Meiotic homologous recombination generates new combinations of preexisting genetic variation and is a crucial process in plant breeding. Within the last decade, our understanding of plant meiotic recombination and genome diversity has advanced considerably. Innovation in DNA sequencing technology has led to the exploration of high-resolution genetic and epigenetic information in plant genomes, which has helped to accelerate plant breeding practices via high-throughput genotyping, and linkage and association mapping. In addition, great advances toward understanding the genetic and epigenetic control mechanisms of meiotic recombination have enabled the expansion of breeding programs and the unlocking of genetic diversity that can be used for crop improvement. This review highlights the recent literature on plant meiotic recombination and discusses the translation of this knowledge to the manipulation of meiotic recombination frequency and location with regards to crop plant breeding.

**Keywords:** breeding, crossover, epigenetics, meiotic DSBs, meiotic recombination

**INTRODUCTION**

Meiosis is a specialized cell division process that occurs in sexually-reproducing organisms in which a single round of DNA replication is followed by two successive rounds of chromosome segregation, resulting in gametes that have half the chromosome number (n) of their parental cells (2n). During fertilization, two gametes fuse together to restore the number of chromosomes in the resulting zygote to the same number that the parent cells had prior to meiosis (2n). During meiosis, programmed double-stranded breaks (DSBs) are formed by an evolutionally conserved SPO11 topoisomerase-like complex and are then repaired resulting in either reciprocal crossover, or non-crossovers by using a homologous template. By these processes, gametes are produced with recombined chromosomes (Hunter, 2015; Mercier et al., 2015; Villeneuve et al., 2001). Therefore, meiosis profoundly affects the genetic diversity of populations, and consequently adaptation potential, through meiotic recombination, independent chromosome segregation, and random fertilization (Barton and Charlesworth, 1998). This population genetic diversity also represents an important resource for selection of desirable traits in crop breeding (Bevan et al., 2017; Chaney et al., 2016).

Extensive genomic diversity has been revealed in plants by next-generation sequencing (NGS) technologies and computational analyses. Innovations in DNA sequencing and bioinformatics allow for the detection of genetic variations within plant populations that are used to recombine or map genomic locations of favorable traits by linkage and association mapping (Bevan et al., 2017; Chaney et al., 2016; Soyk et al., 2017). Genomics and computational approaches such as genotyping-by-sequencing and LDhat that is a package of estimating historical recombination by analyzing patterns of linkage disequilibrium (LD) in population have also contributed to the map of fine-scale meiotic crossovers in plant genomes (Choi et al., 2013; Hellsten et al., 2013; Wijnker et al., 2013). Like other eukaryotes, plant meiotic crossover...
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The frequency of meiotic recombination is not uniform along a chromosome, but instead occurs frequently within narrow regions of approximately 1-2 kb called recombination hotspots (Baudat et al., 2013; Choi and Henderson, 2015; Lichten and Goldman, 1995; Mercier et al., 2015). Fine-scale maps show that plant meiotic crossover hotspots occur near gene promoters and terminators in euchromatin while crossovers are suppressed in heterochromatin regions (Choi and Henderson, 2015; Lambing et al., 2017). Importantly, the suppression of crossover formation within heterochromatin is one of the main bottlenecks in attempts to recombine favorable traits in the place of unfavorable traits in crop plants such as wheat and barley (Bevan et al., 2017; Choulet et al., 2014; Mayer et al., 2012; Tomato Genome Consortium, 2012). Notably, the heterochromatin of crop genomes is large, covering hundreds of mega base-pairs, and also contains functional genes (Bevan et al., 2017; Choulet et al., 2014; Mayer et al., 2012).

Despite the excessive DSBs that are made during the initiation of meiotic recombination, only one to three DSBs result in crossovers along chromosome after repair. The rest of the DSBs result in either repair by a sister chromatid, or non-crossovers through repair by a different repair pathway, such as synthesis-dependent strand annealing (SDSA) (Gray and Cohen, 2016; Hunter, 2015; Mercier et al., 2015). The limited number of crossovers per chromosome is relatively conserved among species with the exception of a few fungal species that generate more crossovers, although at least one crossover is required to ensure proper chromosomal segregation (Mercier et al., 2015). Enhancing the crossover frequency in both euchromatin and heterochromatin should help breeders acquire desirable traits or remove undesirable traits along chromosomes in crop plant genomes, as both the resolution of genetic mapping and the recombination of desirable trait variations are dependent on meiotic crossover rates and locations (Bevan et al., 2017).

