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Comment on “A Centrosome-Independent Role for γ-TuRC Proteins in the Spindle Assembly Checkpoint”

Stephen S. Taylor1,*, Kevin G. Hardwick2, Kenneth E. Sawin2, Sue Biggins3, Simonetta Piatti4, Alexey Khodjakov5, Conly L. Rieder5, Edward D. Salmon6, and Andrea Musacchio7,*

1Faculty of Life Sciences, University of Manchester, Manchester, UK
2Wellcome Trust Centre for Cell Biology, Institute of Cell Biology, University of Edinburgh, Edinburgh, UK
3Fred Hutchinson Cancer Research Center, Seattle, WA, USA
4Department of Biotechnology and Bioscience, University of Milan-Bicocca, Milan, Italy
5Division of Molecular Medicine, Wadsworth Center, New York State Department of Health, Albany, New York, USA
6Department of Biology, University of North Carolina at Chapel Hill, Chapel Hill, NC, USA
7Department of Experimental Oncology, European Institute of Oncology, Milan, Italy

Abstract

Müller et al. (Reports, 27 October 2006, p. 654) showed that inhibition of the γ-tubulin ring complex (γ-TuRC) activates the spindle assembly checkpoint (SAC), which led them to suggest that γ-TuRC proteins play molecular roles in SAC activation. Because γ-TuRC inhibition leads to pleiotropic spindle defects, which are well known to activate kinetochore-derived checkpoint signaling, we believe that this conclusion is premature.
However, in contrast to the authors’ conclusion that γ-tubulin plays a direct role in the SAC, we favor the simple explanation, for two reasons. First, the presence of abundant microtubule arrays is not sufficient to inactivate the SAC. Second, although chromosomes may appear “amphitelic-like,” this does not guarantee that all the kinetochores are stably attached to MTs. The following example illustrates these latter two points. Meta-phase cells treated with low doses of taxol or cooled to 23°C display normal bipolar MT arrays in which almost all the kinetochores are attached to microtubules from opposite spindle poles (i.e., “amphitelic-like”), yet in both cases, a SAC-dependent mitotic delay ensues (11,12). Indeed, because a single unattached kinetochore is sufficient to activate the SAC (13), the simplest explanation for the observations of Müller et al. is that inhibition of γ-TuRC perturbs spindle assembly and/or MT dynamics, which in turn results in inadequate levels of MT attachment and/or tension at all kinetochores, thereby activating the SAC and delaying mitotic progression.

References