Centromeres cross over, a lot

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Recombination at centromeres is higher than anywhere else on the chromosome, even though methyltransferases do their best to prevent it, say Jaco et al., as published in the June 16 issue of the *Journal of Cell Biology*.

Centromeric recombination has been hard to study because the DNA at centromeres is so repetitive—it's hard to see when a segment has switched chromatids. Jaco et al. have now addressed this challenge by using CO-FISH (chromosome orientation fluorescence in situ hybridization).

After replication, the two new strands are digested away, leaving the two old strands. Because the strands are complementary in sequence, they can be tagged with strand-specific fluorescent probes. Using just one probe, only one chromatid would show a signal if no recombination had occurred.

Instead, the authors found that both chromatids fluoresced. And not just at one point—on average, the authors counted, centromeres had undergone 15 recombination events. This is about six times the rate of recombination as that seen for an equal length of telomeric DNA, and 175 times the rate for genomic DNA as a whole.

Telomeric recombination is inhibited by protein complexes called shelterins and by DNA methylation. The centromere has no shelterin, but it is methylated. Knockdown of DNA methyltransferases increased recombination at the centromere by about 50%, and decreased

centromere length, possibly because of misalignment between repeated segments during recombination, a common problem with repetitive DNA. How methylation limits recombination, and why centromeres didn't lengthen as well as shorten, are unknown.

Their repetitive structure makes centromeres recombinogenic by nature, and the authors suggest that epigenetic regulation may ensure the continued stability of essential binding regions for proteins that link to the centromere.

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