This review describes the recent advances in understanding and control of plant meiotic recombination in Arabidopsis, including meiotic DSBs as well as the genetic and epigenetic factors that contribute to crossovers. This is followed by a discussion of the application of these advances to crop breeding using the CRISPR (clustered regularly interspaced palindromic repeats) system.

HIGH RESOLUTION MAPPING OF PLANT MEIOTIC RECOMBINATION INITIATION SITES

In Arabidopsis, SPO11-1 and SPO11-2 are homologs of the archaeal DNA topoisomerase TOPOVI subunit A (TOPOVIA) that interact with MEIOTIC TOPOISOMERASE VIB-LIKE (MTOPVIB) to form a DNA topoisomerase VI-like heterotetrameric complex that catalyzes DSBs to initiate meiotic recombination (Fig. 1A) (Grelon et al., 2001; Hartung et al., 2007; Vrielynck et al., 2016). During DSB formation, the catalytically active tyrosine residue of SPO11 is covalently attached to 5' end of the DNA by a phosphodiester bond. Subsequent endo- and exonuclease activities of the MRNS (MRE11, RAD50, NBS1/XRS2, SAE2/COM1) complex with EXOI process the 3' region near the SPO11 attachment site, which releases SPO11-protein-oligonucleotide as a complex from the DNA (Fig. 1A) (Cannavo and Cejka, 2014; Choi et al., 2017; Garcia et al., 2011; Lam and Keeney, 2014; Neale et al., 2005; Pan et al., 2011). Accordingly, purified SPO11-associated oligonucleotides (~20-40 nucleotide) have been sequenced and used to generate high resolution genome-specific maps of meiotic DSBs in Arabidopsis.

**Fig. 1.** Genome-wide meiotic DSB map in a plant. (A) Production of the SPO11-1 protein-DNA oligonucleotide complex during meiotic DSB formation. (B) Landscapes of SPO11-1-oligonucletides (oligos) and crossovers along Arabidopsis chromosome 1. (C) Representative snapshot of meiotic DSBs, nucleosome occupancy, and H3K4m3 levels, in the chromosomal arm and centromeric region of Arabidopsis chromosome 1. A DNA transposon, Helitron (AT1TE29920) located at a gene promoter displays high DSB levels and low nucleosome occupancy (left image), while centromeric transposons (AT1TE51615, AT1TE51645, Gypsy retrotransposon) display low levels of DSBs (right image).
wide maps of meiotic recombination initiation sites in fungi and mice (Fowler et al., 2014; Lam and Keeney, 2014, 2015; Lange et al., 2016; Pan et al., 2011). The first plant meiotic DSB map was developed by sequencing Arabidopsis SPO11-1 oligonucleotides (30–50 nt) and revealed both conserved and plant-specific features compared with fungal and mammalian maps (Choi et al., 2017).

As in yeasts, meiotic DSB location and frequency in plants are mainly determined by nucleosome occupancy (Fig. 1B–1C) (Choi et al., 2017; Fowler et al., 2014; Pan et al., 2011). In mammals, the PRDM9 protein directs meiotic DSBs to specific DNA sequences (Baudat et al., 2013; Clément and de Massy, 2017; Lange et al., 2016). Since plants and yeasts do not have PRDM9, the nucleosome-depleted gene promoters are the highest DSB hotspots (Choi et al., 2017). Notably, plant gene terminators and introns are also DSB hotspots with low nucleosome occupancy (Fig. 1C), yet gene terminators are not DSB hotspots in yeast, even though they also have lower nucleosome occupancy (Choi et al., 2017; Pan et al., 2011). The DSB hotspots that were identified in Arabidopsis gene terminators are consistent with the occurrence of crossover hotspots in Arabidopsis and monkey flower plants (Choi et al., 2013; 2016; Hellsten et al., 2013; Wijnerk et al., 2013). Bird genomes also display crossover hotspots in both gene promoters and terminators, implying that a similar pattern of recombination hotspots is present in plants and birds (Singhal et al., 2015).

