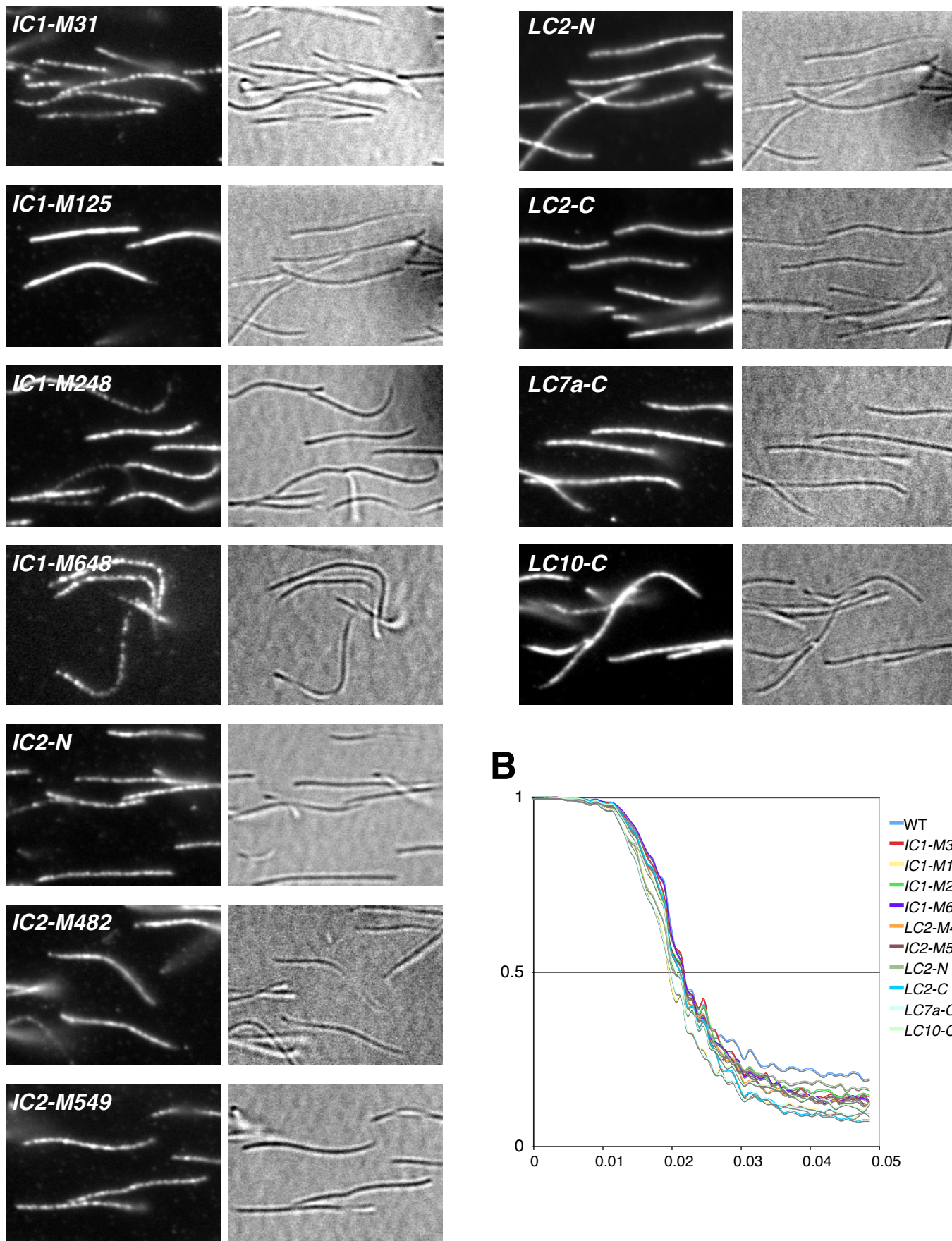
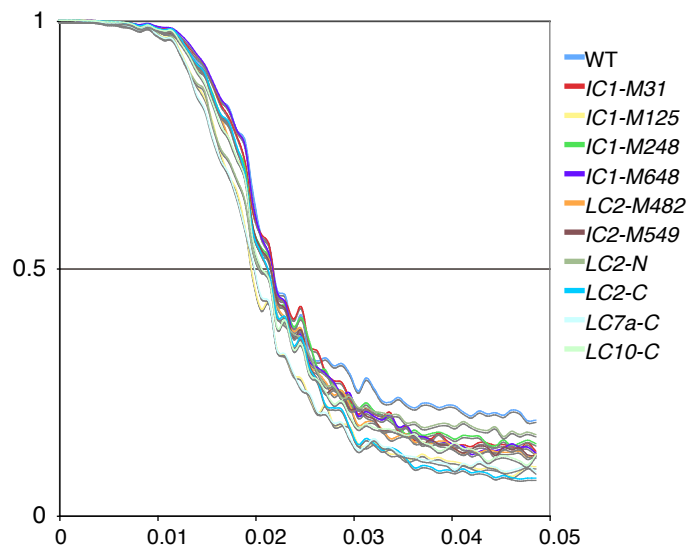


