

Genomic Variation and Mutational Events

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Phytopathologists are actively researching the molecular basis of plant–pathogen interactions. The mechanisms of responses to pathogens have been studied extensively in model crop plant species and natural populations. Today, with the rapid expansion of genomic technologies such as DNA sequencing, transcriptomics, proteomics, and metabolomics, as well as the development of new methods and protocols, data analysis, and bioinformatics, it is now possible to assess the role of genetic variation in plant–microbe interactions and to understand the underlying molecular mechanisms of plant defense and microbe pathogenicity with ever-greater resolution and accuracy. Genetic variation is an important force in evolution that enables organisms to survive in stressful environments. Moreover, understanding the role of genetic variation and mutational events is essential for crop breeders to produce improved cultivars.

Keywords: genomic variation ; mutational events ; breeding for resistance

1. Genomic Variation and Mutational Events in Hosts and Pathogens

Genomic variation describes the differences between individuals' genomes. More precisely, genomic variation is a DNA segment that differs in length, orientation, copy number, or chromosomal location between different individuals ^[1]. Genomic variation encompasses various microscopic (visible under a microscope—for example, chromosomal rearrangements) and submicroscopic (>1000 bp) types of variation in a species' genome, resulting in deletion; duplication; changes in sequence, such as a single nucleotide polymorphism (SNP); and the creation of new genes, resulting in heritable phenotypic changes seen within and between species ^[2]. Genomic variations play a significant role in plant adaptive evolution, functional diversity, and phenotypic diversity ^[3].

There are several causes of genetic variation such as mutation and genetic recombination ^[4]. Genomic structures and mutational events that allow rapid evolution include AT-rich isochores, length polymorphism and chromosomal polysomy, chromosomal rearrangements, conditionally dispensable chromosomes, copy number variation (CNV), de novo genes, epigenetic modification of gene expression, horizontal gene/chromosome transfer (HGT/HCT), hybridization, insertions/deletions (indels), polyploidization, repeat-induced point mutation (RIP), RIP leakage, single nucleotide polymorphisms (SNPs), and transposable elements (TEs).

2. Transposable Elements

Transposable element (TE) insertions and deletions, originally considered selfish DNA or 'genome parasites' ^[5], are mobile genetic components that can jump across genomes. Transposition events are among the most common genetic variations in plants that can result in gene activation, inactivation, duplication, and even the appearance of a new gene ^[6]. TE insertion can disrupt the open reading frame (ORF) by invading the space inhabited by protein-coding genes and yield abnormal phenotypes ^[7]. In fungal phytopathogens, TEs play an important role in rapid evolution by affecting genome plasticity ^{[8][9]}, pathogenicity ^[10], host range ^[11], and evolution ^{[12][13]}. In some fungal plant pathogens, genome compartments on core chromosomes act as accessory islands and encode virulence determinants ^[14]. In *L. maculans* 'brassicae' and *Zymoseptoria tritici*, TE-rich genome sections are exemplified by epigenetic alterations that are further associated with diverse patterns of transcription and accumulation of mutations ^[15]. These compartments can be produced by structural changes or develop in regions with suppressed recombination ^[16]. For example, in *Verticillium dahliae* and *Z. tritici*, accessory genome sections originate through structural changes and unfaithful DNA repair across repeated sequences ^[14]. Pathogen genomes with low TE can still have fast-developing genomic regions that promote effector evolution.

The activity of transposable elements plays a significant role in effector gene evolution ^{[14][17]}. For example, although *Ustilago maydis*, *Sporisorium scitamineum*, and *S. reilianum* have low TE content, the TEs are remarkably linked to virulence gene clusters ^[18]. The association between TEs and effector genes indicates that elevated mutation levels in repetitive genome sections support effector improvement and adaptation, as shown in *Magnaporthe oryzae* and *Fusarium*

oxysporum [17][19][20]. As demonstrated in *M. oryzae*, TEs are frequently found near pathogenicity factors [21]. The TE-pathogenicity gene involvement was also demonstrated in other fungal pathogens—for example, *Mycosphaerella fijiensis*, which causes black Sigatoka in bananas [22] and *M. graminicola*, which causes *Z. tritici* blotch in wheat [23]. TE insertion may alter a fungal pathogen's pathogenicity and host specificity by generating genetic variations in virulence factors to evade detection by the host plants. Collectively, the presence and actions of TEs promote variability and adaptability.