Strikingly, Arabidopsis DSB hotspots occur within specific DNA transposon families (such as Helitron, Tc1/mariner, and pogo) that are nucleosome-depleted, while retrotransposons and nucleosome-occupied DNA transposons are DSB coldspots (Fig. 1C) (Choi et al., 2017). The specific DSB hotspot transposons are enriched in pericentromeres and gene regulatory regions of proximal promoters and introns, whereas DSB coldspot transposons are often found within centromeres. Each transposon family also displays a distinct distribution pattern across chromosomes (Choi et al., 2017; Underwood et al., 2017a). The DSB hotspot DNA transposons are significantly associated with plant immunity genes such as defensin and the NBS-LRR gene family. This association suggests that the DNA transposons may play a role in enhancing the recombination frequency of these genes during adaptation. Hence, the detection of meiotic DSB hotspot inside transposons upon sequencing Arabidopsis SPO11-1 oligonucleotides expands the concept of McClintock’s Controlling Elements from modifying transcription to meiotic recombination that contributes to genome diversity and evolution (Chuong et al., 2016; McClintock, 1956; Slotkin and Martienssen, 2007). Compared with current meiotic DSB maps in other species, the SPO11-1-oligonucleotide maps in Arabidopsis show the strongest quantitative correlations between levels of meiotic DSBs, nucleosome occupancy, and AT sequence richness that excludes nucleosomes in both gene regulatory regions and DNA transposon hotspots (Choi et al., 2017). In addition, the meiotic DSB coldspots in transposons, pericentromeres, and centromeres are associated with heterochromatin marks of DNA methylation and H3K9 dimethylation (me2) (Choi et al., 2017; Underwood et al., 2017b). The inhibition of DSB formation in centromeres is required to limit meiotic, non-allelic, homologous recombination-induced genome instability (Sasaki et al., 2010). Decreased levels of DNA cytosine methylation or H3K9me2 lead to an increase in the number of meiotic DSBs in the heterochromatic regions including centromeres with reduced nucleosome occupancy. This finding demonstrates a crucial role for these epigenetic marks in suppressing meiotic DSB formation on a genomic scale (Choi et al., 2017; Underwood et al., 2017b).

**ENHANCING CROSSOVER RATES BY DISRUPTING ANTI-CROSSOVER GENES**

Following the dissociation of SPO11-oligonucleotide complexes and 5' to 3' single-strand resection, DMC1 recombines and its cofactor RAD51 lead the invasion of the homologous duplex with the 3' end of single-stranded DNA (ssDNA), producing a recombination intermediate molecule (Fig. 1A) (Cloud et al., 2012; Da Ines et al., 2013; Gray and Cohen, 2016; Mercer et al., 2015). Two main DNA repair pathways - class I and class II - process these recombination intermediates to generate the limited number of crossovers (Fig. 2A). In the class I interfering pathway, ZMM (ZIP1, MSH4, MSH5 and MER3) proteins stabilize the recombination intermediate molecules and contribute to most crossovers (85-90%) via MLH1/MLH3. In the class II non-interfering pathway, 10-15% of crossovers rely on MUS81 (Fig. 2A) (Lambing et al., 2017; Mercer et al., 2015). In Arabidopsis, zmm mutants, including zip4, hei10, msh4, msh5 and shoc/zip2, display significantly reduced fertility due to limited crossovers and produce only approximately 5 seeds per siliqule. Suppressors of zmm were identified using forward genetic screens of zmm mutant seeds. These zmm suppressors restore zmm fertility to wild-type levels (~50 seeds/Siliqule) and also display 2-6 fold higher crossover rates than wild-type plants (Crismani et al., 2012; Girard et al., 2015; Ségui&-Arnaud et al., 2015, 2017). The genetic mapping and analyses of zmm suppressor mutants revealed three independent pathways of anti-crossover factors that limit crossover frequency. Any combination of mutations among these three pathways additively increases the number of MUS81-dependent crossovers (Table 1).

Firstly, FANCONI ANEMIA COMPLEMENTATION GROUP M (FANCM) helicase and its cofactors limit crossover formation (Crismani et al., 2012; Girard et al., 2014). The fancm mutation leads to a 3-fold increase in crossover frequency in a homozygous background, although no somatic phenotypic changes were detected. However, the fancm mutants display wild-type crossover rates in hybrid plants, indicating that the strong crossover suppression may be due to the DNA heteroduplex state in recombination intermediate molecules (Borts and Haber, 1987; Fernandes et al., 2017b; Girard et al., 2015; Ziolkowski et al., 2015).

