Current Plant Biology 1 (2014) 10–33

Contents lists available at ScienceDirect

Current Plant Biology

journal homepage: www.elsevier.com/locate/cpb

Review

Plant speciation through chromosome instability and ploidy change: Cellular mechanisms, molecular factors and evolutionary relevance

Nico De Storme[a,*], Annaliese Mason[b,**]

[a] Department of Plant Production, Faculty of Bioscience Engineering, University of Ghent, Coupure Links 653, 9000 Ghent, Belgium
[b] School of Agriculture and Food Sciences and Centre for Integrative Legume Research, The University of Queensland, Level 2 John Hines Building, Brisbane 4072, QLD, Australia

ARTICLE INFO

Article history:
Received 15 May 2014
Received in revised form
17 September 2014
Accepted 17 September 2014

Keywords:
Polyploidy
Speciation
Unreduced gametes
Aneuploidy
Dysploidy
Somatic polyploidization
Meiotic restitution
Cytological mechanisms

ABSTRACT

Plant speciation and diversification strongly rely on structural changes in the nuclear genome, both at the whole ploidy and individual chromosome level. Phylogenetic, comparative mapping and cytological studies have provided insights into the evolutionary mechanisms that shape the plant genome. These include major genome alterations, such as whole genome duplication and hybridization (auto- and allopolyploidy), but also comprise the concomitant or independent occurrence of minor chromosome changes, such as aneuploidization and dysploidy (inversions and translocations). Despite the relevance of chromosomal instability as a driver for genome evolution and adaptation, little is yet known about the cellular mechanisms and processes that actually underlie these modifications. Here, in this paper, we provide a comprehensive overview of somatic and meiotic defects that lead to polyploidy or structural genome changes and discuss their relevance for plant genome evolution and speciation. In addition, we elaborate on the existence of stress-induced changes in chromosome and ploidy integrity in plants and their putative role in boosting adaptive genome evolution in hostile environments.

© 2014 The Authors. Published by Elsevier B.V. This is an open access article under the CC BY-NC-ND license (http://creativecommons.org/licenses/by-nc-nd/3.0/).

Contents

1. Introduction 11
2. Meiotic non-reduction – a major driver of polyploidization in flowering plants 13
   2.1. Sexual polyploidization through unreduced gametes 13
   2.2. Cellular mechanisms and genetic regulation of meiotic restitution in plants 13
   2.3. Meiotic restitution upon hybridization drives allopolyploid induction and speciation 15
      2.3.1. Cellular mechanism of meiotic restitution in F1 hybrids depends on parental genome divergence 15
      2.3.2. Molecular basis for meiotic non-reduction and 2n gamete formation in F1 hybrids 17
      2.3.3. Genetic factors promoting F1 hybrid-associated meiotic restitution: lessons from Triticeae 17
   2.4. Stress-induced meiotic restitution drives WGD under adverse conditions 18
      2.4.1. Stress-induced meiotic restitution in plants: cellular mechanism and molecular regulation 18
      2.4.2. Stress-induced meiotic restitution during plant evolution? 19
3. Somatic polyploidization 20
   3.1. Natural pathways for somatic polyploidy in plants 20
   3.2. Cellular mechanisms causing (pre-)meiotic endomitosis and gametophytic ploidy increase 20
   3.3. Endomitosis and evolutionary speciation 22
4. Karyotype change: aneuploidy, dysploidy and chromosome rearrangements 22
   4.1. Definitions and overview 22
   4.2. Karyotype change primarily results from non-homologous recombination events 23

* Corresponding author. Tel.: +32 9 264 60 69.
** Corresponding author. Tel.: +61 7 334 54325.
E-mail addresses: Nico.DeStorme@UGent.be (N. De Storme), annaliese.mason@uq.edu.au (A. Mason).

http://dx.doi.org/10.1016/j.cpb.2014.09.002
2214-6628/© 2014 The Authors. Published by Elsevier B.V. This is an open access article under the CC BY-NC-ND license (http://creativecommons.org/licenses/by-nc-nd/3.0/).

4.3. Molecular and cytological mechanisms for aneuploidy ... 23
4.4. Somatic aneuploidy ... 24
4.5. The relationship between chromosome rearrangements and speciation ... 24
4.6. Aneuploidy and speciation ... 24
4.7. Cryptic aneuploidy in allopolyploids: homoeologous chromosome substitutions ... 25
4.8. Dispensable, "B" and sex chromosomes ... 25
4.9. Environmental stress can trigger ploidy change ... 25
5. Conclusions ... 25
References ... 26

Definitions

Aneuploidy: Loss or gain of one or more chromosomes or chromosome segments relative to an established chromosome complement for a particular genome.
Autopolyploidy: Presence of three or more sets of homologous chromosomes or chromosomes from the same origin within a single cell or organism.
Allopolyploidy: Presence of three or more sets of chromosomes from at least two different species origins.
Bivalents: Pairs of parental chromosomes physically linked by cross-overs (e.g. chiasmata) during early stage meiotic cell division, more specifically from diakinesis up till metaphase I.
Cytological: Relating to the cell.
Cytomixis: Transfer of DNA or genetic material from cell to cell through cell wall channels.
Cytological diploidization: The process by which regular bivalent chromosome pairing arises or is enforced during meiosis after polyploidization.
Dysploidy: Rearrangement of DNA and sections of chromosome within an established chromosome complement without loss or gain of DNA, but with change in chromosome number.
First Division Restitution: Failure of meiosis to separate homologous chromosomes into separate daughter nuclei, yielding 2n gametes that largely maintain parental heterozygosity.
Gamete: A reproductive cell (e.g. ovum or sperm), generally with half the number of chromosomes present in the parent somatic tissue.
Homeology: Similarity in DNA sequence between chromosomes with shared ancestral homology that do not behave as homologous chromosomes.
Homology: Similarity between whole chromosomes or regions of DNA, usually referring to similarity in DNA sequence and gene order between chromosomes originating from the same species.
Inbreeding depression: Accumulative concentration of deleterious recessive alleles and/or increasing homozygosity resulting in negative phenotypic outcomes.
Interspecific hybridization: Sexual reproduction involving two gametes from different species origins.
Karyotype: The set of chromosomes present within a cell.
Kinetochore: Protein complex that localizes to the chromosome's centromere and that functions as a linker between the chromocenter and the spindle microtubuli, enabling chromosome segregation in meiosis as well in mitosis.
Laggard chromosome: A chromosome left behind on the metaphase plate after anaphase separates chromosomes into daughter nuclei: such chromosomes may either be excluded from daughter nuclei or rejoin forming daughter nuclei later than other chromosomes.
Meiocyte: A cell performing meiotic cell division.
Meiotic restitution: Failure of meiosis to produce gametes with half the chromosome complement of the parent individual, resulting in gametes with the somatic chromosome number.
Microtubule: Proteinaceous cylindrical structures, composed of tubulin components, that are distributed in the cytoplasm of eukaryotic cells, providing structural support and mediating subcellular locomotion and transport microtubule through end processing.
Multivalent: Physical linkage of three or more chromosomes at the meiotic diakinesis-metaphase I stages through establishment of genetic cross-overs or chiasmata.
Neo-functionalization: The acquisition of novel function in one of a pair of genes resulting from a gene or a whole genome duplication event.
Polyploidy: The presence of more than two sets of chromosomes within a single cell or organism.
Somatic: Of the body, not in the germline.
Translocation: The movement of a chromosome segment to a different genomic location, usually via a non-homologous recombination event.
Transposable elements: Specialized pieces of DNA that are able to either reproduce copies of themselves to insert elsewhere in the genome or that are able to excise and to move to different genomic locations.
Second Division Restitution: Failure of the meiotic cell division to separate sister chromatids into separate daughter nuclei, yielding 2n gametes in which parental heterozygosity is largely converted into homozygosity.
Sub-functionalization: The joint functionality of a pair of duplicated genes.
Univalent: An unpaired chromosome at metaphase I of meiosis.
Unreduced gamete: A gamete that contains the same chromosome complement as somatic cells of the parent individual, instead of the normally reduced gametophytic chromosome number.
Whole genome duplication: An event resulting in the presence of two copies of every chromosome and duplication of all genetic information within a single cell or individual.

1. Introduction

Flowering plants (angiosperms) show a high level of biodiversity, comprising an estimated number of 352,000 species subdivided in 14,559 genera and 405 families [1]. From an evolutionary perspective, this high level of variability indicates that plants have undergone extensive diversification and adaptive radiation, progressively generating new species adapted to a multitude of environments [2–5]. As sessile organisms, land plants require enhanced phenotypic adaptability and flexibility to cope with highly variable external parameters, such as extreme climate conditions, nutrient deprivation and ecosystem competition. On the genomic level, plant diversity correlates with a high degree of variation in overall genome size, ploidy level and chromosome number [6,7]. Known plant genome sizes range from 1C = 0.0648 pg in *Genlisea margaretae* to 1C = 152.23 pg in *Paris japonica* [8]. From an evolutionary standpoint, this extreme genomic variability results from a long lasting process of genome adaptation and change [9]. Much of this genomic variation is due to the action of transposable elements [10]; however, of potentially more functional importance are the cytological mechanisms permitting interspecific

hybridization, polyploidization and genome change through meiotic and mitotic mechanisms. Numerous types of chromosomal adaptations and ploidy alterations result from aberrations in the ubiquitous meiotic and mitotic processes, including whole genome duplications and chromosome rearrangements. Increasingly, these processes are found to provide underlying mechanisms for plant speciation, particularly in response to environmental change [11–13].

Recently, a great deal of attention has been paid to the role of individual gene doubling and whole genome duplication (WGD) events in speciation, particularly in plants. The process of WGD has thereby been proposed to act as a major source of evolutionary genomic variability and plasticity, hence constituting one of the main mechanisms driving diversification and speciation [9,14,15]. Indeed, several phylogenetic studies and comparative genome analyses have confirmed that most flowering plants have undergone one or more ancient WGDs early in their evolution, and that several species seem to have experienced one or more additional rounds of more recent, independent polyploidization events [16–20]. In addition, recent comprehensive phylogenomic analyses revealed the occurrence of two WGD events in ancestral plant lineages shortly before the divergence of extant seed plants and angiosperms. A common genome triplication event preceded the rapid radiation of core eudicot lineages, providing substantial evidence that gene and genome duplication and associated changes in chromosome stability have triggered evolutionary novelties and radiative adaptation, contributing to the rise and dominance of flowering plants [21,22].

