Année de publication : 2001

E Wojcik, R Basto, M Serr, F Scaërou, R Karess, T Hays (2001 Nov 21)
Kinetochore dynein: its dynamics and role in the transport of the Rough deal checkpoint protein.
Nature cell biology : 1001-7

Résumé

We describe the dynamics of kinetochore dynein-dynactin in living Drosophila embryos and examine the effect of mutant dynein on the metaphase checkpoint. A functional conjugate of dynamitin with green fluorescent protein accumulates rapidly at prometaphase kinetochores, and subsequently migrates off kinetochores towards the poles during late prometaphase and metaphase. This behaviour is seen for several metaphase checkpoint proteins, including Rough deal (Rod). In neuroblasts, hypomorphic dynein mutants accumulate in metaphase and block the normal redistribution of Rod from kinetochores to microtubules. By transporting checkpoint proteins away from correctly attached kinetochores, dynein might contribute to shutting off the metaphase checkpoint, allowing anaphase to ensue.

R Basto, R Gomes, R E Karess (2001 Jan 9)
Rough deal and Zw10 are required for the metaphase checkpoint in Drosophila.
Nature cell biology : 939-43

Résumé

The metaphase-anaphase transition during mitosis is carefully regulated in order to assure high-fidelity transmission of genetic information to the daughter cells. A surveillance mechanism known as the metaphase checkpoint (or spindle-assembly checkpoint) monitors the attachment of kinetochores to the spindle microtubules, and inhibits anaphase onset until all chromosomes have achieved a proper bipolar orientation on the spindle. Defects in this checkpoint lead to premature anaphase onset, and consequently to greatly increased rates of aneuploidy. Here we show that the Drosophila kinetochore components Rough deal (Rod) and Zeste-White 10 (Zw10) are required for the proper functioning of the metaphase checkpoint in flies. Drosophila cells lacking either ROD or Zw10 exhibit a phenotype that is similar to that of bub1 mutants – they do not arrest in metaphase in response to spindle damage, but instead separate sister chromatids, degrade cyclin B and exit mitosis. These are the first checkpoint components to be identified that do not have obvious homologues in budding yeast.