IN BRIEF

Putting the cenH3 in the Centromere: Arabidopsis KINETOCHORE NULL2 Acts Upstream of cenH3 Deposition

Imagine yourself as a chromatin-crawling nano-robot—Your mission: to explore the Arabidopsis thaliana centromere. If you ventured from the euchromatin, through the heterochromatin, into the centromere, you would find yourself in a strange place. Even the histones are different, with the familiar H3 replaced by the centromere-specific histone cenH3 (reviewed in De Rop et al., 2012). Mission control requests that you report on the topology of centromere chromatin, as centromeric nucleosomes may be wound in a right-handed spiral, in contrast with the euchromatic left-handed winding. Now crawl back to a safe distance and observe cell division. In the rest of the genome, you saw histones deposited at S phase, but, in Arabidopsis, you will observe cenH3 deposition into centromere nucleosomes at G2 (Lermontova et al., 2006).

Despite the lack of nano-robots, researchers have long recognized the central importance of cenH3 for centromere specification. The initiation, deposition, and maintenance of cenH3 localization require specific structural differences between H3 and cenH3 and interactions with chaperones and regulatory proteins (reviewed in De Rop et al., 2012). For example, work in fungal and animal systems recognized localization of KINETOCHORE NULL2 (KNL2) to the centromere as the earliest upstream event in deposition of cenH3. To examine cenH3 deposition in plants, Lermontova et al. (pages 2013) isolated an Arabidopsis homolog of KNL2. As expected, Arabidopsis KNL2 localized with cenH3 at the chromosome centers in interphase and the centromeres in mitosis (except in metaphase through the middle of anaphase). This localization is similar to fission yeast (Schizosaccharomyces pombe) KNL2 but different from human KNL2. In a serendipitous discovery, difficulties in finding a signal for fluorescent-tagged KNL2 led to the identification of putative ubiquitination sites in the KNL2 N terminus. Indeed, tagged KNL2 could be visualized only after treatment with the proteasome inhibitor MG115 or deletion of the N-terminal sites, indicating an important role for ubiquitin-mediated proteolysis in regulation of KNL2.

The authors also examined the phenotype of knl2 knockout mutants, which showed less cenH3 at the centromeres. This affected mitosis and meiosis, producing lagging chromosomes, anaphase bridges, and micronuclei (mn), indicating aberrant chromosome segregation. (Reprinted from Lermontova et al. [2013], Figure 4.)

The knl2 mutants show defects in mitosis and meiosis. Mitotic anaphase chromosomes (top) and meiotic pollen tetrads (bottom) in the wild type and knl2 mutants. The mutants show lagging chromosomes, bridges, and micronuclei (mn), indicating aberrant chromosome segregation. (Reprinted from Lermontova et al. [2013], Figure 4.)

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REFERENCES