In plants, like in other species, polyploid genomes are typically associated with major changes in genomic structure and phenotypic outcome, providing a broader basis for adaptivity and evolvability compared to their diploid counterparts. Studies using neo- and synthetic polyploids have revealed that polyploidy induces distinct phenotypic and morphological changes, such as differences in flowering time and flower number [23], plant structure and root architecture, as well as alterations in plant physiology, (a)biotic stress tolerance and other developmental processes [24]. Polyploidy has also been associated with increased heterozygosity, higher selfing rates, induction of asexuality and reduced inbreeding depression [25]. In the search for the putative mechanisms behind enhanced phenotypic variability of polyploids, a multitude of recent molecular and genomic studies have revealed that de novo polyploid induction causes both rapid and more prolonged changes at the genetic and epigenetic level, together with major alterations in the transcriptional landscape [26,27]. At the onset, polyploidy is associated with rapid and extensive restructuring of the genome, including profound changes in chromosome number and structure (translocations, deletions) [28–32] and epigenetic alterations, such as transposon activation, chromatin modifications and altered methylation patterning [33–38]. As a result of this initial 'genomic shock', newly formed polyploid plants often show distinct changes in their gene expression profile (e.g. gene silencing), often reflected by associated changes in the phenotype [39–42]. This initial period of genomic stress is often associated with plant lethality or reproductive sterility, largely impairing the reproductive success of the newly formed polyploidy [36,43]. Polyploids that are able to pass this initial bottleneck of genomic instability subsequently enter a second, more prolonged phase of genome evolution, whereby duplicated genes are either progressively lost [44] or retained, often showing sub- or neofunctionalization to yield novel genetic combinations and gene complexes [45–51]. As this process reduces genomic redundancy and converts the polyploid cell into a diploid one, both on the cytological and genomic level, this evolutionary process is often referred to as 'diploidization'. Hence, through the combined changes in genetic and epigenetic structure, genome duplication in the long term provides an important source of genetic flexibility, allowing an increased level of mutation, drift and selection and the associated emergence of evolutionary novelties [52]. Based on these observations, it is now generally assumed that plant evolution is characterized by repeated rounds of large-scale genome duplications (WGDs), followed by selective loss of individual genes, chromosomes or genome fragments and associated diploidization [25,53].

Historically, two types of polyploids are recognized: auto- and allopolyploids (sometimes referred to as polysomic and disomic). Although there is ambiguity about the definition of these two categories, the primary criterion for classifying a polyploid is its mode of origin. Autopolyploidy refers to polyploids originating from a polyploidization event within or between populations of a single species (intraspecific), whereas allopolyploids are the result of hybridization events between different biological species (interspecific) [25,54]. Early cytogeneticists believed chromosome pairing to be a reliable indicator of chromosome divergence and homology, using frequency of multivalent formation as a cytological parameter to distinguish between auto- and allopolyploidy [55]. Hereby, a high level of multivalent pairing in meiosis I suggested strong homology between chromosome sets and hence autopolyploidy. On the contrary, the predominant formation of bivalents was thought to result from the presence of non-homologous parental chromosome sets, hence indicating allopolyploidy. In spite of these classifications, the differentiation between auto- and allopolyploids is not absolute, since multivalent formation has been observed in hybrid polyploids, and bivalent pairing has occasionally been retrieved in intraspecific polyploids [56,57]. As a consensus and with the advent of molecular genetics, auto- and allopolyploids are now considered two extreme ends of a genome duplication-constituted continuum, in which the gradient of divergence between the parental genomes (and thus also the level of bivalent chromosome pairing) determines the level of auto- or allopolyploidy.

Comparative genomics and phylogenetic studies suggest that both auto- and allopolyploidization have played a prominent role in plant speciation and diversification. For example, DNA sequencing technology has detected remnants of interspecific hybridization events in the evolutionary history of many modern polyploid species [9,58–61]. Similarly, autopolyploid origins have been established for apple (*Malus* x *domestica*) [62] and for a triploid tropical lucerne cytotype (*Arachis pintoi*) [63]. However, despite the evolutionary relevance and occurrence, the exact mechanism(s) and cellular process(es) underlying evolutionary events of polyploid origin remain largely unknown. Currently, there seems to be a major discrepancy between studies of speciation, which tend to take an ecological and population genetics perspective, and cytological studies of mitotic and meiotic cell division and associated alterations in chromosome behavior and genome stability. Meiotic mechanisms have occasionally been investigated in relation to speciation [64,65], but experimental investigation into the role of chromosome change (aneuploidy and dysploidy) in plant speciation has traditionally lagged behind similar studies in animals [66]. However, with the advent of molecular genetics and genomics, next-generation sequencing and cytogenetic techniques, the role of chromosome change in speciation events can be more thoroughly investigated [67]. Hybridization may also occur without associated genome doubling [68] and increasing evidence suggests that hybridization without polyploidy can also play a major role in speciation events [69]. However, despite the abundant knowledge on hybridization- and polyploidization-induced genome flexibility and associated chromosome instability, the exact cytological mechanism(s) and cellular process(es) fuelling repeated boosts of diversification and speciation during plant evolution remain largely unknown.

During the last decade, a plethora of mitotic and meiotic cell division anomalies have been implicated in trans-generational

ploidy change and chromosome instability. To what extent these anomalies have contributed to plant speciation events remains largely elusive. However, recent studies provide preliminary evidence for the involvement of distinct cytological processes, strongly dependent on the type of speciation (e.g. hybridization or polyploidy) and the presence of genetic or environmental factors. In this review, we describe the three major cytological mechanisms causing ploidy change: (1) meiotic non-reduction and 2n gamete formation, (2) somatic genome duplication and (3) minor karyotype changes through aneuploidy and/or dysploidy, and outline their role as drivers of plant speciation. We focus on underlying cellular defects and associated molecular regulators and outline specific induction through genomic and environmental stresses, suggesting a role for stress-induced polyploidization and chromosome change in plant evolution. Recent advances in our understanding of the cytological mechanisms facilitating rapid chromosome change highlight an intimate association with environmental stress conditions and specific genomic conditions (mutations, hybridization), and suggest pathways for natural and induced species formation through changes in chromosome or ploidy constitution.

2. Meiotic non-reduction – a major driver of polyploidization in flowering plants

2.1. Sexual polyploidization through unreduced gametes

The meiotic cell division is a critical reproductive process, and is tightly controlled to guarantee reductional homologous chromosome segregation and subsequent formation of haploid male and female gametes. In some instances, however, alterations in the meiotic program or cellular defects in meiosis I (MI) or meiosis II (MII) may switch the meiotic cell division into a mitotic-like one, generating diploid spores out of a diploid mother cell. This mechanism is generally termed meiotic restitution or meiotic non-reduction, and the resulting gametes are referred to as unreduced or 2n gametes [70–73]. Importantly, the ectopic formation of diploid gametes, instead of the normal haploid ones, intrinsically leads to progeny with an increased chromosome number. This mechanism of genome polyploidization is termed 'sexual polyploidization' and can be subdivided into two types: bi- and unilateral sexual polyploidization [54]. In the former, fusion between two diploid gametes yields a tetraploid individual, which, depending on the selective conditions, may initiate the de novo establishment of a stable tetraploid lineage. By contrast, in unilateral sexual polyploidization events, one diploid gamete fuses with a normal haploid one to generate a triploid embryo. Although triploid seeds are often non-viable through imbalances in parental genome dosage input in the endosperm [74], this triploid block is occasionally incomplete or absent allowing triploid plant formation [75,76]. Meiotic cell division in these triploids is generally extremely unbalanced yielding aneuploid gametes. However, through random segregation triploids also produce some euploid gametes, both haploid and diploid, which may contribute to the establishment of stable polyploid populations over time [77]. This process is generally referred to as the triploid bridge hypothesis [54] and is suggested to play a role in evolutionary polyploidization events.

A large number of different cellular defects conferring meiotic restitution have been described. From a genetic point of view, these mechanisms are classically subdivided into two main groups, namely First Division Restitution (FDR) and Second Division Restitution (SDR) (extensively reviewed in Refs. [71,73,76]). In brief, SDR mechanisms yield 2n gametes that are genetically equivalent to those formed by a loss of the second meiotic cell division. SDR gametes hence contain both sister chromatids from the same chromosome and display loss of parental heterozygosity from the centromere to the first site of crossing-over (CO). In contrast, 2n gametes resulting from FDR-type meiotic restitution are genetically equivalent to those formed by a loss of MI. Hence, in FDR-type restitution, sister chromatids from the same homolog are split but homologous chromosomes are retained, and resulting gametes maintain parental heterozygosity in chromosomal regions spanning the centromere to the first CO. Interestingly, when FDR also includes a complete loss of meiotic recombination, the meiotic cell division is converted into a mitotic one, yielding 2n gametes that fully retain the parental genome constitution, including heterozygosity and epistatic interactions. This type of meiotic restitution is often observed in female gametogenesis of apomictic plants and parthenogenically reproducing animals as a reproductive adaptation to generate clonal progeny.

2.2. Cellular mechanisms and genetic regulation of meiotic restitution in plants

Detailed cytological studies in several plant species have revealed that meiotic restitution can originate from a plethora of cellular defects. These are generally subdivided into three main classes: (1) alterations in meiotic spindle dynamics; (2) defects in meiotic cell plate formation and (3) omission of meiosis I or II (reviewed in Refs. [71–73]).

Alterations in meiotic spindle dynamics occur in either meiosis I or II and are caused by structural defects in spindle biogenesis, microtubule (MT) nucleation, kinetochore functioning or spindle orientation and organization. Although these defects generally lead to imbalances in chromosome dynamics and segregation, yielding aneuploid gametes, occasionally the presence of non-separated chromosomes induces a meiotic restitution event [78]. For example in cereal hybrids, such as wheat-rye F1 plants, both the ectopic formation of curved MI spindles and defects in spindle-kinetochore attachment completely block metaphase I chromosome separation and cell plate formation, yielding restituted cells which progress through MII to form dyads containing two unreduced gametes [79]. However, despite this, meiotic restitution in plants more commonly results from alterations in the three-dimensional organization of the spindle structure(s). In meiosis I, defects in bipolar spindle orientation either partially or fully omit polar-directed chromosome segregation, yielding restituted MI nuclei capable of undergoing meiosis II [80]. In meiosis II, alterations in the spatial positioning of the two metaphase spindles may lead to a rejoining or even a non-disjunction of the two haploid chromosome sets, eventually yielding restituted nuclei [81]. Particularly in male meiocytes of dicotyledonous plants, proper perpendicular orientation of the two MII spindles is crucial for correct chromosome segregation and meiotic ploidy reduction [82,83]. Indeed, the ectopic induction of tripolar (tps) and parallel (ps) or fused (fs) spindles has been found to reduce MII polarity from tetrahedral to tri- or bipolar, respectively, so that MII spindles rejoin chromatids at one or both poles, generating meiotically restituted FDR-type 2n gametes [84,85]. Interestingly, in most cases, ps, fs and tps are jointly observed in the same flower [82,83,86,87], indicating that all three processes constitute a different outcome of one common cellular defect. However, up till now, the underlying mechanism has not been revealed yet.

Studies in potato and *Arabidopsis thaliana* have revealed a genetic background for the *ps/tps/fs* male meiotic defect and resulted in the identification of several proteins required for MII spindle polarity, including JASON, AtPS1 (*A. thaliana* PARALLEL SPINDLES 1) and AFH14 [82,83,88]. Mutant forms of these proteins induce the formation of dyads and triads in male meiosis through alteration of MII spindle orientation, yielding 2n gametes capable of inducing sexual polyploidization. AFH14 is an *Arabidopsis* type II formin (FORMIN 14) that functions as a linking protein between

microtubules (MTs) and microfilaments (MFs) [88]. AtPS1 encodes an unknown plant-specific protein with an N-terminal Forkhead Associated (FHA) domain and C-terminal PINc domain, typically involved in protein-protein interactions and RNA processing and decay (i.e. Nonsense mediated mRNA decay), respectively [82]. JASON, on the other hand, is an unknown, plant-specific protein that positively regulates *AtPS1* transcript levels in early stage flower buds, suggesting that JAS controls MII spindle organization through AtPS1 [83].