3. Repeat-Induced Point Mutation

The repeat-induced point (RIP) mutation is a genome defense mechanism specifically found in fungi that protects against the harmful effects of repetitive genomic regions and TEs by mutating cytosine to thymine in repetitive sequences [24]. The RIP pathway protects the fungal genome from the genetic implications of repeated sequence elements, so-called “selfish” sequences, especially those connected with transposable elements [24][25]. The spread of duplicated sequences into neighboring nonrepetitive regions is called RIP leakage [6]. RIP was first identified in *Neurospora crassa* [26]. RIP-like C: G to T: A transitions were reported in the sequences of transposable elements in several fungi such as *Aspergillus fumigatus* [27], *Aspergillus nidulans* [28], *F. oxysporum* [29], and *Magnaporthe grisea* [30]. RIPs are prevalent in *L. maculans* [31], as shown by the degeneration of the retrotransposons (found in the *AvrLm1-AvrLm6* regions), as well as the low GC content in corresponding retrotransposon-rich isochores. Furthermore, in *L. maculans* ‘brassicae’, the RIP mutation can play a crucial role in transposable element silencing and effector evolution [17]. Furthermore, it was shown that RIP operates in *M. grisea* during the sexual phase [32]. The development of specific genes is also influenced by the emergence of RIP-driven lineage-specific regions [17]. The widespread conservation of RIP indicates that RIP is mostly useful for fungal survival and plays critical roles in genome development and evolution, supporting or hindering gene variety and the revolution of novel genes [33].

4. AT-Rich Isochores

The AT-rich isochores is a region with high content of thymine and adenine residues. AT-rich isochores usually concur with deactivated repetitive elements [6]. AT-rich regions can arise through a variety of mechanisms such as repeat-induced point mutation (RIP), a fungal-specific process mainly considered a means of preventing transposon propagation [24][34]. In most fungi, AT-rich regions are a hallmark of RIP that aim for repetitive DNA and reduce GC-content [34]. The AT-rich region is where DNA synthesis is initiated and the replication complex is formed. High AT-content causes lower thermodynamic stability, which describes the role of AT in the initiating of the replication process [35]. In fungal genomes with substantial numbers of AT-rich regions, a bimodal pattern of GC-content bias can be observed. The *L. maculans* genome was the first fungal genome published with a considerable proportion of AT-rich regions (~33% of the assembly) [17]. Since then, AT-rich regions have been discovered in various fungal genomes such as *Passalora fulva* [36], *Blastomyces dermatitidis* [37], multiple *Epichloë* spp. [38], and *Z. tritici* [39]. Studies on genes encoding avirulence/effector-like proteins such as *L. maculans* genes *AvrLm6*, *AvrLm4-7*, and *AvrLm1*, have increased interest in AT-rich regions [40]. In *L. maculans*, it was reported that like all other *AvrLm* genes, *AvrLmS-AvrLep2* exist in an AT-rich genome environment; encode for small, secreted proteins rich in cysteines; and are extremely overexpressed in the initial cotyledon infections [41]. In *Venturia inaequalis*, the region comprising *AvrVg* is located in isochores with significantly different GC content [42]. The organization is also recognizable in the genomes of *M. fijiensis* and *Passalora fulva*, which have effector-encoding genes in repeat-rich regions [36]. In a study on *Lupinus angustifolius* L., 22 genes were linked with AT-rich regions. While none were expected to be effector candidates, four continued the Pfam-related domain [43]. AT-rich regions were examined in *Pyrenochaeta lycopersici* ER1211 and *L. maculans* genomes in another work. AT-rich regions made up about one-third of the *L. maculans* genome and ~10% of the *P. lycopersici* ER1211 genome [34]. It was suggested that pathogenic fungi with putative effector genes located near AT-rich regions have competitive evolutionary power [44].