Secondly, the RECQ4A and RECQ4B proteins in Arabidopsis are two redundant orthologs of BLM/Sgs1 helicase that form a complex with TOP3a and RMI1 that has the strongest anti-crossover activity observed so far (Ségui&-Arnaud et al., 2015, 2017). Arabidopsis recq4a recq4b double mutants show a 6.2-fold increase in crossover frequency in homozygous
Table 1. Arabidopsis genes involved in meiotic crossover frequency and location

| Complex/Pathway     | Gene     | AT number | zmm sup | Increased fold CO rate in mutant or transgene | Reference                  |
|---------------------|----------|-----------|---------|-----------------------------------------------|----------------------------|
|                     |          |           |         | Inbred context | Hybrid context |                                   |
| FANCM helicase      | FANCM    | AT1G35530 | yes     | 3 | 1 | Crismani et al., 2012; Fernandes et al., 2017b |
| MHF1                | AT5G60930| yes       | 1.5-2   | n.d | Girard et al., 2014                           |
| MHF2                | AT1G78790| yes       | 1.5-2   | n.d | Girard et al., 2014                           |
| RTR complex         | RECO4A   | AT1G10930 | yes     | 6 | 5 | Fernandes et al., 2017b                      |
|                     | RECO4B   | AT1G60930 |         |    |                                               |
|                     | TOP3a    | AT5G63920 | yes     | 3 | n.d | Séguela-Arnaud et al., 2015                  |
|                     | RMI1     | AT5G63540 | yes     | 3 | n.d | Séguela-Arnaud et al., 2017                  |
| FIGL1 helicase      | FIGL1    | AT3G27120 | yes     | 2 | 2 | Girard et al., 2015                         |
|                     | FLIP     | AT1G04650 | yes     | 1.2| n.d| Séra et al., 2017; Ziółkowski et al., 2017   |
| Class I CO pathway  | HEI10    | AT1G53490 | no      | 2 | 2 | Serra et al., 2017; Ziółkowski et al., 2017   |
| non-CG methylation  | CMT3     | AT1G69770 | no      | * | * | Underwood et al., 2017b                     |
| H3K9me2             | SUVH4    | AT5G13960 | no      | * | n.d.| Underwood et al., 2017b                     |
|                     | SUVH5    | AT2G35160 | no      |    |                                               |
|                     | SUVH6    | AT2G22740 | no      |    |                                               |
| Anti-crossover      | MSH2     | AT3G18524 | n.d     | 1.4| Emmanuel et al., 2006                       |

n.d. indicates that crossover rate is not determined.
Star (*) indicates that crossover frequency is increased in pericentromeric regions.

Fig. 2. Control of crossover frequency and location in plant genomes. (A) Limitation of crossover number in the Arabidopsis genome. During Arabidopsis male meiosis, an average of 250 meiotic DSBs and 11 crossovers occur throughout the genome. (B) A strategy for plant breeding programs to increase crossovers in both euchromatin and heterochromatin of hybrid lines by genetic and epigenetic disruptions. Enhancing the number of crossovers in the genome helps accelerate genetic mapping of desirable traits and generation of new varieties.

backgrounds and even an approximately 5-fold increase in heterozygous states. The recombination intermediates in recq4a recq4b mutants appear to be less sensitive to DNA heteroduplex-mediated crossover suppression than those in fancm mutants, and strongly favor being repaired to crossovers (Borts and Haber, 1987; Fernandes et al., 2017b). Finally, the AAA-ATPase FIDGETIN-LIKE 1 (FIGL1) and its interacting factor FLIP restrict the invasion by single-stranded DNA and thus crossover formation by controlling the dynamics of recombinase DMC1 and RAD51 (Fernandes et al., 2017a; Girard et al., 2015). Mutating figl1 results in an approximately 2-fold increase in crossover frequency in both inbred and hybrid lines (Girard et al., 2015). Genetic analyses of these three pathways revealed that the highest increase in crossover rate in hybrid plants (9-fold) was obtained with the recq4a recq4b figl1 triple mutants (Fernandes et al., 2017b). Like recq4a recq4b figl1, the recq4a recq4b fancm mutant also displays a 9-fold increase in crossover frequency in in-
bred lines. This crossover rate is slightly higher than that of a recq4a recq4b double mutant hybrid line because the fanCM mutation is not present to additionally increase crossovers in hybrid lines. Notably, the recq4a recq4b fig1 fanCM mutant that is defective in all three pathways does not show a significantly higher crossover frequency than the recq4a recq4b fig1 mutant when tested in hybrid plants (Fernandes et al., 2017b). Ultimately, the studies on ZMM suppressing in Arabidopsis indicate that the mutations of RECQ4A, RECQ4B and FIGL1 anti-crossover genes can be applied to crop plant breeding programs to increase crossover frequency (Fig. 2B).