Ps-induced 2n gamete formation has already been documented in several plant species and is hence considered one of the major routes for 2n gamete formation and sexual polyploidization in plants. Whether the meiotic ps defect and one or more underlying causative mutations has driven WGD in the evolution of sexually reproducing plants remains unknown. Interestingly, studies in potato revealed that tetraploid cultivars and related wild taxa contain a higher *ps* allele frequency compared to the ancestral diploid population, indicating that *ps* and the associated formation of 2n gametes has been the driving force behind the origin of cultivated tetraploid potatoes [89,90]. Although not yet demonstrated in other naturally evolved populations, this study shows that *ps* and other 2n gamete-forming mutations may have laid the basis for evolutionary WGD events and associated speciation and diversification in plants.

A second type of meiotic restitution in plants involves alterations in meiotic cytokinesis. Defects in meiotic cell plate formation either originate from (1) precocious induction of cytokinesis or (2) partial or complete loss of meiotic cell plate formation, either after MI or MII [73,84,91,92]. In both cases, physical separation of nuclei following MI or MII is affected, with two or more haploid nuclei enclosed in a common cytoplasm. Subsequent fusion of syncytial nuclei in these bi- or polynuclear cells eventually yields diploid or polyploid spores, forming a sexual basis for whole genome doubling [93–96].

Precocious induction of meiotic cell wall formation is only sporadically reported, either in MI [95] or MII [85], and hence not considered an important mechanism for meiotic restitution. In contrast, unreduced gamete formation through loss of cell plate formation has frequently been observed in different plant species [78,91,95–98], suggesting it is an important cellular mechanism driving sexual polyploidization. In meiocytes with a successive-type of cell division, loss of cell plate formation may either occur after MI or MII, generating FDR- or SDR-type 2n gametes, respectively [93,99]. In simultaneous-type PMCs, which form a “double wall” at the end of MII, loss of meiotic cell wall formation may either be partial or complete, yielding diploid, triploid or tetraploid gametes [96].

From a mechanistic point of view, loss of meiotic cell plate formation can result from several types of cellular anomalies, including alterations in microtubule (MT) array biogenesis or stability [100,101], defective transport of cell wall material, disturbed membrane vesicle fusion [102] and reduced deposition of callose [103]. For example, several studies using *A. thaliana* mutants have revealed that structural or functional irregularities in the establishment of internuclear radial microtubule arrays (RMAs) at the end of MII causes defects in cytokinesis, hence yielding bi- or polynuclear spores [100,101,104,105]. In addition to these ‘basal’ cytokinetic defects, alterations in meiotic cytokinesis may also occur as a secondary effect resulting from irregularities in spindle elongation or orientation. Indeed, studies on the maize MATH-BTB domain protein MAB1 revealed that the shorter spindles in *mab1* RNAi meiocytes cause an insufficient separation of telophase II nuclei, impairing subsequent internuclear cell wall formation and hence generating bi- or polynuclear spores [106]. Moreover, an extensive analysis of MT structures in potato and *Populus* meiocytes demonstrated that alterations in tetrahedral MII nuclei positioning through irregularities in spindle orientation cause defects in interzonal RMA formation, indicating that meiotic non-reduction by ps, fs and tps actually results from ‘secondary’ defects in MII cell plate formation [85,92].

Thirdly, meiotic restitution may also originate from a complete omission of one of the meiotic cell divisions. In the case of loss of MI, both the processes of meiotic recombination and reductional cell division are omitted, and MII separates sister chromatids into two diploid FDR-type daughter cells, genetically identical to the parental line [107–110]. This process of clonal gamete formation is referred to as diplosporous apomeiosis [111,112] and is often observed in apomictically reproducing species, both in plants as well as in other eukaryotic clades [113–115]. Contrary to loss of MI, failure of MII still enables homologous recombination and MI chromosome segregation, however, separation of sister chromatids does not occur. Instead, centromeric cohesion is lost at the end of MII, yielding dyads that contain SDR-type 2n gametes [109,116].

In sexually reproducing species, loss of meiotic cell division has also repeatedly been observed [107,117–120], and hence may be considered an alternative mechanism driving natural polyploidization. Moreover, apomeiosis and clonal 2n gamete formation is the rule in apomictically reproducing species, indicating that the developmental switch from meiosis to apomeiosis forms a natural pathway for plant reproductive evolution [121,122]. In support of this, phylogenetic studies revealed that in many species apomixis evolved repeatedly from sexual pathways and this in several independent origins [123,124]. More importantly, in most species apomixis has been found strongly correlated with genomic instability and polyploidy [25,125,126], suggesting that the process of apomeiosis not only confers asexual reproduction but also induces transgenerational ploidy increase, and hence drives evolutionary polyploidization [127,128]. Although still under debate, it is postulated that polyploidy hereby functions as a genomic stabilizing factor, reducing the impact of deleterious mutations in the short term and hence providing a selective advantage over diploid apomicts [129–131].

Progression of the meiotic cell cycle and consolidation of reductional division in sexually reproducing species is tightly controlled by a complex network of (epi-)genetic factors [132,133]. Interestingly, genetic defects in some of these regulators have been found to induce meiotic restitution and 2n gamete formation (extensively reviewed in Refs. [72,73]), indicating that (epi-)genetic defects in meiotic cell cycle regulation may constitute a basis for sexual WGD. For example, genetic studies in *Arabidopsis* have identified two proteins, e.g. TAM (TARDY ASYNCHRONOUS MEIOSIS) and GIG1/OSD1 (GIGAS CELL1/OMISSION OF MEIOTIC DIVISION1) that are required for progression of meiotic cell division, and more specifically the MI-to-MII transition. As a result, functional loss of one of these proteins causes a complete omission of MII, generating dyads that contain SDR-type 2n gametes [109,116]. GIG1/OSD1 is an inhibitor of the Anaphase Promoting Complex/Cyclosome (APC/C) and functions in the maintenance of elevated CDK (CYCLIN-DEPENDENT KINASE) levels after MI [134,135]. TAM, on the other hand, encodes an A-type cyclin, e.g. CYCA1;2, that forms a functional complex with CDKA;1, to regulate meiotic cell cycle progression [116,136]. Besides regulators of MI-to-MII cell cycle transition, several other proteins implicated in the initiation of meiotic cell division, e.g. the mitosis-to-meiosis switch, have been identified. These include the *Arabidopsis* meiotic prophase I protein DYAD/SWITCH1 [108,137,138], the APOLLO (APOmixis-Linked Locus) histidine nuclease recently identified in Boechera [139], the maize DMT102 and DMT103 DNA-methyltransferases [140], the maize AGO104 (ARGONAUTE 104) protein [110] and its *Arabidopsis* ortholog AGO9 [141] and other proteins acting in the 24 nucleotide siRNA-mediated silencing pathway, such as RNA-dependent RNA polymerase 2 and 6 (RDR2 and RDR6), SUPRESSOR of GENE

SILENCING 3 (SGS3), DICER-LIKE 3 (DCL3) and POLYMERASE IV and V (NRPD1a and b). Functional loss of function of each of these proteins induces a complete omission of MI, yielding meiocytes that skip recombination and reductional cell division and directly undergo equational cell division to produce clonal 2n megaspores. Genetic analyses revealed that all these proteins are either involved in the regulation of MI chromosome dynamics [142,143] and histone patterning [144], small RNA-mediated signalling and gene silencing or DNA methylation [140], indicating that meiotic induction and the meiosis-apomeiosis decision is under a strong epigenetic control (extensively reviewed in [73]). All together, these findings demonstrate that alterations in the (epi-)genetic machinery controlling reproductive pathways, such as initiation of meiosis and regulation of meiotic cell cycle progression, may cause meiotic non-reduction (e.g. loss of MI or MII), hence forming a molecular trigger for the formation of 2n gametes capable of conferring sexual polyploidization. However, whether such aberrations have actually contributed to evolutionary relevant polyploidization and speciation events other than induction of apomictic reproduction remains unknown.

2.3. Meiotic restitution upon hybridization drives allopolyploid induction and speciation

Many polyploid speciation events involve the intercrossing of two closely or more distantly related species to obtain a stable allopolyploid lineage. At the genomic level, these 'polyploid hybrids' benefit both from fixed heterozygosity as well as from chromosome redundancy, providing them an increased genomic flexibility upon which selection can act. Studies in wheat and *Brassica* have also revealed that neo-allopolyploids display rapid and pervasive alterations at the DNA sequence and epigenetic profile level [145–147], including alterations in DNA methylation patterning [148,149], reciprocal translocations [30], insertions/deletions, elimination of low-copy non-coding DNA sequences [150], aneuploidy [151,152] and loss of 5S DNA unit classes [153]. This (epi-)genetic variability and resulting transgressive segregation is thought to provide allopolyploids a strong evolutionary advantage, which may explain their widespread occurrence, in natural as well as agronomic populations. A substantial amount of research has been performed in search of the cellular mechanism(s) underlying allopolyploid origin. The very early maxim of "hybridization followed by genome doubling" put forward by Ö. Winge (1917) was repudiated by (1975) [154], who made the first strong case for the involvement of meiotic mechanisms, and in particular unreduced gametes, in allopolyploid formation events. Subsequently, increasing evidence has supported the role of meiotic non-reduction and sexual polyploidization in F1 hybrids as a major route for allopolyploid formation (comprehensively reviewed by Ramsey and Schemske [54]).

F1 plants resulting from wide hybridization events generally produce non-viable gametes due to instabilities in meiotic chromosome segregation and gametophytic aneuploidy. These F1 meiotic defects typically originate from irregularities in MI homologous chromosome pairing; a process that strongly depends on the sequence similarity of the two parental genotypes [155–157]. If homologous chromosome pairs are not present, as is the case in wide hybridization events (e.g. genome composition AB), pairing of homoeologous chromosomes is strongly disrupted and achiasmate univalents instead of recombining bivalents are formed at metaphase I. Due to the absence of bivalent-based bipolarity, univalents segregate randomly at anaphase I, yielding unbalanced MI products that develop into aneuploid gametes [158], similar as in a- and desynaptic mutants and haploid lines [159–161]. Although the induction of gametophytic aneuploidy may occasionally lead to variations in chromosomal structure and copy number [162], resulting gametes are generally non-viable [158]. Strikingly, despite this meiosis-based gametophytic sterility, F1 hybrids generally still produce a small or sometimes large number of seeds, which in most cases have a duplicated chromosome number [163–166]. Cytological and genotypic analysis of de novo F1 amphi- or polyhaploids revealed that this is caused by the induction of meiotic restitution and the associated formation of unreduced gametes [163,167–171]. Importantly, studies in several plant species have revealed that the exact mechanism of meiotic restitution differs by hybrid type, largely depending on the level of homologous chromosome pairing and hence on the relatedness of the original parent lines.

