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Push me if you can: role of antiparallel microtubule sliding in anaphase spindle elongation

The nature of forces driving chromosome segregation in human mitotic spindle remains one of the most challenging questions in the field. Although different microtubule populations have been well characterized in anaphase, their contribution to segregation remains unclear. Recently, it was shown that antiparallel interpolar microtubules in central spindle are strongly crosslinked with kinetochore fibers and are able to slide apart in order to separate kinetochores and spindle poles [1]. However, despite extensive knowledge on sliding proteins *in vitro* and in spindle formation, it remains unknown what proteins generate and transmit sliding forces in anaphase. Here we show that spindle elongation requires combined action of the antiparallel passive crosslinker PRC1 and kinesin-5 (Eg5) motor. Combined depletion of PRC1 and inhibition of Eg5 by STLC prevents spindle poles to elongate when Eg5 is inhibited in metaphase or stops spindle elongation when inhibited in anaphase in RPE-1 cells. However, poleward movement of kinetochores continues, though at a slower rate, when compared to control cells. Interestingly, we found that depletion/inhibition of neither these proteins individually or other candidate proteins (kinesin-12, kinesin-6 and kinesin-4) caused reduced rates of spindle elongation. Our results indicate that sliding of microtubules in the spindle midzone is controlled by combined action of Eg5 and PRC1 and is essential for spindle pole separation in human cells.

[1] Vukusic K, Buda R et al., *Dev Cell.* 2017.

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