DOSE-DEPENDENT INCREASE IN CROSVERS BY HEI10

Besides the genetic disruptions of anti-crossover genes for enhancing crossovers, the natural variations in meiotic crossover-promoting factor genes such as RNF212, PRDM9 and HEI10, also contribute to regulating crossover frequency and distribution (Kong et al., 2008; Sandor et al., 2012; Ziolkowski et al., 2017). In Arabidopsis, recombination quantitative trait loci (rQTL) mapping between Col-0 and Ler-0 accesses shows that one of two genetic variations of transacting modifiers maps to the HEI10 locus, while the other remains to be elucidated (Ziolkowski et al., 2017). HEI10 encodes a conserved meiotic E3 ubiquitin ligase required for crossover formation through the ZMM pathway (De Muyt et al., 2014; Gray and Cohen, 2016; Hunter, 2015). A single substitution is found in the HEI10 protein sequence (R264G) between Col-0 and Ler-0, Bur-0 and Cvi accesses. The lines that have the R264G substitution in HEI10 display a lower crossover rate than Col-0 and Ct-1, which express the R264G substitution in HEI10 display a lower crossover frequency than Col-0 and Ct-1, which express the R264G substitution in HEI10 protein sequence (R264G). A single extra genomic copy of the R264 HEI10 variant (Ziolkowski et al., 2017). Like in mammals, Arabidopsis hei10 mutants display haploinsufficiency in crossover frequency, and HEI10 is also able to promote crossover formation in a dosage-dependent manner (De Muyt et al., 2014; Qiao et al., 2014; Serra et al., 2017; Ziolkowski et al., 2017). Adding a single extra genomic copy of the R264 HEI10 variant results in a 2-fold increase in the total crossover number and a 4-fold increase when combined with recq4a recq4b mutations in hybrid plants (Serra et al., 2017). It remains undetermined whether the extra HEI10 transgenes can increase crossover frequency even further in a recq4a recq4b fig1 mutant background. Since an extra HEI10 gene lead to a dosage-dependent increase in crossover frequency in hybrid plants, this might be a useful breeding approach for increasing crossovers in hybrid plants where the HEI10 elite cultivar can be crossed with diverse cultivars.

EPIGENETIC ACTIVATION OF CROSVERS IN HETEROCHROMATIN

Although genetic disruption of the anti-crossover genes and adding extra copies of the HEI10 gene result in the formation of more crossovers in gene-rich chromosome arms, crossovers in the pericentromeres and centromeres remain suppressed in Arabidopsis (Fig. 1B) (Fernandes et al., 2017b; Serra et al., 2017). The heterochromatic features DNA cytosine methylation, H3K9me2, H2A.W, and transposons are highly enriched in the pericentromeres and centromeres (Lippman et al., 2004; Lister et al., 2008; Stroud et al., 2013; Yelagandula et al., 2014). DNA demethylation was expected to increase crossovers in the heterochromatic regions; however, the loss of DNA CG methylation maintenance by mutation of MET1 or DDM1 surprisingly led to a reduction in the number of crossovers around the centromeres (Melamed-Bessudo and Levy, 2012; Yelina et al., 2012). Furthermore, an increase in crossovers within subtelomeric regions was also observed, ultimately reshaping the crossover distribution along chromosomes in met1 and ddm1 mutants (Melamed-Bessudo and Levy, 2012; Yelina et al., 2012: 2015). The cytosine methylation in CHG contexts is introduced into plant genomes by CHROMOMETHYLASEs (CMT3) and CMT2 cooperatively, while the methylation in CHH contexts is introduced by CMT2 (Du et al., 2012; Lindroth et al., 2001; Stroud et al., 2013: 2014; Zemach et al., 2013). During CHG methylation by CMT3, the chromo and BAH (Bromo-Adjacent Homology) domains of CMT3 simultaneously bind to H3K9me2 on the nucleosome, while histone methylation is catalyzed by the histone methyltransferases KRYPTONITE/ SUVH4, SUVH5 and SUVH6 (Du et al., 2012). The SRA domain of KRYPTONITE directly recognizes the DNA methyl cytosines that are maintained by CMT2 and CMT3 (Johnson et al., 2007). Therefore, CMT2/3 and SUVH4/5/6 proteins reinforce recruitment of each other and catalysis of non-CG methylation and H3K9 methylation, respectively, on the same nucleosome (Du et al., 2012; Stroud et al., 2014). Non-CG methylation also can be introduced by the de novo DNA methyltransferases DOMAINS REARRANGED METHYTRANSFREASE1 (DRM1) and DRM2 (Cao and Jacobsen, 2002; Cao et al., 2003). Notably, the loss of non-CG methylation and/or H3K9me2 enhance crossover frequency around pericentromeres but not centromeres, even though the number of meiotic DSBs substantially increases in centromeres (Underwood et al., 2017b). The increased crossovers in pericentromeric regions occur in both inbred and hybrid suvh4 suvh5 suvh6 or cmt3 mutant lines (Underwood et al., 2017b). Therefore, the epigenetic disruption of non-CG methylation or H3K9me2 can be translated to crop breeding programs that introduce new combinations of genetic alleles into the large pericentromeric regions (Tables 1 and 2; Fig. 2B).