2.3.1. Cellular mechanism of meiotic restitution in F1 hybrids depends on parental genome divergence

In the case of hybridization between two remote genotypes, such as wheat-rye and other cereal wide crosses, resulting F1 hybrids shows a complete lack of pairing and crossing-over, yielding univalents instead of bivalents at metaphase I (Fig. 1). Cytological analysis revealed that these univalents either show a unidirectional segregation to one pole, yielding an asymmetrical dyad composed of one anucleate cell and one cell with a restituted nucleus, or alternatively display chromosome lagging and thus remain positioned at the cell equator [80,164,172]. Alternatively, in some cases, MI shows a retrograde migration of telophase I chromosomes from the poles back to the center of the cell [79]. In all these cases, MI yields a non-reduced diploid cell that progresses through the second meiotic cell division to form a dyad containing two unreduced 2n gametes [173]. Interestingly, studies in *Lilium* interspecific hybrids and haploid *Arabidopsis* have revealed that univalents have the potential to divide equationally during MI, indicating that the complete loss of bivalent formation may convert the double meiotic cell division into a single mitotic one [174,175]. This process is typically referred to as a 'single-division meiosis' (SDM) [173,176] or a 'mitotic-like division' [169,170]. However, in a strict sense, SDM represents an extreme form of univalent-induced delay of MI chromosome segregation, completely impairing distinction between the two meiotic cell divisions and hence mimicking a mitotic cell division. In support of this, SDM and FDR have often been found to co-exist in F1 amphihaploids [165,173]. Interestingly, a similar type of meiotic restitution also occurs in asynaptic accessions and (poly-)haploid lines that show a complete loss of homolog interaction and bivalent formation [102,164,177,178], indicating that meiotic restitution is directly due to structural alterations in MI chromosome segregation and is not per se caused by genetic defects that alter meiotic cell division. Since in all these cases meiotic restitution involves a complete loss of MI, including both reductional cell division and recombination (FDR-type *sensu strictu*), resulting gametes are genetically identical to the parent and hence generate an autopolyploid version of the original hybrid. Despite their hybrid origin, these so-called strict allopolyploids act as homozygous diploids with strict bivalent chromosome pairing and disomic inheritance, as for example demonstrated in newly formed *Arabidopsis suecica* (2x = 26) allopolyploids [179].

In the case of intercrossing two more closely related species that share a certain level of genomic sequence similarity (e.g. homeology), resulting F1 hybrids may also exhibit events of meiotic restitution and 2n gamete formation, albeit at a significantly lower rate compared to hybrids with more divergent genomes [180,181]. From a cytological perspective, the mechanism of meiotic non-reduction is highly similar to the one observed in (amphi-)haploid meiosis, with lagging chromosomes remaining at the MI equatorial cell plate that eventually restitute the first meiotic cell division. However, in hybrids with homeologous subgenomes, the genotypic constitution of resulting 2n gametes differs significantly from the ones produced by a strict FDR-type mechanism, as

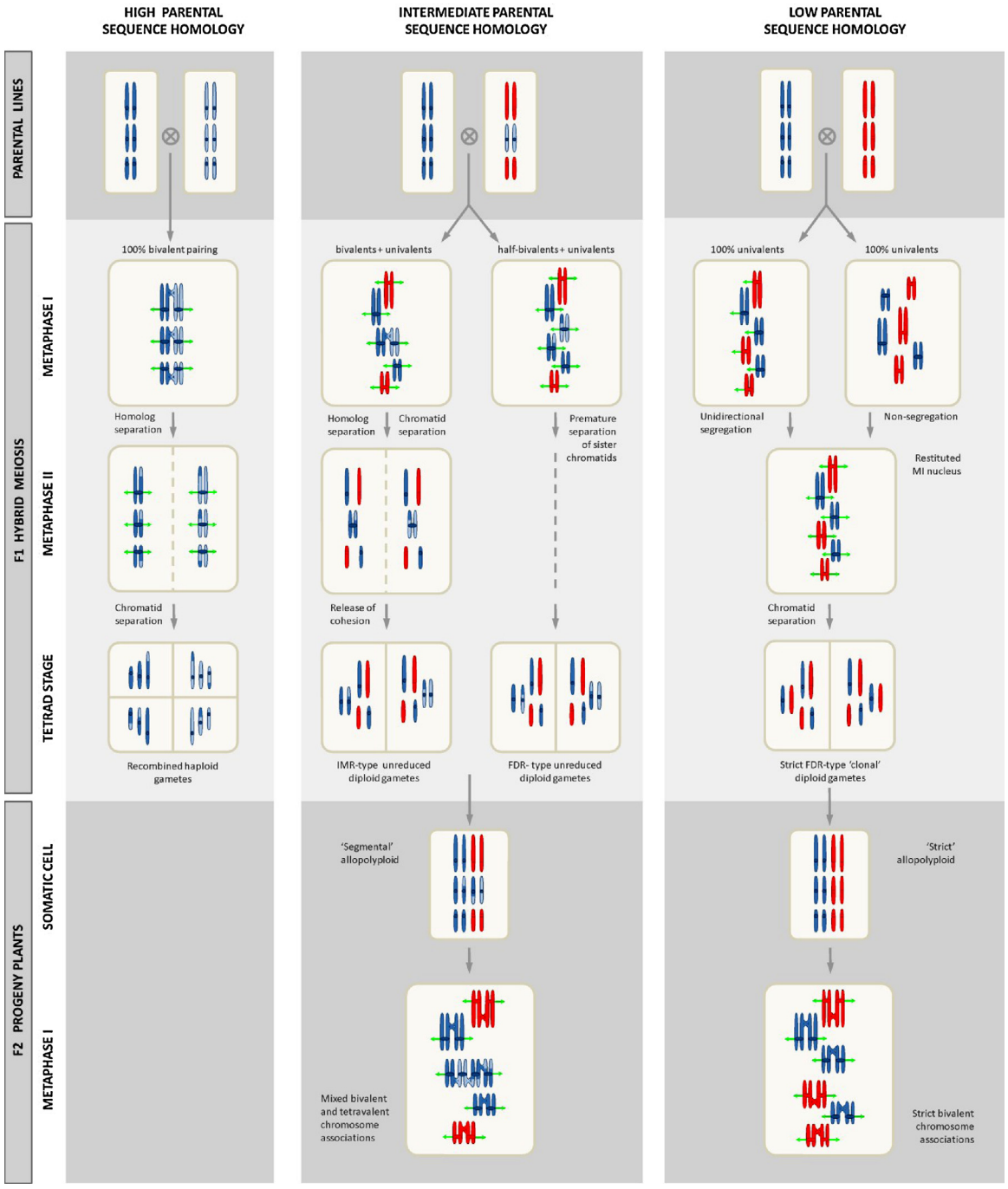

Fig. 1. The impact of sequence homology between parent progenitor genomes on meiotic restitution in their F1 progeny.

pairing and recombination may occur between (some) homeologous chromosomes. This behavior is exemplified by the presence of bivalents and/or multivalents, physically exchanging genetic material, amongst the other non-recombining univalents [63,182,183]. Depending on the exact mode of chromosome segregation and more specifically on the timing of bivalent dissociation, two putative mechanisms of meiotic restitution are possible, each producing 2n gametes that slightly differ in their genetic make-up. In case of a premature loss of bivalent association, for example in a prolonged metaphase I state, both univalents and half-bivalents exhibit an equational cell division, yielding FDR 2n gametes that largely maintain parental heterozygosity, except for the regions that have undergone reciprocal recombination (non-strict FDR). Alternatively, when bivalents maintain chiasmatal links up till anaphase I and properly attach to the bipolar spindle, meiotic non-reduction typically involves a reductional division of bivalents together with an equational segregation of univalents, giving rise to unreduced gametes that do not comply to an FDR-type, but instead are equivalent to a so-called indeterminate (IMR)-type of meiotic restitution [73,174]. Importantly, besides parental genome exchange through homeologous recombination, genetic variability in IMR 2n gametes is also increased by the dissimilar transmission of parental chromosomes [174]. Due to the partial homology of the parental taxa, resulting allopolyploids of both types of meiotic restitution (i.e. non-strict FDR and IMR) show a combination of bivalent and quadrivalent MI chromosome pairing, hence conferring a mixture of di- and tetrasomic inheritance. These polyploids, often referred to as segmental allopolyploids [184], form an

important part of the current natural and agronomic polyploid population, e.g. as shown by several cytological, molecular and genome sequencing studies [152,185–188] providing substantial evidence for the occurrence of both FDR- and IMR-type restitution, including partial homeologous recombination, in allopolyploid plant speciation [29].

All together these findings support the hypothesis that meiotic restitution upon hybridization forms an important driver for allopolyploid origin, and additionally indicate that the frequency and type of meiotic restitution (FDR *sensu strictu*, FDR- and IMR-type) strongly depends on the parental genome constitution, and more specifically on DNA sequence divergence and structural dissimilarities. As a general rule, increasing divergence of the parental genomes progressively hinders homeologous pairing and recombination, inducing FDR- and IMR-type meiotic restitution in meiocytes containing partially divergent genomes and FDR *sensu strictu* when genomes are highly divergent. Although the presented model for allopolyploidization most likely presents an oversimplified view, excluding the role of gene dosage and transcriptional and genetic alterations, the basic principle of hybridization-induced meiotic restitution may constitute a general system explaining allopolyploid origin (strict and segmental) during plant evolution. In support of this, Hunter et al. [189] demonstrated that progenitors of polyploid hybrids show significantly higher genetic divergence than those underlying homoploid hybridization, confirming the notion that parental genome divergence in a hybridization context drives whole-genome duplication and hence allopolyploid speciation.

2.3.2. Molecular basis for meiotic non-reduction and 2n gamete formation in F1 hybrids

From a mechanistic point of view, it is suggested that the induction of meiotic restitution and the frequency of its occurrence in F1 hybrids largely depends on the presence of univalents and hence on the lack of pairing caused by sequence non-homology, rather than on specific genetic defects [164]. In support of this, Wang et al. [190] found that F1 hybrids resulting from allotetraploid *T. turgidum* x tetraploid *Ae. tausschii* crosses (yielding ABDD genotypes) do not undergo meiotic non-reduction, whereas their triploid polyhaploid variants (ABD genotype) do, indicating that homologous pairing interferes with induction of meiotic restitution. In agreement with this, induction of pairing and bivalent formation in wheat/rye F1 hybrids, by using wheat parents with single rye chromosome substitutions, caused higher preference for the reductional, meiotic-like pathway, whereas a complete failure of bivalent pairing induces meiotic restitution and 2n gamete formation [191]. Similarly, studies in durum wheat revealed that the univalent-associated induction of meiotic restitution normally occurring in synthetic haploids [164] is impaired in 5D-5B chromosome substitution haploids, due to the induction of homeologous chromosome pairing and bivalent formation (by absence of the *Ph1* locus) [192]. In contrast to this hypothesis, Pignone [193] found that amphihaploid *T. turgidum* x *Ae. longissima* hybrids (ABS^1, 2n = 21) and the corresponding backcrosses to *T. turgidum* (AABBS1, 2n = 35), which demonstrate a high level of bivalent formation, both exhibit an equational division of univalents, indicating that in some cases genetic factors or other parameters may be involved in the induction of meiotic non-reduction.