5. Chromosomal Rearrangements and Homeologous Exchanges

A chromosomal rearrangement encompasses different events, including duplications, inversions, and translocations of pieces of chromosomes between the sub-genomes. Sequence exchanges between homeologous chromosomes in polyploid plants result in immediate gene deletions and amplification or homeologous exchanges (HEs) [3][45]. HEs are caused by chromosome mispairing between two genomes that are ancestrally linked. Increased homeologous exchanges (HEs) and gene conversion events result from a meiotic chromosomal pairing between homeologous chromosomes with a high degree of sequence identity [46]. It was shown that HEs generate novel gene combinations and phenotypes in a range of polyploid species [47][48]. For instance, gene deletions and HEs between sub-genomes in *B. napus* were shown to reduce seed glucosinolate content [49]. The structures of plant pathogen genes simplify the rapid rearrangements and genomic variation in virulence-associated regions [50]. These rearrangements include chromosomal

length variations on a broad scale and the presence of isolate-specific supernumerary chromosomes (small and non-essential chromosomes in addition to the standard chromosomes) [51]. In eukaryotic pathogens, supernumerary chromosomes can be observed at different rates [50][52]. Supernumerary chromosomes have been linked to establishing novel virulence features in several fungus species [49]. The homologous exchange was defined by Shi et al. [53] as an alternate mechanism by which CNV-associated disease resistance QTLs evolved. Quantitative disease resistance was previously linked to homoeologous recombination [54] and the presence/absence of variation [55] in *B. napus*. In addition, Song et al. [56] discovered the genetic diversity affecting disease resistance to be enhanced in genomic regions affected by structural variation, including that caused by homoeologous recombination [57]. Several publications discuss how the genetic rearrangement between fungal isolates contributes to pathogenesis, whether by parasexual recombination, sexual recombination, or hybridization [58][59].

6. Presence/Absence Variation

Insertions/deletions (InDels) are small fragments of DNA (a few nucleotides up to 50 bp) that are present or absent compared to a reference genome. InDels are prevalent in many species and cause frame shifts by deleting or altering genes [6]. In contrast, presence/absence variation (PAV) is found in the size ranges of genes (up to a few kb) and result in severe functional and phenotypic changes [55]. Homeologous exchanges have also been the primary cause of gene PAV [47]. Since discovering PAV in the *RPM1* gene in *Arabidopsis* [60], many PAVs have been found in disease resistance genes in different species [61][62][63][64]. It was reported that PAV is a key determining factor of *Verticillium longisporum* resistance such that both short- and long-range PAV assist with *V. longisporum* resistance in canola [55]. Gabur et al. [55] also stated that PAVs in the genes primarily implicated in cell wall integrity, growth, and alteration are colocalizing with major resistance QTL in a *B. napus* population. In addition, Bakker et al. [65] showed that the concentrations of cell wall-associated components are considerably associated with *V. longisporum* resistance. In *L. maculans*, *V. dahliae*, *Phytophthora infestans*, *Z. tritici*, and *M. oryzae*, many effector genes show within-species PAV and remarkable connections with transposable-element-rich regions of chromosomes [12][14][66][67]. Despite these findings, little is known about the extent of gene PAV in fungal plant pathogens [68]. One reason for the paucity of data is that a pathogen's virulence is usually a quantitative trait [69], suggesting that the PAVs of effector genes may be a less common mechanism of coevolution than that in crops, in which virulence is more often a binary trait, with resistant varieties completely preventing infection [52].

7. Copy Number Variations

Copy number variations (CNVs) are chromosome insertions, deletions, and/or duplications, and are generally described as a DNA fragment with a different copy number than the reference genome [70]. CNVs implicate DNA segments usually larger than 1 kb in length [71]. CNVs can be inherited from a previous generation or emerge de novo because of duplication/deletion. The fixation of CNVs by drift or selection may contribute to genetic novelty, leading to species adaptations to stressful or new environments [72]. The biological roles of CNVs range from an apparent lack of influence on the overall variability of physiological features through morphological variability to, altered metabolic states, susceptibility to infectious diseases, and interactions between hosts and microbes. As a result, CNVs have great potential to contribute to population diversity [73]. Copy number variations affect many traits, including an organism's fitness and disease susceptibility, and contribute to co-evolutionary processes between pathogens and hosts or symbionts [74]. Plant disease defense genes were shown to have CNV in various species [63][75][76][77][78][79]. For instance, *Rhg1* confers resistance to soybean cyst nematodes and seems to act via the multiplication of the locus [77]. In a study, Qutob et al. [80] identified *Avr1a* and *Avr3a* from *P. sojae* and showed how the copy number variation and transcriptional differences of these *Avr* genes represent mechanisms for the evasion of *Rps*-mediated immunity. It was reported that *R* genes present higher CNVs than the rest of the genome [81]. For example, high levels of CNV were found in maize (129 *R* genes) and rice (508 *R* genes) [82].