MANIPULATION OF MEIOTIC RECOMBINATION FOR CROP IMPROVEMENT

The dependence of plant crop breeding on meiotic crossover frequency and crossover distribution can delay breeding time and restrict both combination and genetic mapping of desirable traits. To accelerate crop breeding and unlock genetic diversity, we can use the CRISPR system as a precise genome editing tool to manipulate the genomes of higher eukaryotes and control of meiotic recombination (Doudna and Charpentier, 2014; Hsu et al., 2014; Kim, 2016; Puchta, 2017). CRISPR approaches represent DNA-free gene editing tools that can be applied directly to the genomes of elite crop cultivars to implement genetic variations that are
Table 2. Crop plant orthologous genes to Arabidopsis FIGL1, RECQ4 and CMT3 genes

| Arabidopsis gene | Plant species | Locus |
|------------------|---------------|-------|
| FIGL1            | Rice          | LOC9271031 |
|                  | Wheat         | AK331006 |
|                  | Maize         | LOC100193153 |
|                  | Tomato        | LOC101262887 |
|                  | Soybean       | LOC100789161, LOC100776024 |
| RECQ4A           | Rice          | LOC_Os11g48090(A), LOC_Os04g35420(B) |
| RACQ4B           | Wheat         | AK334643 |
|                  | Maize         | LOC100274706 |
|                  | Tomato        | LOC101260976 |
|                  | Soybean       | LOC100800006, LOC100817867 |
| CMT3             | Rice          | OsCMT3a(LOC_Os10g01570), OsCMT3b(LOC_Os03g12570) |
|                  | Wheat         | AK332918 |
|                  | Maize         | Zme2(GQ923937) |
|                  | Tomato        | LOC101265056, LOC101267211 |
|                  | Soybean       | LOC100799480 |

known to contribute to desirable traits, such as productivity and herbicide resistance (Kim, 2016; Puchta, 2017; Soyk et al., 2017; Wolter and Puchta, 2017; Yin et al., 2017). In addition, CRISPR or RNAi systems can be used to mutate or knock-down the genes encoding anti-crossover and epigenetic factors simultaneously, which may lead to an increase in crossover frequency in both euchromatin and heterochromatin (Tables 1 and 2; Fig. 2B). The DNA mismatch repair protein MSH2 represents an additional target for disruption using these systems, as mutation of the MSH2 gene attenuates heterozygosity-mediated crossover suppression and results in a 40% increase in crossover rate (Emmanuel et al., 2006). Finally, CRISPR based tools can be further modified with either meiotic specific expression, or fusion with a meiotic protein as an effector, to induce meiotic recombination at a specific target site and potentially increase genetic and epigenetic variations of favorable traits that involve linked alleles such as clustered plant immunity genes (Deng et al., 2017).

**CONCLUSION**

Meiotic recombination has a profound effect on genetic diversity, which is crucial for both the adaptation of plants to their environment and the improvement of crop traits of agricultural value. CRISPR tools can now be used to precisely edit the genomes of elite cultivars. However, the natural variation in the wild relatives of crop plants, landraces, and diverse cultivars also provide a valuable resource for crop improvement, as does genetic variation by chemical and radiation-driven mutagenesis. The discovery of anti-crossover and epigenetic factor genes that affect meiotic crossover in Arabidopsis will add new branches to crop breeding programs that aim to create new combinations of favorable traits and unlock unexplored genetic diversity. To further understand the mechanism of crossover formation in plants, the meiotic factors that control DSB formation and act in the ZMM pathway that limits crossovers must be elucidated. In addition to the first plant SPO11-1 oligonucleotide maps, genomic profiling of meiotic proteins will provide insights into how meiotic recombination is controlled, and how it can be induced at specific sites in plant genomes.

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