As an underlying molecular basis, it has been suggested that increasing deviations in DNA sequence similarity between parental chromosome sets may impair the single strand-based DNA homology search in prophase I, reducing the number of D-loop structures that form a transient interhomolog linkage and that are required for homologous pairing, synapsis and recombination [194,195]. Alternatively, divergence in sequence homology may alter chromatin remodelling capacities during early meiotic stages, hence affecting the process of pairing and recombination [196,197]. In support of this, studies in wheat-rye hybrids revealed that perturbations in hom(e)ologous chromosome pairing are closely associated with asynchronies in prophase I chromatin condensation and failure in heterochromatin change [198,199] and eventually induce meiotic restitution and 2n gamete formation [172]. Additionally, chemical induction of chromosome condensation has been found to induce homeologous pairing in wheat interspecific hybrids, indicating that a synchronized change in chromatin remodelling is essential for MI reductional cell division [200]. Related to this, Rezaei et al. [201] suggested that meiotic instabilities in triticale are caused by structural differences in parental chromatin configuration, with rye displaying large telomeric blocks of heterochromatin and wheat showing smaller and intercalary band of heterochromatin. Since alignment of meiotic homologues generally initiates from the (sub-)telomeric regions [202], differences in chromatin state may hence impair hom(e)olog recognition and pairing [197], thereby yielding achiasmate chromosomes capable of inducing meiotic restitution. Alternatively, loss of reductional cell division in F1 hybrids may also result from defects in kinetochore functioning and associated delay in meiotic cell cycle progression or by inactivation of MTs or kinetochores due to structural and/or functional incompatibilities in the amphihaploid MI chromosome set [172]. In support of this, Cai et al. [175] revealed that tetraploid wheat cv. 'Langdon' (LND) displays a syntelic orientation of sister kinetochores at MI, whereas its polyhaploid variant and interspecific hybrids with *Ae. tauschii* display an amphitelic orientation, conferring bipolar segregation of sister chromatids instead of hom(e)ologs. The tension created by this amphitelic orientation of sister kinetochores, together with the persistence of centromeric cohesion up till anaphase II, is thereby suggested to form a mechanistic basis for the onset of 'SDM' meiotic restitution. Moreover, since amphitelic association was only observed in asynapsed chromosomes, Cai et al. [175] suggested that synapsis acts as the predominant factor in determination of MI kinetochore orientation, and as such forms an important structural factor in the decision whether to divide reductionally or equationally. This is in agreement with the observation that a high level of chromosome pairing and synapsis prevents meiotic non-reduction and 2n gamete formation [190]. In support of this, Ressurreição et al. [203] found that the induction of asynapsis in the N5DT5B variant of Chinese Spring wheat (5D nullisomic and 5B tetrasomic, absence of the *Lpt* gene) [204] by low temperatures induces meiotic non-reduction, even when the two homologs are present. These findings provide strong evidence that the absence of synapsis rather than the haploid condition is the key feature switching syntelic to amphitelic kinetochore attachment and eventually inducing meiotic restitution in wheat. Moreover, since N5DT5B shows a reduced level of crossing-over [205], haploidy-dependent induction of meiotic restitution, as for example observed in newly formed F1 hybrids and other amphihaploids, may be directly attributable to defects in synapsis and recombination, typically occurring as secondary effects of alterations in meiotic homo(eo)logous recognition and pairing.

2.3.3. Genetic factors promoting F1 hybrid-associated meiotic restitution: lessons from Triticeae

Besides spontaneously occurring meiotic non-reduction, several studies have demonstrated the existence of genetic factors that induce/enhance the level of meiotic restitution upon hybridization. Particularly in the Triticeae tribe, genetic influence of the parents on the genome doubling capacity of resulting F1 hybrids has repeatedly been documented. A recent study of more than 100 types of *T. turgidum* x *Ae. tausschii* combinations revealed a high variability in selfed seed set, reflecting genetic differences in the *T. turgidum* germplasm to induce meiotic restitution [180]. This is in agreement with earlier reports, which demonstrate a high

variability in chromosome doubling capacity of specific tetraploid wheat varieties upon hybridization with *Ae. tausschii* [206,207]. Genetic studies hereby found that the durum wheat cultivar Langdon (LDN) carries a gene for meiotic restitution, causing high frequency FDR and partial fertility in hybrid combinations with rye and *Ae. squarrosa* [208]. Based on observations using LDN durum D-genome disomic substitution lines, Xu and Joppa [209] found that the underlying FDR-inducing gene is most likely located on chromosome 4A, however, the exact gene and underlying molecular mechanism has not yet been identified. Similarly, Zhang et al. [169] found that emmer wheat also induces high level FDR-type meiotic restitution in F1 hybrids with *Ae. tauschii* and that this is controlled by one or more nuclear genes. Although little is known about the function of these *Triticum* 'meiotic restitution' genes, the finding that 2n gamete formation is only promoted in an (amphi)haploid background and not in the usual diploid state Zhang et al. [169] suggests that these genes may only function in the partial or complete absence of chromosome pairing.

Interestingly, besides emmer and durum wheat, *Ae. tauschii* also harbors some level of natural variability in its genome doubling capacity upon hybridization with *T. turgidum* [210,211]. Using two representative *Ae. tausschi* accessions, an intensive QTL mapping approach hereby identified six QTLs that positively regulate F1 genome doubling and fertility recovery [212]. Although these QTLs may harbor genes implicated in different reproductive activities, Matsuoka et al. [212] argued that most if not all QTLs are involved in the regulation of meiotic non-reduction, with two QTLs containing putative 'meiotic restitution' genes; namely *Taf1* which is involved in female sterility [213] and *Ph2*, which is a suppressor of homeologous pairing. *Ph2* is part of the pairing homoeologous (*Ph*) gene system in wheat, which negatively regulates interactions between non-homologous chromosomes (A, B, D, etc.), thereby ensuring diploid-like meiosis and disomic inheritance in polyploid genomes. The *Ph* system consists of a major pairing locus, e.g. *Ph1*, on chromosome 5B [214,215], an intermediate one, e.g. *Ph2*, on 3D [216,217] and several additional minor loci [218,219]. Interestingly, loss of *Ph1* in amphihaploid genome combinations and associated induction of homoeologous pairing has been found to reduce the capacity of WGD in corresponding hybrids, whereas its presence significantly enhances meiotic restitution [168,220], indicating that *Ph1* and related genes may be important drivers for F1 amphihaploid meiotic non-reduction and allopolyploid induction. Recently, genetic studies identified *Ph1* as a major regulator of meiotic CDK activity [221–223], with major implications in early MI stage chromatin remodelling and induction of synapsis [196,222,224–226], whereas the intermediate pairing locus *Ph2* positively controls progression of synapsis [227], providing a molecular basis for the regulatory function of *Ph*-related genes in the induction of meiotic restitution and 2n gamete formation.

Recently, studies using haploid variants of wheat-rye substitution lines revealed interesting behaviors related to meiotic restitution. Silkova et al. [228] thereby found that a 6R(6A) substitution induces equational-type division of univalents leading to meiotically restituted dyads, whereas other lines (2R(2D)1 and 2R(2D)3) exhibit a reductional-type of meiotic chromosome segregation. These findings indicate the existence of one or more 'promotive or suppressive meiotic restitution genes' on chromosomes 6R and 2R, respectively. In line with this, rye chromosome 2R has already been found to act as a suppressor of meiotic non-reduction in wheat-rye polyhaploids [229]. Although a putative role for the underlying genes in the structural and functional organization of centromeres and associated kinetochore orientation has been postulated, corresponding genes and associated molecular mechanism(s) have not been identified yet. It is possible, however, that both the 6R and 2R genes have an impact on homoeologous pairing and recombination, and hence act as indirect molecular regulators of haploid-induced meiotic restitution, similar to *Ph* in wheat.

All together, these data suggest that loci involved in the suppression of homoeologous pairing, e.g. through chromatin remodelling and initiation of synapsis, may be involved in the induction and/or promotion of meiotic restitution and sexual polyploidization in F1 hybrids. However, whether these loci actually operated as genetic drivers of hybrid polyploidization, fuelling allopolyploid speciation, remains unknown. Classically, *Ph1* and related loci are thought to result from mutations occurring after allopolyploid origin, i.e. following hybridization and polyploidization, as a mechanism to stabilize newly formed allopolyploid genotypes by cytological diploidization of meiotic cell division [221]. However, based on the above mentioned observations, one could assume that *Ph1* or other loci suppressing homeologous interaction, were already present in the parental lines and hence significantly promoted sexual polyploidization in newly formed hybrids through the inherent induction of meiotic restitution (e.g. because of absence of pairing). Moreover, since this process directly produces neo-allopolyploids with an inherent suppression of homeologous chromosome pairing, this theory not only implicates an enhanced success rate of allopolyploid origin but also provides a mechanism for reducing adverse meiotic irregularities and genomic instabilities in early stage allopolyploid speciation [25,30,230,231], substantially promoting its fitness and establishment. In support of this, several authors have suggested the existence of *Ph*-like genes in diploid wheat species [182,232,233], thereby postulating that they only became effective in amphi(ha)ploid situations as a result of hybridization or polyploidization [234]. Similarly, standing variation for the ability to suppress homeologous recombination in newly formed hybrids has also been reported in *Lilium* diploids [235]. Interestingly, a similar theory has recently been postulated for the origin and evolution of autopolyploids. Based on an extensive genome sequence analysis Hollister et al. [230] found that the AaASY1 allele, i.e. one of the alleles reducing pairing frequency in *Arabidopsis arenosa* tetraploids [236], occurs at very low frequencies in the corresponding diploid cytotype, indicating that it may have formed a genetic basis for promoting ancient polyploidization events, thereby inherently providing a molecular basis for the diploidization and hence stabilization of autopolyploid meiosis.

2.4. Stress-induced meiotic restitution drives WGD under adverse conditions

2.4.1. Stress-induced meiotic restitution in plants: cellular mechanism and molecular regulation

In flowering plants, the reproductive pathway and particularly the process of male gametogenesis is highly sensitive to abiotic stresses. Indeed, several studies on different types of plants have revealed that adverse environmental conditions, such as heat, cold, drought and salt stress have a detrimental effect on male spore formation and pollen maturation, significantly affecting male fertility and seed set [237]. In most cases, stress-induced male sterility is caused by a precocious or delayed programmed degeneration of the tapetal cell layer, i.e. the surrounding cell layer that nurtures the developing microspores, and associated changes in microspore and pollen homeostasis [238]. Alternatively, under certain stress conditions, male sterility is directly caused by alterations in sugar metabolism, impairing proper energy supply to the developing microspores, and other functional irregularities that lead to a failure of gamete formation and/or fertilization (reviewed in [237]).