CNVs were found in various plant pathogens, especially fungi, with some promising instances in an express link between CNVs and pathogenicity. For instance, grape powdery mildew (*Erysiphe necator*) can be controlled by sterol demethylase inhibitor (DMI) fungicides. A point mutation in the target gene *EnCYP51A* is a known mode of resistance to DMIs; however, resequencing DMI-resistant *E. necator* isolates showed frequent increases in the copy number of the mutant allele [83].

8. Single Nucleotide Polymorphisms

The replacement of a single nucleotide at a specific position in the genome is called a single nucleotide polymorphism (SNP). SNPs can occur within coding regions in amino acid substitutions, mis-splicing, or premature stop codons. SNPs have a broad distribution and can be detected in any region of a gene, mRNA, or intergenic region [3]. SNPs can result from deficiencies in DNA polymerase replication during meiosis/mitosis or damaged DNA [6]. With the advent of high-throughput genotyping technologies, genome-wide association or multi-SNP association approaches were developed as helpful tools for analyzing the interactions of complicated genetic characteristics in plants, including disease resistance [84]. Genetic variation can be assessed using phenotypic data in plant and pathogen species, and genome-wide association studies (GWAS) can be used to find genes and link them to phenotypes [85]. SNP discovery using GWAS analysis is feasible through various target-enrichment or reduction-of-genome-complexity methods such as genotyping-by-sequencing (GBS) [86] and the restriction of site-associated DNA sequencing (RADSeq) [87]. Several identified SNPs associated with plant diseases such as SNPs associated with anthracnose diseases in common bean [88], resistance to *Aphanomyces euteiches* in *Pisum sativum* [89], *Aphanomyces* root rot resistance against *Medicago truncatula* [90], resistance to *Uromyces pisi* in pea [91], verticillium wilt resistance in alfalfa [92], and resistance sites against *Plasmodiophora brassicae* in *B. napus* [93]. Using expressed sequence tag-based SNP markers, Kifuji et al. [94] mapped black rot resistance genes in cabbage and detected three QTLs. Similarly, Sharma et al. [95] developed a *Brassica carinata* F2 mapping population and mapped the black rot race 1 resistance locus Xca1bc. SNPs linked with plant colonization were found upstream of the Required for Arbuscule Development 1 (RAD1) locus, a positive regulator of arbuscular mycorrhizal (AM) fungal colonization in *M. truncatula* roots infected by *Phytophthora palmivora* [96]. Single nucleotide variant (SNV) is a substitution of a single nucleotide for another. Sometimes SNVs are known as SNPs, although SNVs and SNPs are not interchangeable. SNVs are only apparent in diploid or higher copy-number genomes and can be important for genomic differentiation for diploid/dikaryotic pathogenic fungi, as well as plants.

9. Chromosomal Polysomy or Length Polymorphism

Chromosomal polysomy occurs when an individual has at least one more chromosome than normal. Thus, instead of the expected two copies, there may be three or more copies of a chromosome. Core or dispensable chromosomes can become duplicated. Chromosomal polysomy occurs in various species, including plants, fungi, insects, and mammals [97]. Polysomy exists in many plant species, including *Brassica* species [98]. In plants, the mechanisms of polysomes includes non-disjunction (the failure of a pair of homologous chromosomes to separate), mis-segregation in diploids or polyploids, and mis-segregation from the multivalent interchange of heterozygotes [99]. In fungi, the polysomy of chromosome 13 was studied in yeast species *Saccharomyces cerevisiae* [100]. In addition, homologous chromosomes between individuals of the same species can have considerable length differences [6]. In fungi, chromosome translocations, deletion/insertion/duplication events, changes in repetitive DNA sequences, and dispensable chromosomes are the main causes of chromosome length polymorphisms [101]. In *Magnapotha grisea* and *F. oxysporum*, many families of TEs were discovered and linked as key factors affecting karyotypic instability [102].