Supplemental Materials

Molecular Biology of the Cell

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A**B****Figure S1**

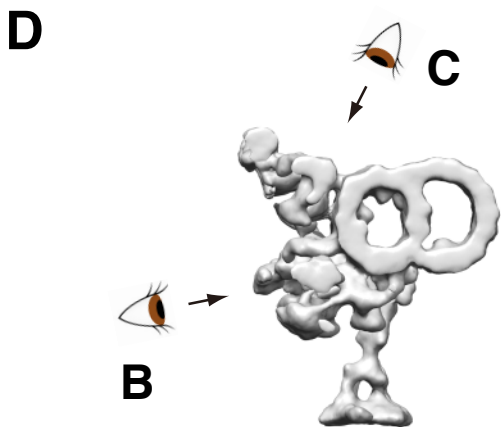
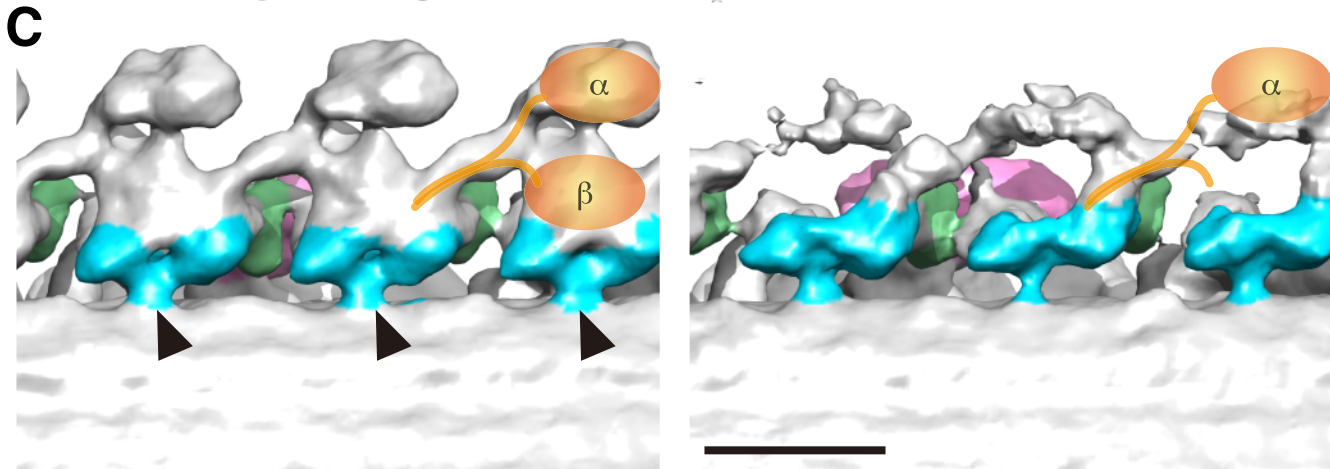
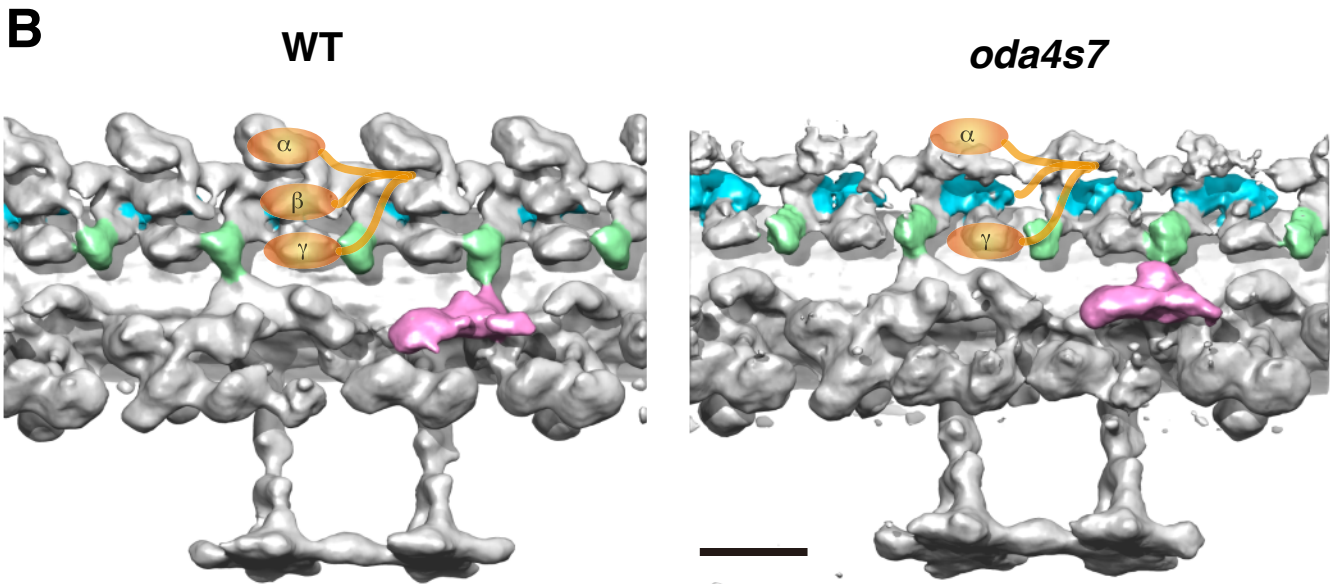
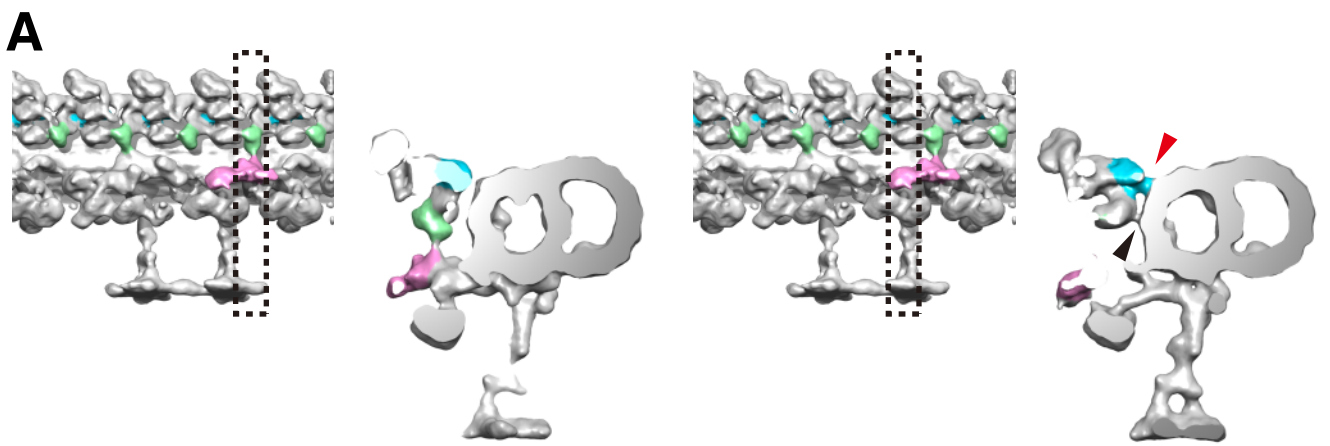


