The nuclear periphery acts as a regulator of recombinatorial potential

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Repetitive regions of the genome are prone to recombination and thus can lead to genome instability, but this same property may also support molecular diversity and adaptation. Little is known about cellular mechanisms that may control the recombinatorial potential of repetitive DNA. Since many repetitive DNA elements are preferentially associated with the inner nuclear membrane, we hypothesized that their association with the nuclear periphery could serve as an input to genome stability by regulating the likelihood that recombination occurs. To test this concept, we examined recombination in the repeat-rich cell surface adhesin genes of the fission yeast, *S. pombe*. This gene family is repetitive intragenically (repeat modules encode a repetitive peptide domain) and intergenically (related genes reside at distinct genomic loci). We show that the adhesin genes are found associated with the nuclear envelope; this association is dependent on the function of proteins bound to proximal transposons and LTRs. Using assays designed to measure intragenic and intergenic recombination, we found that recombination rates are sensitive to internal repeat number but not transcriptional status. Further, disrupting their nuclear envelope tethers weakened the association of adhesin genes with the nuclear periphery and led to an increase in recombination rate. Release from the nuclear periphery and the concomitant increase in recombination could also be recapitulated in response to specific environmental inputs. Our data suggest that the recombinatorial potential of repetitive elements can be tuned by their nuclear compartmentalization, which may provide a mechanism to balance genome integrity with adaptation in response to a changing environment. This fundamental mechanism may also contribute to pathogen evasion of the host immune system.