10. Conditionally Dispensable Chromosomes

Unlike core chromosomes, conditionally dispensable (CDCs), or accessory chromosomes, are not essential for an organism. CDCs often differ from the core chromosomes in their size (typically less than 2.0 MB), gene content, and sequence characteristics [52]. Additionally, CDCs can be passed horizontally between isolates, potentially conferring new pathogenic characteristics on the recipient isolate [103]. In the case of plant pathogens, CDCs harbor virulence genes [6]. In fungi, CDCs were reported in several plant-pathogenic species, such as *Alternaria* species [104], *Fusarium solani* [105], and *F. oxysporum* [106]. Dispensable chromosomes were found in 14 species of fungi [107], including *Colletotrichum gloeosporioides* [108]. Plaumann et al. [109] showed that the deficiency of a dispensable chromosome in *Colletotrichum higginsianum* has critical effects on the fungus' pathogenicity. Additionally, Ayukawa et al. [110] indicated that *F. oxysporum* f. sp. *conglutinans* (*Focn*) has multiple CDCs. The researchers identified specific CDCs required for virulence on *Arabidopsis*, cabbage, and both. They also described a pair of effectors encoded on one of the CDCs required to suppress *Arabidopsis*-specific phytoalexin-based immunity. It was proposed genes playing a role in coding for host-specific toxins (HSTs), including AF-toxin from the strawberry pathotype [111], AK-toxin from the Japanese pear pathotype [112] and ACT-toxin from the tangerine pathotype [113], are positioned on CDCs. CDC loss can happen due to repeated sub-culturing, causing the fungus to shift from a pathogenic to saprophytic state [114].

11. De Novo or Orphan Genes

De novo genes are species-specific (orphan) genes that derive from DNA sequences that previously lacked coding potential [6]. De novo genes are a subgroup of new genes that can code for proteins or serve as RNA genes [115]. De novo genes have different features than other genes in the genome. For example, de novo genes are shorter in size, have a lower expression rate, and contain more extensively varied sequences [116]. De novo gene birth is how new genes emerge from previously non-genic DNA sequences. De novo gene birth is essential for the divergence and adaptation of an organism [117]. The *BSC4* gene in *Saccharomyces cerevisiae* is an example of de novo gene birth [118]. The origins of de novo genes in plants have been widely studied [119][120][121][122]. Based on similarities to non-genic regions of *Arabidopsis lyrata*, almost half of the orphan genes in *A. thaliana* appear to have originated de novo [120]. Plant responses to the environment seem to be influenced by orphan genes [123]. For example, more than 80% of knockout mutants of unknown function genes in *A. thaliana* showed an altered phenotype when stressed, conferring either protection against, or serving as suppressors of, different abiotic stressors, notably oxidative and osmotic stresses [124]. A group of orphan genes was found in fungal pathogens limited to a single species or narrow clade. Pathogenic fungi may develop unique orphan genes to help infection or increase virulence. Because orphan genes lack homologs in closely related species, fungal effectors are ideal for orphan genes that developed for plant infection. Hundreds of orphan genes are encoded in the *Fusarium graminearum* genome [125]. The role of de novo or orphan genes in the pathogenic interactions and coevolution of pathogens with their host plants, however, remains unknown.

12. Epigenetic Modification of Gene Expression

Epigenetic modifications (e.g., DNA methylation, histone post-translational modifications, microRNAs, and the positioning of nucleosomes) are heritable alterations in gene expression patterns that occur without affecting the underlying DNA sequence and impacting the outcome of a locus or chromosome [126]. Epigenetic changes can affect only a particular gene (RNA interference (RNAi)-based silencing), or they can affect whole chromosomal regions (for example, epigenetic silencing of sub-telomeric regions due to histone modifications) [6]. Plant genomes are altered by various epigenetic pathways that regulate plant growth, development, and reproduction. Recent studies discovered many epigenetic factors participate in biotic and abiotic stress responses and adaptations in plants [127][128].

