New repair mechanism for DNA breaks

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Chromosomal breaks are the most harmful damage for cells. If they are not repaired, they block the duplication and segregation of chromosomes, stop the growth cycle and cause cell death. These breaks appear frequently in tumor cells and are produced spontaneously during the replication of genetic material. To be able to repair this damage in the genetic material, the cell transfers the information from the intact daughter copy to the broken copy, which is known as recombination of sister chromatids.

In a project recently published by *Nature Communications*, researchers from the University of Seville and the Andalusian Centre of Molecular Biology and Regenerative Medicine (CABIMER) have identified new factors that are necessary for the repair of these breaks. These factors, in contrast with those already known, only affect the repair between sister chromatids of breaks that have arisen during chromosome duplication. Specifically, they are proteins that modify 'histones," which are the basic proteins that form the structure of the chromosomes.

The research group has shown that the inability to repair breaks in cells lacking these proteins derives from a deficient cohesin load. These are the proteins that keep the sister chromatids paired and together until their segregation in meiosis. With the lessening of cohesion between the chromatids, the repair is defective which leave many breaks unrepaired and increases the chromosomal reorganisation.

The project carried out on the organism model Saccharomyces has identified new factors involved on the maintenance of genome integrity and a new mechanism with which the cohesin load in the chromosomes can be regulated, which could be of great value for deciphering the multiple mechanisms responsible for genome instability in the tumor cells and different neurodegenerative pathologies.

More information: Pedro Ortega et al. Rpd3L and Hda1 histone deacetylases facilitate repair of broken forks by promoting sister chromatid cohesion, *Nature Communications* (2019). DOI: 10.1038/s41467-019-13210-5

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