

## IN BRIEF

 **TECHNIQUE****Live imaging of sugars**

Despite advances in imaging, visualizing non-genetically encoded molecules such as glycans has been challenging. Attreed *et al.* have developed a technique that allows the visualization of the complex modification patterns of heparan sulphates — polysaccharides that interact with and modify the function of many proteins in their glycosylated, proteoglycan form. The authors used single chain variable fragment (scFv) antibodies that recognize distinctly modified forms of heparan sulphates. These were fused with green fluorescent protein and a secretion signal to ensure that the proteins were released from the cell and bound their targets, and the full constructs were expressed in scavenger cells in *Caenorhabditis elegans*. The authors were able to label distinct heparan sulphate modification patterns with specificity *in vivo*. As long as specific scFv antibodies are available, this approach could be used for the live imaging of other molecules, such as lipids, as well as of post-translationally modified proteins.

**ORIGINAL RESEARCH PAPER** Attreed, M. *et al.* Direct visualization of specifically modified extracellular glycans in living animals. *Nature Methods* 1 Apr 2012 (doi:10.1038/nmeth.1945)

 **DNA REPAIR****How chromosomes find their ‘soul mate’**

In eukaryotes, DNA damage is frequently repaired by homologous recombination. In this study the authors asked whether there are any changes in chromosome dynamics that facilitate the search for a homologous sequence, using budding yeast as a model. They showed that induction of a DNA double-strand break (DSB) increases the mobility of both the broken chromosome and its unbroken homologue, allowing the chromosomes to explore a larger area in the nucleus. Interestingly, induction of a DSB also increases the mobility of unbroken, non-homologous chromosomes, albeit at lower levels. These changes in chromosome dynamics and the consequent homologue pairing depend on two repair proteins, the endonuclease Sae2 and the recombinase Rad51. The authors propose a model in which DSBs increase chromosome mobility, thereby facilitating homologue pairing by expanding the nuclear area that chromosomes explore.

**ORIGINAL RESEARCH PAPER** Miné-Hattab, J. & Rothstein, R. Increased chromosome mobility facilitates homology search during recombination. *Nature Cell Biol.* 8 Apr 2012 (doi:10.1038/ncb2472)

 **GENE EXPRESSION****Keeping p53 targets quiet**

The histone methyltransferase SUV39H1 trimethylates histone 3 at Lys9 (H3K9me3), which represses gene expression. Here, the authors find that SUV39H1 is targeted by the tumour suppressor p53. Specifically, the authors observed that p53 represses SUV39H1 expression through two of its targets, p21 (which represses SUV39H1 at the transcriptional level) and the E3 ubiquitin ligase MDM2 (which promotes SUV39H1 degradation). So why does p53 target SUV39H1? The authors found that the H3K9me3 mark was present on the promoters of p53 target genes but that it decreased after p53 induction. Moreover, overexpression of SUV39H1 inhibited p53-dependent cell fate decisions — cell cycle arrest and apoptosis — and SUV39H1 silencing enhanced p53-dependent apoptosis. Together, these findings reveal that p53 represses SUV39H1 to promote the expression of its target genes.

**ORIGINAL RESEARCH PAPER** Mungamuri, S. K. *et al.* p53-mediated heterochromatin reorganization regulates its cell fate decisions. *Nature Struct. Mol. Biol.* 1 Apr 2012 (doi:10.1038/nsmb.2271)