Figure S2

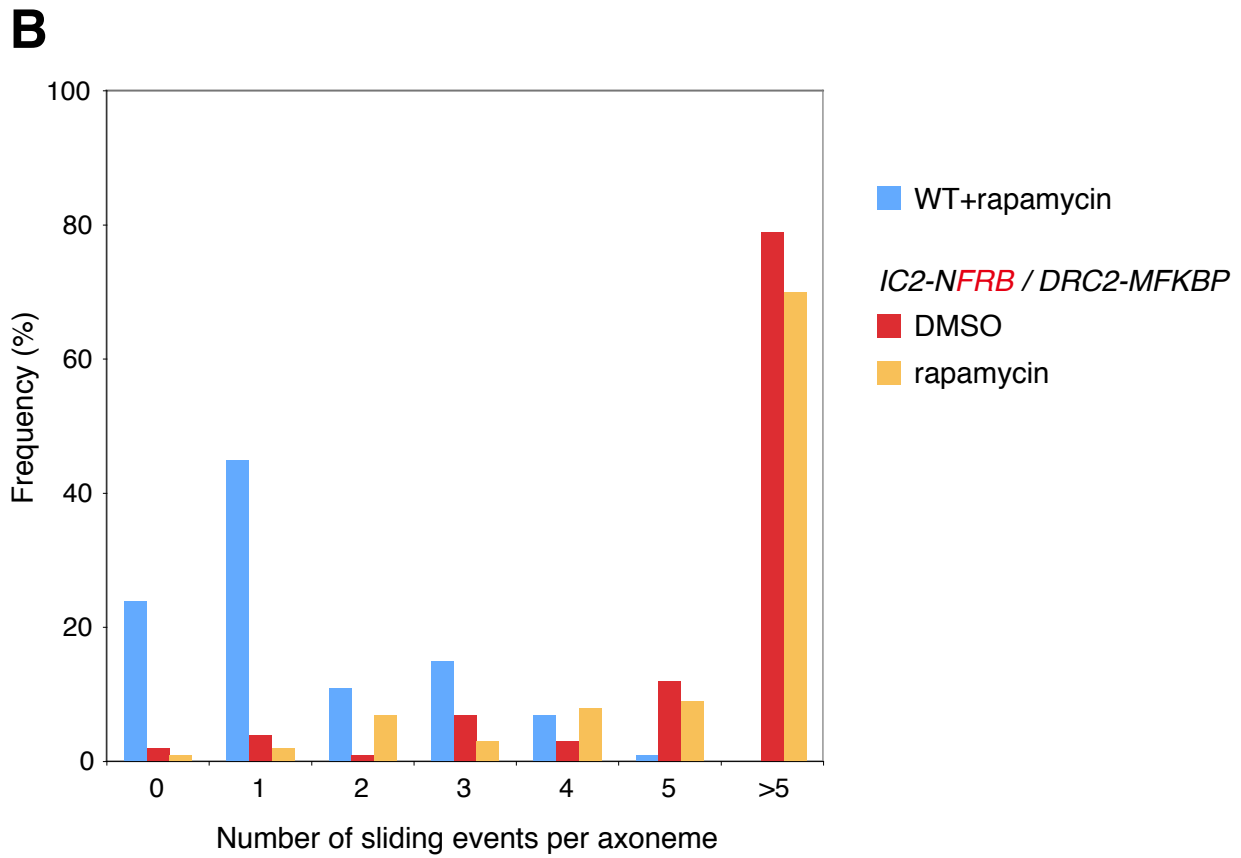
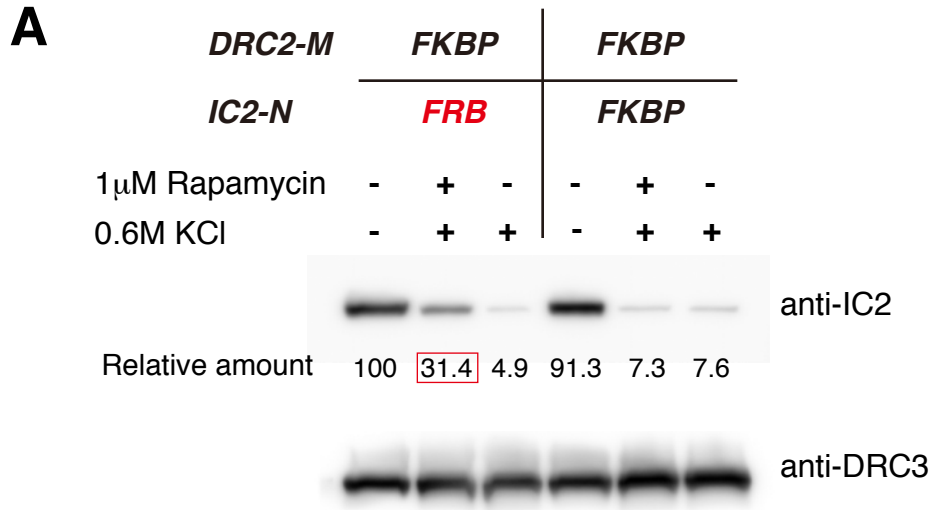


Figure S3

Figure S1

Characterization of *IC-BCCP* and *LC-BCCP* mutants. (A) Immunofluorescence images of BCCP-tagged axonemes. Biotinylation of BCCP tags were confirmed by streptavidin-Alexa 546 staining (left). Right: DIC images. (B) Fourier shell correlation curves of averaged tomograms used in this study. Intersection between each curve and horizontal line at 0.5 was taken as the effective resolution. Effective resolution of each structure was 4.5~ 5.0 nm. WT:wild-type.

Figure S2

3D structure of *oda4-s7* DMT. (A) Slabs of tomograms, showing DMT-attachment sites. (left) There is not apparent connection between ODA-Beak (green) and DMT. (right) There are two possible DMT-attachment sites projecting from γ HC (black arrowhead) and the outermost region of ODA (blue, red arrowhead). (B, C) DMT structure of *oda4s7* mutant. Diagrams in orange indicate approximate positions of the HCs. Green: ODA-Beak; pink:

N-DRC; blue: DMT-binding complex. Arrowheads indicate DMT-binding site of ODA.

Bars = 20 nm. (D) Direction of views in B and C.

Figure S3

Additional data for Figure 3 and 4. (A) Related to Figure 3D and 3E. Immunoblots of

axonemal proteins with and without rapamycin-treatment and KCl-extraction. With

rapamycin-treatment, about 30% of ODA remained attached to axonemes after

KCl-extraction. (B) Related to Figure 4D. Histograms of the sliding events per axoneme.

Sliding disintegration was enhanced regardless of rapamycin-treatment.