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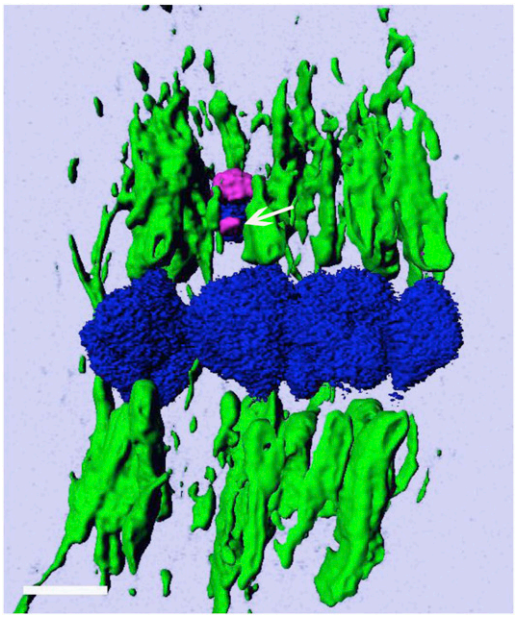
IN BRIEF

Flipping the Centromere Switch: Reactivation of a Dormant Centromere in Maize

For centromeres, one per chromosome is the magic number; any more, or any fewer, and things get messy. During cell division, a chromosome with no centromere can be lost and a chromosome with two centromeres can be pulled apart. Thus, plasticity in centromere specification is subject to strong selection. The centromeres of many species, including mammals and many plants (reviewed in Ma et al., 2007), share a similar structure containing retroelements and large arrays of tandem centromere repeats. These repeats evolve rapidly such that they show little sequence homology in related taxa but do have a similar repeat length (156 bp in maize). However, presence of the centromere repeat does not guarantee formation of a centromere, and neocentromeres can be established in areas of the genome that do not contain the centromere repeats, showing a clear epigenetic component to centromere specification (reviewed in Allshire and Karpen, 2008). Interestingly, mammalian artificial chromosome studies indicate that the underlying sequence is also important for centromere specification (Grimes et al., 2002). How do structural elements and epigenetic controls allow for plasticity of centromere specification?

The maize B chromosome is a nonessential chromosome with a unique repeated sequence (in addition to the maize centromere repeat and retroelements) in its centromere, making it an ideal experimental system to study centromere function. To examine centromere specification, **Han et al. (pages 1929–1939)** produced a B chromosome derivative containing two centromeres, one large and one small. During meiosis, this dicentric chromosome creates bridges and breaks, making its inheritance unstable. They isolated a stable derivative, called Dic-15, in which the smaller of the two centromeres has become inactive. The inactive centromere no longer stains for molecular markers of centromere activity, such as the centromere-specific histone H3 variant (CENH3), and no longer attracts microtubules at metaphase (see figure). This centromere can be inherited in its dormant state and attached to the active centromere.

In a striking finding, shutting off the small centromere of Dic-15 is not the end of the story. Because of the structure of Dic-15, the inactive centromere can be separated from the active centromere by intrachromosomal recombination, allowing the isolation of derivatives carrying only two copies of the smaller centromere. These derivatives can enter metaphase, attract tubulin fibers, and stain positive for CENH3 and other markers of active centromeres, indicating that the dormant small centromere has become reactivated. However, in the recovered cases, only one of the two small centromeres in the dicentric derivative is active, so the derivative is as stable as Dic-15. Reactivation of a centromere at the same location indicates that, in addition to epigenetic mechanisms that specify centromere activity, there may also be structural aspects of the underlying sequence that make a genomic site better suited for centromere function.



The larger, active centromere of Dic15 attaches to tubulin, but the smaller, inactive centromere (arrow) does not. Meiosis metaphase I is shown, with the B chromosome centromeres in magenta, DNA in blue, and tubulin in green. Bar = 10 μm.

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