Abstract

DNA double-strand break (DSB) is a dangerous type of DNA damage, but it also serves in controlled increase of genetic variability. The two major DSB repair pathways are homologous recombination (HR) using homologous sequences and non-homologous end joining (C-NHEJ). Two model plants *Arabidopsis thaliana* (*Arabidopsis*) and the moss *Physcomitrella patens* (*Physcomitrella*) differ in DSB repair strategies. *Arabidopsis* uses C-NHEJ, however *Physcomitrella* prefers HR. These plant models are compared on the basis of measurement of DSB and single strand breaks (SSB) repair by comet assay.

The half-life of the first rapid phase of the DSB repair is about 5 minutes in both plant species. Although the C-NHEJ is considered as the main DSB repair pathway in *Arabidopsis*, rapid repair is independent of AtLIG4 and AtKu80, suggesting the existence of the effective backup non-homologous repair pathways (A-NHEJ). In *Physcomitrella*, the rapid DBS repair dominates in mitotically active cells and is also independent of PpLIG4. Conversely, PpLIG4 is surprisingly involved in the repair of the DNA alkylation damage.

An essential DNA ligase of the rapid DSB repair pathway in *Arabidopsis* is the replication ligase AtLIG1, which is also responsible for the alkylation DNA damage repair, and thus represents a functional homolog of LIG3. The rapid DSB repair is totally dependent on structural maintenance of chromosomes protein (SMC) AtSMC6b. A slight defect in the DSB repair is also observed in mutant of cohesin subunit AtRAD21 and AtGMI1, a newly identified member of a SMC-hinge domain-containing protein family (SMCHD). The role of AtSMC6b, AtRAD21 and AtGMI1 in the DSB repair lays mainly in the organisation of sister chromatids.

The complex of proteins MRE11, RAD50 and NBS1 (MRN) is one of the key sensors and mediators of the DSB repair. In *Physcomitrella*, only PpMRE11 and PpRAD50 participate on MRN complex functions. Extreme sensitivity of *ppmre11* and *pprad50* to the induction of DSB suggests extensive defect in repair, which is not related to the rate of repair, but rather to the accumulation of mutations, especially deletions. In this regard, PpRAD50 is particularly important, because in its absence the mutability increases up to two orders of magnitude.