Interestingly, despite the overall negative impact of abiotic stress on sporogenesis and reproduction, under certain instances (a)biotic stress alters the process of gametogenesis in such a way that it promotes genetic flexibility and evolutionary adaptiveness of the resulting progeny. More specifically, there is accumulating

evidence that temperature stress, and putatively other stresses, induces or enhances meiotic non-reduction and the associated formation of 2n gametes [239], hence forming a basis for stress-induced sexual polyploidization events. In rose (genus *Rosa*), for example, short periods of heat stress (e.g. 48 h at 36 °C) ectopically induce the formation of parallel and tripolar MII spindles, instead of the normal perpendicular ones, producing dyads and triads that contain FDR-type 2n gametes [240]. These heat-induced changes in MII spindle orientation can be caused by alterations in the structural set-up of MII cell polarity, e.g. through defects in γ-tubulin-based MT organizing centers, or may alternatively rely on changes in the molecular regulation of MII meiocyte polarity, e.g. for example through a decreased AtPS1 or JASON functionality [237]. The occurrence of fused, parallel and/or tripolar spindles in male MII has already been described in several plant species, including *Solanum* [241], *Populus* [87,92], *Medicago sativa*, *Impatiens* [242], *Agave* [99], *Lotus tenuis* [243] and *Ipomoea batatas* [86], potentially reflecting a mild form of heat-induced meiotic non-reduction. Moreover, similar to in rose, high temperatures have also been reported to enhance male 2n gamete formation in other species, including *Lotus tenuis* [243], diploid chilli (*Capsicum annuum* L. 'Xianjiao'; 35.5 °C, 4 h) [244] and wheat species [201], suggesting that high temperatures or heat shocks have a general potential to induce sexual polyploidization and WGD, most likely via ps-mediated male meiotic restitution. Interestingly, recent studies in poplar (*Populus* L.) have revealed that heat stress may also affect the reductional character of female meiotic cell division, yielding unreduced megaspores capable of inducing sexual polyploidization [245,246]. Moreover, depending on the timing of the heat treatment (during MI or MII), female sporogenesis either produces FDR or SDR 2n gametes [245]. In all flowering plants, female meiosis exhibits a successive-type of cytokinesis and hence does not depend on specific spindle orientations during MII. Thus, in this case, heat-induced meiotic restitution is not based on MII spindle irregularities, but rather on alterations in cell cycle regulation or cell wall formation. Alternatively, heat-induced 2n gamete formation may result from defects in synapsis and CO and associated failures in bivalent formation, inducing meiotic non-reduction in a similar way as in amphihaploid and asynaptic meiocytes. In support of this, both Wang et al. [245] and Lu et al. [246] found that the MI pachytene to diplotene stages comprise the most optimal period for heat-induced restitution of MI in poplar. Additionally, studies in *Allium ursinum* have revealed that high temperatures affect the biogenesis and stability of the synaptonemal complex in early stage prophase I [247], precluding recombination and CO and reducing the number of interhomologous recombination events [248]. A similar reduction in chiasma frequency has been reported in heat-stressed grasshoppers, however, in this case, univalents and the associated induction of meiotic restitution has never been observed [249]. Likewise, mild heat stresses (from 22 to 30 °C) in barley were found to induce spatiotemporal alterations in meiotic axis formation and recombinational protein loading, eventually causing a small but significant reduction in mean chiasma frequency, although without induction of meiotic restitution [250]. These observations all together suggest that heat-induced meiotic non-reduction through asynapsis only occurs under specific conditions, namely in a temperature range that causes complete loss of chiasmata without impairing meiocyte viability.

Similar to heat stress, short periods of cold also increase the gametophytic ploidy level, however, the underlying mechanism appears completely different. A recent study in *A. thaliana* revealed that short periods of cold (1–40 h at 4–5 °C) disrupt the final step of meiotic cell division, i.e. post-meiotic cytokinesis and cell wall formation, eventually yielding dyads, triads and monads that contain syncytial microspores [96]. Moreover, since syncytial nuclei fuse before pollen mitosis I and then show a normal progression through microsporogenesis, this process generates diploid or polyploid pollen, capable of conferring sexual polyploidization. Cytological examination of *Arabidopsis* PMCs revealed that cold stress does not affect meiotic chromosome behavior, as has repeatedly been observed in animal meiosis [251], but instead specifically disrupts the biogenesis of the internuclear radial microtubule arrays (RMAs) at telophase II, which normally function as phragmoplast-like structures that mediate post-meiotic cell wall formation [252]. Accordingly, subcellular localization of organelles and subsequent deposition of callose at developing MII cell plates is impaired, resulting in a partial or complete loss of meiotic cell plate formation. Formation of diploid and polyploid pollen upon exposure to low temperature has also been observed in several other plant species, including Japanese Persimmon (*Diospyros kaki* Thunb.) [253], *Brassica* [254] and *Dasypyrum* [255]. However, for these species the exact cellular mode of polyploid gamete formation has not yet been resolved. Molecular insights into the mechanism by which cold affects meiotic cell plate establishment may come from observations in a wheat thermosensitive genic male sterile (TMGS) line that shows alterations in MI cell plate assembly upon exposure to low temperature stress (10 °C) [256,257]. Large-scale transcriptomics hereby revealed that cold alters the expression of several key actin regulators and other genes implicated in the dynamic organization of the cytoskeleton, such as actin-depolymerization factor, profilin, formin, villin and LIM domain protein, suggesting that cold-induced defects in meiotic cytokinesis may have a transcriptional basis [257]. Moreover, since formins play a role in meiotic RMA formation [88], these proteins are thought to be one of the primary factors underlying cold sensitivity of meiotic cell plate formation [237]. Generally, in both mitotic and meiotic cells, low temperature stress has a direct negative impact on the stability of microtubules and associated cytoskeletal figures [258,259]. In budding yeast meiosis, this is accompanied by an arrest of cell division and an associated down-regulation of genes required for cell cycle progression, meiotic differentiation and development [260]. Based on this, it is thought that meiotic RMA and phragmoplast structures in plants are structurally more sensitive to cold and that their disintegration causes alterations in meiotic cell cycle progression, including defects in cell plate formation and polynuclear spore formation. Despite the absence of clear underlying regulatory mechanisms, the impact of adverse temperatures on male sporogenesis and the associated induction of sexual polyploidization through 2n gametes constitutes an elegant mechanism to increase genomic flexibility (e.g. polyploidy) as an adaptive mechanism to cope with adverse conditions [96,240]. Whether this process is actively regulated or forms an indirect consequence of structural defects in meiotic cell division still remains elusive and forms an important subject of future studies.

2.4.2. Stress-induced meiotic restitution during plant evolution?

Obviously, the repeated observation of stress-induced formation of 2n gametes in plants triggers the question of whether this process could have driven WGD during plant evolution. Although the precise mechanism(s) inducing ancient polyploidization events are extremely difficult to determine, there is accumulating evidence that stress-induced formation of 2n gametes may have played an important role. Indeed, during the last decade, several phylogenetic and comparative sequence analyses have revealed that ancient WGD events in plants appear clustered in time, and often coincide with periods of dramatic climate changes and catastrophic extinction events. Fawcett et al. [11], for example, reported that the majority of recent genome duplication events, occurring independently in the major plant lineages, coincide with the Cretaceous-Tertiary (KT) boundary (±65 million years ago) [13], representing the most recent mass extinction event caused by extreme global climate changes resulting from volcanic activity

and asteroid impact [261–263]. Similarly, other WGD events during plant and vertebrate evolution have been dated to periods of abiotic stress, including glaciation, global warming and atmosphere cooling [16,264,265]. Although this stress-related 'evolutionary boost' of WGD events may simply reflect a selective advantage of the 'more adaptive' polyploid lineages [9,266–269] to adverse environments [11,270–272], these observations may alternatively or concomitantly indicate an increased incidence of polyploid origin, e.g. through increased levels of sexual polyploidization. In support of this, recent paleological analysis of *Classopollis* conifer pollen extracted from sediments of the Triassic-Jurassic transition (200 Mya) revealed the presence of aberrant tetrads, dyads, triads and larger pollen grains, indicative of meiotic restitution and 2n pollen formation during the End-Triassic biotic crisis [273]. In a similar way, aberrations in lycophyte spores and conifer pollen have been found to occur during the end-Permian mass extinction event [274,275], indicating that ancient global climate changes are associated with irregularities in reproductive ploidy stability and enhanced induction of sexual polyploidization.

Further evidence supporting a role for stress-induced 2n gamete formation in plant evolutionary WGD events comes from the observation that polyploid lineages are more frequent in habitats affected by climatic and edaphic fluctuations, such as high altitudes and recurrently glaciated areas [189,276]. Although this may again reflect a higher adaptability of polyploid genomes to adverse environments or ecological niches [277], the additional finding that these polyploids have recurrently formed at different scales in time and space [278] and still show evidence of ongoing polyploidization suggests that polyploidization occurs more frequently under conditions of (a)biotic stress. Indeed, both for allo- and autopolyploids, biogeographic and ecological assays point to an association between polyploid induction and environmental change [279]. All together, these findings support the notion that stress-induced meiotic non-reduction in plants may constitute an important driver of evolutionary whole genome duplications and associated events of speciation.

3. Somatic polyploidization

3.1. Natural pathways for somatic polyploidy in plants

Whole genome doubling in plants does not only rely on alterations of the meiotic cell cycle, but may also be conferred by somatic ploidy instability [54,181]. Indeed, the ectopic induction of polyploidy in mitotically dividing somatic or reproductive tissues forms an alternative mechanism for inducing WGD in plants. However, whether such ectopic events of ploidy increase actually lead to a stable transgenerational induction of whole genome duplication largely depends on the mode of reproduction and the affected tissue type. In plants with a vegetative mode of reproduction, ectopic genome duplication in tissues required for asexual propagation (stolons, bulbs, rhizomes) may lead to the establishment of stable polyploid lineages. In contrast, in plants that reproduce sexually, only somatic polyploidization events in the L2 layer [280], that gives rise to the reproductive tissues, and in (pre-)meiotic or gametophytic cells, such as micro- and megaspores [281], lead to a transgenerational fixation of polyploidy. Ectopic polyploidization events in other tissue types do provide a temporary increase in ploidy, but are not maintained in the next generation, and as such cannot form a basis for evolutionary WGD events.

In theory, somatic polyploidization in plants can originate from two different mechanisms; endoreduplication and endomitosis. Although both mechanisms confer duplication of genomic DNA content, endoreplication basically involves an terminal switch from the mitotic G1-S-G2-M cell cycle to repeated cycles of S- and G-phases, generating chromosomes with multiple sister chromatids (e.g. polytenal), whereas endomitosis is caused by a specific loss of the M phase, yielding cells with a duplicated number of chromosomes [247,282]. Endoreduplication is rather rare in animals but common in plants, and is thought to provide a cellular mechanism for rapid increase of metabolic activity and cell size in specific organ types [283–285]. However, despite its biological relevance, endoreduplication has only in a few cases been reported in reproductive tissues [286]. Moreover, since this only occurred in artificially generated lines (e.g. AP3::FZR OE) and eventually led to an arrest of subsequent embryo development [287], ectopic induction of endoreduplication is most likely not a common mechanism for transgenerational induction of WGD. In addition, ectopic genome doubling through endoreduplication has often been associated with terminal differentiation [284] and cell division arrest and, in more extreme cases, with tumorigenesis and defects in ploidy stability [282], possibly indicating that it constitutes an evolutionary dead end. In contrast, somatic polyploidy through endomitosis does not represent a common plant biological process [288], except for those events occurring in anther tapetum development [289,290], but instead mostly occurs as an ectopic aberrant cell division under special conditions [291], eventually yielding cells with a doubled set of chromosomes. Since these cells behave as real tetraploid or polyploid cells, showing normal mitotic cell division and balanced chromosome segregation [292], endomitosis does not cause developmental aberrations, hence forming a putative basis for whole genome duplication. In support of this, several studies have reported the occurrence of ectopic endomitosis (e.g. by antimitotic drugs) as a basis for polyploid tissue induction [293,294] and, occasionally, for polyploid progeny formation [295]. In the latter cases, ectopic occurrence of endomitosis specifically occurred in pre-meiotic, meiotic or gametophytic cells, hence yielding diploid and/or polyploid gametes that were capable of performing sexual polyploidization. Based on their mode of formation, diploid gametes resulting from somatic polyploidization events are termed 2x gametes, contrasting with the 2n (unreduced) gametes formed by meiotic restitution [71].

