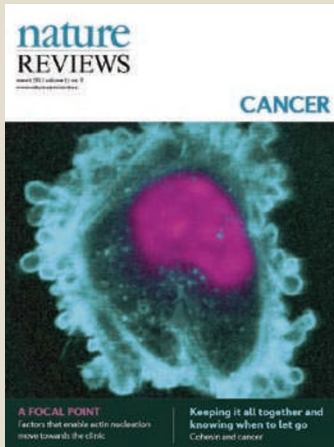




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► **COVER:** 'Get it together' by Simon Fenwick, inspired by the Review on p177. Image courtesy of R. Grosse.



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The elixir of eternal youth could well be long telomeres keeping chromosomes, and hence cells, in good shape thanks to efficient telomerase. However, it is not always convenient to keep cells healthy and youthful, especially if these cells are cancerous.

As María A. Blasco and Paula Martínez discuss on page 161, telomerase seems to have many different roles that are not always associated with telomere maintenance but that can also be linked to cancer. There are still many unanswered questions about these roles and how they can be regulated in cancer therapy, but while these questions are being addressed, perhaps keeping telomerase away from elongating dysfunctional telomeres that lead to genomic instability might be one way to go.

Like dysfunctional telomerase in cancer cells, defects in the cohesin complex — which regulates chromosome separation in mitosis — induces chromosome and genetic instability. Mutations in cohesins and their regulators have been found in cancer, but how reasonable would it be to target cohesin components for cancer therapy? This subject is discussed by Michael J. McKay and colleagues on page 199.

Finally, on page 218, Damien Brégeon and Paul W. Doetsch explain how mutant proteins that drive tumorigenesis do not necessarily have to originate from aberrant chromosomes or an unstable genome. During translation, the wrong amino acid can be incorporated opposite a DNA lesion, and this can generate mutant proteins with the potential to change the cellular phenotype. Thanks to new technologies that allow the sequencing of mRNAs from different populations of cells, it will be possible to determine how common these mechanisms are and their role in tumour development.

Articles on the importance of keeping genomic instability in check and cells in tip top condition will appear in our '[Genomic instability in cancer](#)' series.

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