Abstract

Plants are highly resistant to ionizing radiation, also thanks to a high-quality repair system for repairing double-stranded breaks. Double-strand breaks in plants are repaired by four repair pathways. Most often, double-strand breaks are repaired by non-homologous end joining (NHEJ), which joins the broken ends without further processing. More accurate but slower and more complex is repair through homologous recombination (HR), which repairs the break using a homologous sequence. HR repair takes place preferentially in a region with active transcription and during the S and G2 phases of the cell cycle. Alternatively, repair further proceeds through single-strand annealing (SSA) or Theta mediated end joining (TMEJ). Both pathways are based on short homology between the overlapping ends of the double-strand break. An often neglected part of repairs is the overcoming of repressive chromatin, which protects the genome from DNA damage and prevents access of nucleases but also acts as a barrier for repair proteins. This work summarizes the current knowledge about DNA repair in plants. Furthermore, describe the influence of chromatin not only on the repair but also on the activity of programmable nucleases used in genetic engineering, such as zinc finger nucleases (ZFNs), transcription activator-like effector nucleases (TALENs) and clustered regularly interspaced short palindromic repeats / CRISPR associated (CRISPR/Cas).

Keywords: chromatin, double strand break, CRISPR/Cas, DNA repair, HR, NHEJ, SSA, TALENs, TMEJ, ZNFs