3.2. Cellular mechanisms causing (pre-)meiotic endomitosis and gametophytic ploidy increase

Generally, ectopic induction of endomitosis occurs through alterations in the final steps of mitotic cell division, e.g. in chromosome segregation or in post-mitotic cell wall formation. Indeed, all cellular alterations that impair bipolar chromosome separation after metaphase I or that affect the biogenesis of the internuclear cell wall at the end of mitosis may cause 'mitotic restitution', eventually leading to a duplication of the cell's chromosome number (Fig. 2). Bipolar chromosome segregation during anaphase I largely depends on three factors: (1) intercellular organization of the spindle origins to opposite sides of the cytoplasm; (2) amphitelic attachment of the spindle microtubules to the centromeres through kinetochore functioning [296] and (3) progressive movement of sister chromatids to the spindle poles. Cytological studies in several species have demonstrated that irregularities in one of these processes, such as defects in spindle body duplication in yeast and loss of essential kinetochore components [297,298], causes a complete failure of mitotic karyodivision, yielding polyploid endomitotic cells [299]. In plants, similar defects have been found to induce chromosomal instabilities, including endomitotic polyploidization. However, reported cases have only been observed under artificial conditions (genetic knock-outs and overexpression) and rarely lead to polyploid progeny [134,300].

Subsequent to mitotic chromosome segregation, cytokinesis consolidates the ploidy stability of the cell lineage by generating an internuclear cell wall. In plants, the building of the cell wall

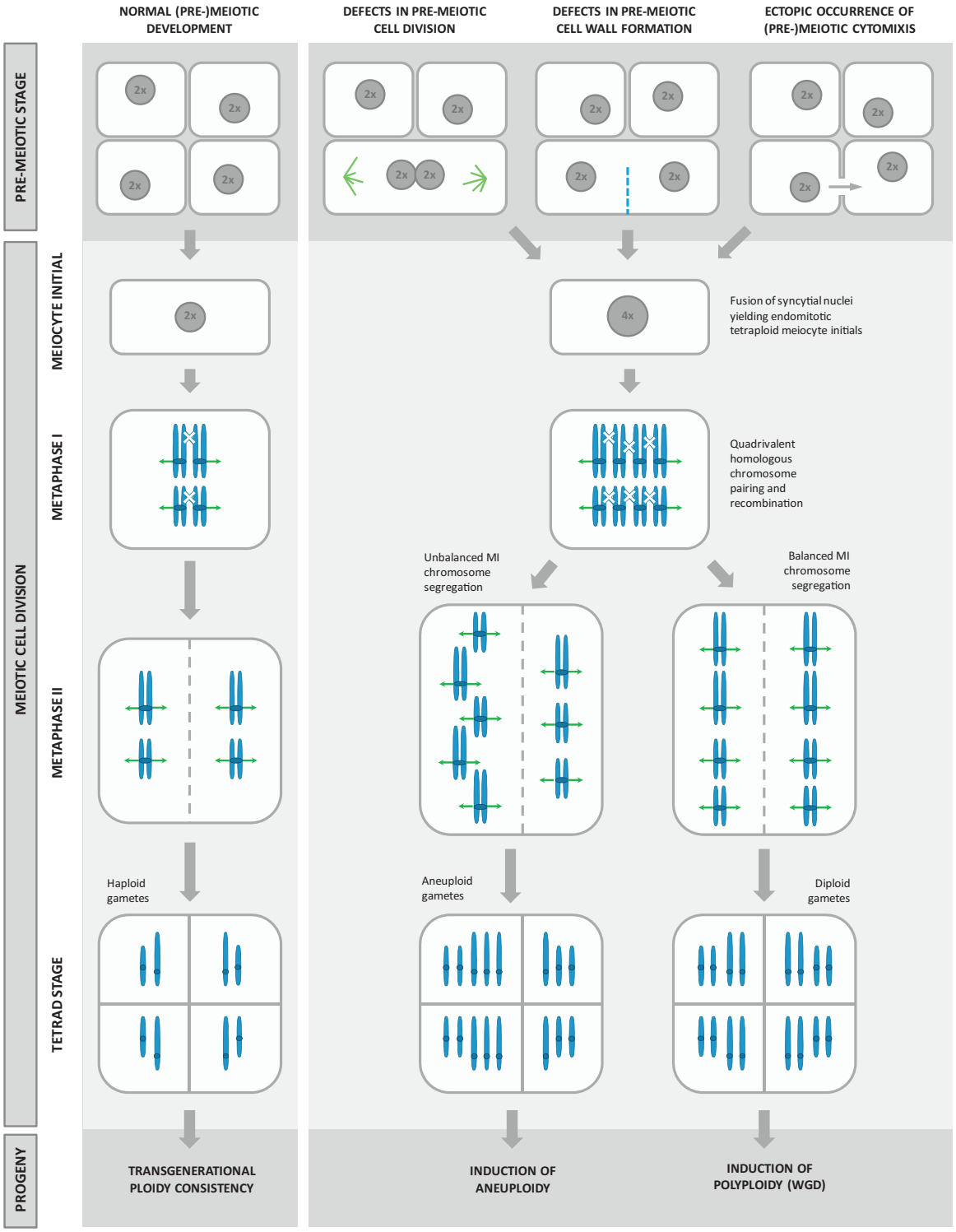

Fig. 2. Mechanisms and outcomes of pre-meiotic endomitosis.

involves many tightly regulated subprocesses, including phragmoplast formation, fusion of Golgi-derived vesicles, callose deposition, etc. [301,302], and alterations in each of these processes have been found to generate syncytial nuclei, that eventually fuse to form polyploid endomitotic cells [303–306]. Studies using *Arabidopsis* mutants have revealed that strong and prolonged defects in somatic cell plate formation typically lead to lethality (apoptosis), most likely caused by developmental irregularities and progressive genomic instabilities associated with uncontrolled polyploidization. In contrast, when defects in somatic cell wall formation are rather mild and only occur occasionally, associated polyploidization events can be tolerated, eventually yielding chimaeric mixoploid plants [304,306]. Moreover, if such mild defects in cell wall formation occur in reproductive tissues, such as archesporal pre-meiotic or early stage meiotic cells, associated polyploidization events forms a basis for 2x gamete formation and sexual

polyploidization. Indeed, cytological studies in *A. thaliana* and tomato revealed that cytokinetic defects in meiocyte archesporal cells result in tetraploid and sometimes polyploid meiocyte initials that, upon progression through meiosis I and II, yield functional diploid and polyploid gametes, capable of generating polyploid progeny [306,307]. However, in line with the autopolyploid nature of resulting meiocytes, MI often shows multivalent pairing and unbalanced chromosome segregation, typically resulting in a mix of polyploid and aneuploid gametes. As such, ectopic induction of pre-meiotic endomitosis through defects in cytokinesis may form a basis for euploid WGD events, but also for other, rather minor or additional changes in chromosome stability, such as aneuploidy, nullisomy and polysomy [304]. As cytokinesis is a highly regulated process, requiring an intricate interplay between several cellular processes, including cytoskeletal dynamics, cell cycle regulation, vesicle trafficking, lipid metabolism and signalling pathways, a multitude of genetic disorders have been described that affect somatic cell wall formation and associated ploidy stability [308]. However, most of these genetic defects (e.g. *knolle*, *keule*, *hinkel*, runkel, pilz, etc.) cause early stage lethality [309], whereas only a few result in polyploid meiocytes and sexual polyploidization. For example, *Arabidopsis* loss-of-function mutants for SMT2, a sterol-methyl transferase implicated in structural sterol synthesis, exhibit non-lethal defects in somatic cell wall formation and thereby ectopically form tetraploid meiocytes and diploid gametes in both male and female sporogenesis [306]. A similar phenotype has been observed in the specific *et2* allele of GSL8, i.e. a callose synthase required for somatic cell wall establishment, whereas other *gsl8* alleles cause seedling lethality. These findings taken together indicate that specific defects in different processes implicated in wall establishment may form a cellular basis for pre-meiotic endomitosis and associated induction of sexual polyploidization.

Ectopic induction of (pre-)meiotic endomitosis and associated formation of 4x meiocytes and 2x gametes may not only result from defects in mitotic cell division, but can also originate from cellular defects leading to migration of chromosomes from one cell to the neighboring one [310]. This process is generally termed cytomixis and refers to the intercellular transfer of chromatin, single chromosomes or whole chromosome sets (nuclear migration) through cytoplasmic connections or via direct cell fusion. When occurring in meiotic or pre-meiotic cells, cytomixis often leads to erratic meiosis, characterized by defects in chromosome organization and segregation [311–315]. However, occasionally, cytomixis may also result in the formation of stable poly- or aneuploid meiocytes capable of generating functional gametes with an increased chromosome number, hence forming a putative basis for WGD and karyotype change [310,316,317]. In *Chrysanthemum zawadskii* and *C. indicum*, for example, Kim et al. [318] demonstrated that fusion of two adjacent PMCs occasionally occurs early in meiosis I, generating tetraploid meiocytes that proceed through meiosis and that consequently yield diploid pollen. Cytomixis has been described in many species [310,312,316,317,319–321] and is considered a widespread naturally occurring phenomenon, characteristic of both vegetative and generative tissues. Interestingly, cytomixis is most frequently detected in meiotic cells, especially in microsporocytes and never in megasporocytes, and thereby predominantly occurs in meiotic prophase I [313,317,319,320], indicating that the early PMC stage structurally or functionally facilitates intercellular chromatin movement. Preliminary studies in several plants, including *Medicago*, *Chlorophytum* and Himalayan poppy suggest that that process of cytomixis is under genetic control and that in the case of meiotic cells this most likely involves meiosis-specific genes and associated signal transduction pathways [312,314]. However, the exact underlying molecular mechanisms and genetic control systems of cytomixis have not yet been elucidated [322].

