ERRATUM

How chromosomes find their ‘soul mate’

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It has come to our attention that Dion *et al.* published simultaneously a related paper to that discussed in this In Brief, showing that damaged chromosomes increase their mobility to facilitate homologue pairing. In this study, Dion *et al.* tracked the mobility of damaged loci by tagging Rad52, a factor required for homologous recombination, with yellow fluorescent protein. They saw that mobility was altered in response to damage specifically in the broken chromosome but not at another undamaged locus. This increased mobility required the recombination proteins Rad51 and Rad54, the checkpoint factor Rad9 and activation of the ATR kinase, Mec1. Increased mobility of damaged loci was not common to all types of damage, as DNA damage produced by camptothecin or by a protein-bound single-stranded nick did not trigger changes in DNA mobility. The authors also showed that recombination intermediates form more slowly in cells lacking Rad9, supporting the idea that this induced mobility is important for productive homologous recombination. We apologize to the authors of this paper for this omission.

ORIGINAL RESEARCH PAPER Dion, V. *et al.* Increased mobility of double-strand breaks requires Mec1, Rad9 and the homologous recombination machinery. *Nature Cell Biol.* **14**, 502–509 (2012)