DNA methylation refers to adding a methyl (CH₃) group to DNA and is an epigenetic mechanism that controls gene expression. As part of the plant's defensive system, DNA methylation due to pathogen infection was reported in many plant species such as *Oryza sativa*, *A. thaliana*, *Nicotiana tabacum*, *Brassica rapa*, *Glycine max*, *Citrullus lanatus*, and *Aegilops tauschii* [129][130][131][132][133][134][135][136][137][138]. It was reported that pathogen detection provokes active changes in plant DNA methylation. For example, in *Arabidopsis*, infection with *P. syringae* pv. *tomato* DC3000 led to DNA hypomethylation in several genomic regions, such as peri/centromeric repeats and *Athila* retrotransposon [139]. Additionally, RNA-directed DNA methylation (RdDM) controls plant responses to pathogen attack. *Arabidopsis ago4* (ARGONAUTE 4, a vital component of the RdDM pathway) mutants feature reduced DNA methylation rates at different genomic locations and showed increased susceptibility to virulent *P. syringae* pv. *tomato* DC3000 [140]. Moreover, DNA demethylation in transposon-containing promoters enhances plant disease resistance. For instance, the *Arabidopsis ros1* (REPRESSOR OF SILENCING 1, a DNA demethylase) mutant presented greater susceptibility to *P. syringae* pv. *tomato* DC3000, which corresponded with substantially elevated cytosine methylation in a TE (*AtREP11*) present in the promoter of an *R* gene (*RMG1* or *At4g11170*) and consequently decreased gene expression [130].

As other epigenetic mechanisms, histone methylation and histone acetylation are active and reversible processes controlled by histone methyltransferases and histone demethylases and histone acetyltransferases and histone deacetylases, respectively [141]. Histone methylation and demethylation turn the genes in DNA "off" and "on", respectively. Histone acetylation, on the other hand, is exclusively associated with gene activation [142]. In plant-biotic interactions, histone (de)methylation regulates plant defense. For example, the methyltransferases SDG8 and SDG25 were implicated in PTI, ETI, and systemic acquired resistance against bacterial and fungal pathogens. Moreover, *sdg8* and *sdg25* single and *sdg8 sdg25* double mutants displayed increased susceptibility to *B. cinerea* and *Pst* [143][144]. The role of histone (de)acetylation in plant-pathogen interactions on *Arabidopsis* has been examined in many studies [145][146][147]. In addition, the control of plant-pathogen interactions via histone (de)acetylation was investigated in the wheat histone acetyltransferase complex TaGCN5-TaADA2, which triggers wheat wax biosynthesis, thereby delivering wax signals for germinating conidia in fungal pathogen *Bgt* [148]. Additionally, rice HDAC OsHDT701 cooperates with the rice RNase P subunit Rpp30, and negatively controls rice defense responses to *M. oryzae* and Xoo by facilitating histone deacetylation at PRR and defense genes [149].

The transfer of ubiquitin to histone core proteins is known as histone ubiquitination. Histone ubiquitination, whether monoubiquitination or polyubiquitination, controls a series of cellular processes in plants. In *Arabidopsis*, histone H2B monoubiquitination (H2Bub) is carried out via HISTONE MONOUBIQUITINATION (HUB1) and HUB2 [150], which control *SNC1* and *RPP4* expression following *P. syringae* pv. *tomato* DC3000 attack [151].

