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Molecular crowding at microtubule plus-ends acts as a physical barrier to microtubule sliding for the organization of stable anti-parallel overlaps by PRC1 and Kif4A SITARA WIJERATNE, RADHIKA SUBRAMANIAN, Harvard Medical School — The relative sliding of microtubules by motor proteins is important for the organization of specialized cellular microtubule networks. In cells, sliding filaments are likely to encounter crowded regions of microtubules, such as the plus-ends, which are densely occupied by motor and non-motor proteins. How molecular crowding impacts microtubule sliding is not well understood. Here, we reconstitute the collective activities of the non-motor protein PRC1 and the motor protein Kif4A on anti-parallel microtubules to address this question. We find that the accumulation of PRC1 and Kif4A at microtubule-plus ends ('end-tags') can act as a physical barrier to Kif4A-mediated microtubule sliding. This enables the formation of stable microtubule overlaps that persist even after the deactivation of the motor protein. Our data suggest that while end-tags stabilize anti-parallel overlaps by inhibiting relative sliding, they permit the remodeling of the microtubule bundles by external forces, as may be required for the reorganization of microtubule networks during dynamic cellular processes.

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