13. Horizontal Gene/Chromosome Transfer

The non-sexual transfer of genetic material, either a single gene or whole chromosomes between unicellular and/or multicellular organisms and acceptor organisms without a parent–offspring relationship is known as horizontal gene transfer (HGT). *Agrobacterium*-mediated transformation is the best example of HGT. After transferring a segment of *Agrobacterium* DNA into the host's genome, *Agrobacterium* induces neoplastic growth or unregulated cell division, leading to crown galls or growing roots [152]. HGT plays an important role in the evolution of prokaryotic clones by providing new genes involved in pathogenicity and promoting adaptive traits [153]. Studies on fungal genomes suggest that HGT significantly influenced the evolution of pathogenic traits in fungal pathogens [154][155]. There is also evidence that some characteristics of fungal biology may allow for gene transfer. For example, the anastomosis of fungal conidia, germ tubes, and hyphae results in cytoplasmic cell–cell linkages between cells of different species [156]. In a study, Qiu et al. [157] analyzed genomic data from the fungal pathogen *Magnaportheopsis incrustans*. The researchers discovered two instances of exclusive sharing of HGT-derived gene markers between Magnaporthales and another lineage of plant–pathogenic fungi in the genus *Colletotrichum*. Yin et al. [158] identified 32 HGT events in *Valsa mali*, most of which were HGTs from bacteria, along with several others from eukaryotes.

HCT between two vegetative incompatible biotypes of *C. gloeosporioides* [159] and the transfer of supernumerary chromosomes (extra chromosomes composed primarily of DNA not found in all representatives of the species) into nonpathogenic strains of *A. alternata* [160] are examples of HCT between fungi. Moreover, the horizontal transfer of chromosome 14 from *F. oxysporum* f.sp. *lycopersici* to nonpathogenic *F. oxysporum* strains confers the pathogenicity of these strains towards tomato [19].

14. Hybridization

The process of interbreeding individuals of different varieties or species to produce a hybrid is called hybridization. Breeding programs have yielded extensive hybridization between individuals of the same or different plant species. The introgression of genes for disease resistance between species has been widely studied in *Brassica* species. For example, chromosome B4 from *Brassica nigra* was introgressed into the rapeseed variety “Darmor” as a source of resistance against *L. maculans* (causal agent of blackleg) and led to high resistance [161]. Similarly, a B genome chromosome was introgressed from *B. carinata* to *B. napus* indicating high resistance against *L. maculans* [162].

Other cases of resistance transfer through hybridization include hybridization between *B. carinata* (donor) and *B. oleracea* to enhance resistance against *Erysiphe polygoni* (which can cause powdery mildew disease) [163], the transfer of black rot resistance from *B. carinata* to *B. oleracea* [164], the transfer of brassica leaf blight resistance (caused by *Alternaria brassicae*) from *B. hirta* to *B. juncea* [165], and the production of powdery mildew resistance from *B. carinata* to *B. oleracea* through embryo rescue followed by backcrossing to *B. oleracea* [163]. From the pathogen side, Bertier et al. [166] showed that hybridization increased *Phytophthora* clade 8b pathogenicity.

15. Polyploidization

Polyploidization, or whole-genome duplication, refers to the acquisition of extra sets of chromosomes in a cell or organism and frequently occurs in vascular plants. Polyploidization is an essential aspect of plant evolution and can significantly modify a plant's genetic make-up, physiology, morphology, and ecology within one or more generations [167]. Polyploidization can affect biotic interactions and resistance to pathogens, with polyploids generally having enhanced pathogen resistance. Differences between diploids and polyploids in *R* genes reflects altered pathogen resistance [168]. For example, polyploidy can increase resistance within the gene-for-gene interactions that underlie many host–pathogen interactions and where genotype × genotype interactions are important [169]. Quantitative resistance against *P. infestans* and *Tecia solanivora* in 4x potato was, moreover, observed using QTL analysis [170]. In a study, neopolyploids of a monogenic resistant apple cultivar showed increased resistance to *V. inaequalis* compared to diploid cultivars [171]. Another study found that synthetic tetraploids of Livingstone potato (*Plectranthus esculentus*) were more resistant to root-knot nematodes than diploids [172]. Pathogens can also change ploidy during infections; the phenomenon occurred with *P. infestans*, which caused the Great Irish Potato Famine [173]. From the evidence available, polyploidy can induce changes in pathogen interactions and increase disease resistance by regulating genome expression, resulting in alterations in

physiological characteristics, hormone biosynthesis, and improved antioxidant systems ^[174], which make polyploids better competitors than diploids. For example, polyploidy was investigated in *Bremia lactucae* by Fletcher et al. ^[175] who reported a high incidence of heterokaryosis in *B. lactucae*. Heterokaryosis has phenotypic consequences on fitness that may include an increased sporulation rate and qualitative differences in virulence.

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