3.3. Endomitosis and evolutionary speciation

From the perspective of evolutionary significance, several lines of evidence suggest that (pre-)meiotic endomitosis, either through mitotic or cytokinetic defects or cytomixis, may have contributed to evolutionary speciation, either by inducing WGD events or by conferring other changes in chromosomal stability. Firstly, the ectopic formation of tetraploid meiocytes and the associated production of 2n gametes through (pre-)meiotic genome doubling has been observed to occur spontaneously in several crops and natural plant species, including *Aegilops*, Himalayan poppy, *Dactylis*, *Phleum Pratense*, *Medicago*, *Festuca*, *Avena* and rye [310,312,323–328], indicating that it is a naturally occurring phenomenon, capable of conferring sexual polyploidization. Secondly, in some plant species, (pre-)meiotic endomitosis and polyploid meiocyte formation appears strongly correlated with adverse climatic conditions [304,329,330], indicating that somatic ploidy change may act as a stress-induced mechanism, conferring adaptive chromosomal change or polyploidization to cope with (a)biotic stress environments. For example, in interspecific sorghum hybrids, syncytial microsporocytes were only observed under conditions of high temperature and moisture stress and not under more optimal growing conditions [331]. Similarly, in *Lindelofia longiflora* (Royle ex Benth.) (Family: Boraginaceae), sporadic events of PMC fusion and early PMC syncyte formation were only observed upon exposure to low temperatures [332]. Also, in *Salvia miltiorrhiza* PMCs, cytomictic chromosome migration is more frequent under high temperature conditions compared to under control conditions [320]. Interestingly, studies in *Arabidopsis* revealed that the ectopic induction of endomitotic polyploidy through defects in cytokinesis predominantly occurs in flower organs, and only rarely in vegetative tissues, suggesting that the ploidy stability of reproductive tissues is extremely sensitive to defects in cell plate and cell wall formation [306]. Abiotic stress-induced alterations in plant cytokinesis and cell wall establishment may therefore constitute a cellular pathway for the ectopic induction of WGD in reproductive tissues, thereby representing an alternative mechanism of stress-induced 2n gamete formation and sexual polyploidization. Thirdly, (pre-)meiotic endomitosis and particularly cytomixis have been found to occur more frequently in polyploid lineages and genetically unbalanced plants, such as haploids, aneuploids and hybrids [321], indicating that these phenomena may have driven WGD and other changes in ploidy and chromosome configuration during plant evolution. Conversely, Sidorchuk et al. [319] found that a duplication of the chromosome number increases the frequency of meiotic cytomixis, suggesting that polyploidy enhances cytomixis and not vice versa. However, despite this discrepancy, there is ample evidence suggesting that cellular mechanism inducing somatic endomitosis may have contributed to evolutionary WGD events or other changes in genomic speciation.

4. Karyotype change: aneuploidy, dysploidy and chromosome rearrangements

4.1. Definitions and overview

Aneuploidy refers to the loss or gain of whole chromosomes, or in a broader sense parts of chromosomes, relative to an established karyotype [333]. Dysploidy on the other hand involves structural changes in the genome that do not result in the loss or gain of genetic information, but that alter the gross chromosome number via chromosome rearrangements [334]. Other chromosome rearrangements such as translocations, inversions, chromosome fusions and breakages may not result in changes in chromosome number and hence dysploidy, but may nevertheless play a role

in speciation events. Chromosome rearrangements are often tolerated where aneuploidy is not: monosomics and nullisomics are often lethal in non-polyploid lineages. Somatic aneuploidy is rarely detected in established plant species, although this may be due to the decreased viability of aneuploid chromosome complements rather than the absence of aneuploid gametes [335,336]. However, aneuploid progeny does commonly result from de novo allopolyploids [333] or triploids [77,337]. Major karyotypic changes such as dys- and aneuploidy can result from alterations in somatic cell division, but in general result from irregularities in meiotic cell division. Meiotic mechanisms inducing both aneuploidy and dysploidy in plants include non-homologous recombination, asynapsis, loss of recombination and chromosome segregation defects [71,162,338], similar to as described in human aneuploidy [339]. Interestingly, the same mechanisms that give rise to unreduced gametes, e.g. through incorrect spindle fibre alignment, asynapsis or defects in recombination, can also result in the exclusion of one or more chromosomes from the resulting nuclei, yielding aneuploid gametes. "Micronuclei" observable post-meiosis are attributable to such excluded chromosomes, either comprising univalents, acentric fragments resulting from non-homologous recombination events or inversion heterozygotes, or "laggard" chromosomes.

4.2. Karyotype change primarily results from non-homologous recombination events

Generally, changes in karyotype configuration primarily result from non-homologous recombination events: interpretations of chromosome fusions, fissions, translocations and inversions are all readily explicable through this single, experimentally validated mechanism [334]. During meiosis, recognition of homologous chromosomes occurs based on DNA sequence similarity, although with elimination of repetitive sequences [156]. Although this is not a new concept, exactly how DNA sequence similarity dictates homolog recognition and the exact cellular mechanisms underlying this recognition process are still unknown [340]. In hybrid genotypes or allopolyploidy genomes, the process of homolog recognition often suffers from the presence of homeologous sequences, e.g. resulting from previous whole genome duplication events and/or sequence diversification. Stretches of ancestrally-related chromosomes (homeologs) or chromosome fragments are often similar enough to initiate pairing and recombination, physically exchanging dissimilar parental sequences or chromosome parts by establishing physical sites of crossing-over (e.g. chiasmata) [341]. Depending on the pre-existing degree of fractionation between ancestral subgenomes (how rearranged homeologous chromosomes are relative to each other) crossover events may thereby form the basis for major or minor chromosome rearrangements.

Different genetic and genomic factors influence the odds of non-homologous recombination and hence putative chromosome rearrangement events occurring. Autopolyploids and allopolyploids, newly formed between closely related species, usually have the highest degree of sequence similarity between hom(e)ologs and hence display a relatively low degree of genome differentiation due to chromosome rearrangements. Indeed, individuals or species with high homeology between chromosomes or subgenomes are predicted to show a greater degree of non-homologous chromosome association than individuals or species with more distantly related subgenomes or chromosomes (see Section 2.3.1). This has been experimentally verified in *Brassica* haploids and hybrids [342,343], confirming the notion that both genome structure [344,345] and presence of additional unpaired chromosomes [346] also affect non-homologous recombination and genome rearrangement.

Genetic control of non-homologous chromosome pairing is also a major factor affecting the odds and rates of chromosomal rearrangements. In allopolyploid bread wheat, the *Ph1* locus effectively prevents non-homologous pairing interactions between closely related chromosomes from the A, B and D genomes [221]. Recent studies have found that *Ph1* comprises a cluster of cyclin-dependent kinase (*Cdk*-like) genes [221,222], and most likely regulates meiotic chromosome dynamics, e.g. homoeolog recognition and pairing, by suppressing *Cdk2*-type activity through the production of defective *Cdk2*-like gene products [223,226]. More specifically, *Ph1* is thought to regulate homolog pairing and synapsis by coordinating chromatin remodelling on both homologues chromosomes [196,200]. In support of this, in allopolyploids, lack of synchronization between homoeologous chromosomes for chromatin conformational changes is thought to reduce the chance of pairing between homeologues [341].

Other genetic loci affecting the frequency of non-homologous chromosome pairing have also been identified in *Brassica* [347–349] and recently in *Arabidopsis* polyploids [231]. However, these genetic loci have a quantitative rather than qualitative effect on non-homologous pairing, and appear to operate differently to *Ph1*. For example, the pairing locus *PrBn* in *B. napus* was found to affect frequency but not distribution of crossover events at meiosis [349], which suggests a different regulation of non-homologous pairing to *Ph1*.

In genera and families with stronger genetic control of non-homologous chromosome pairing, chromosomal rearrangement events may be rarer than in families which are more permissive of non-homologous chromosome associations at meiosis. However, genetic control of non-homologous chromosome pairing is still not well understood. In plants, the only allopolyploid species for which this has been characterized at a molecular level is bread wheat (*Triticum aestivum*) [341], although elucidation of these mechanisms in *B. napus* is ongoing [350]. In the future and with the advent of whole genome sequencing approaches to phylogenetics and ancestral karyotype reconstruction, the relationship between permissiveness of non-homologous recombination and phylogenetic relationships within genera may be elucidated.

4.3. Molecular and cytological mechanisms for aneuploidy

Laggard chromosomes are common in hybrids and new allopolyploids, and may result from differences in progression through the meiotic or mitotic cell cycle between chromosomes belonging to each of the parental subgenomes [351]. In some wide hybrids, such as wheat (*Triticum aestivum*) × pearl millet (*Pennisetum glaucum*) [352], wheat x maize crosses [353] or *Hordeum vulgare* × *H. Bulbosum* [354], chromosome elimination by laggard exclusion occurs rapidly during meiosis or early mitotic divisions, eventually eliminating one complete parental chromosome set. This was hypothesized to occur due to unequal interaction of centromeres from each parent with the mitotic spindle, and the molecular basis of this effect has since been attributed to the action of centromeric histone H3 (CenH3) [161,355]. Loss of CenH3 proteins has since been found to cause centromere inactivation and subsequent chromosome loss, and uniparental genome elimination in newly formed hybrids attributed to cross-species differences in centromeric CenH3 incorporation [356]. In interspecific *Hordeum bulbosum* x *H. vulgare* hybrid zygotes, CenH3 is gradually lost and not replenished in *H. bulbosum* chromosomes over successive mitoses after zygote formation [356], an effect hypothesized to be related to poor synchronization of *H. bulbusom* chromosome replication with the cell cycle [351]. Interestingly, timing of chromatin condensation during chromosome replication is also thought to be the primary mechanism preventing non-homologous chromosome pairing in allopolyploids [226,341], as mentioned previously.

Hence, timing of chromosome replication in each subgenome may constitute the primary means by which chromosome segregation and interactions are regulated in allopolyploids to ensure meiotic stability. CenH3-mediated chromosome loss is also temperature-dependent [351], supporting a common theme of meiotic and mitotic instability in response to stress, potentially providing a source of novel variation for stress escape.

4.4. Somatic aneuploidy

Somatic aneuploidy has rarely been assessed in plants, but may occur naturally in many established species and sometimes in particular tissue types, particularly in polyploid plants. Somatic aneuploidy has been detected in *Arabidopsis suecica* [357], a natural allotetraploid, in potato-tomato hybrids [358] and also in several other crop plant species [359]. Aneuploidy is also commonly induced by tissue culture (reviewed by Damato [360]), and by a number of known chemical and environmental mutagens (reviewed by Sharma [359]). In plants, somatic aneuploidy may occur in undifferentiated tissues that then form generative organs, subsequently resulting in meiotic production of aneuploid progeny. Somatic aneuploidy may also be tolerated at high levels in plants that can reproduce clonally, and chimeric aneuploid sectors may contribute to formation of new plants through vegetative propagation (e.g. tillers or rhizomes).

4.5. The relationship between chromosome rearrangements and speciation

From an evolutionary or phylogenic perspective, most speciation events appear to involve dysploidy, as these forms of genome change differentiate extant species. Comparative karyotypes between mammals demonstrate this concept readily: overall genome conservation is high across species, but with chromosome fusions, fissions and rearrangements differentiating species karyotypes [361]. Modern molecular, genomic and cytogenetic tools are allowing greater elucidation of historical karyotype rearrangements than ever before. In *Arabidopsis* [362], *Cucumis* [363], *Brassica* [364], *Sinapis* [365], maize, rice, sorghum and *Brachypodium* [366], among others, ancestral karyotypes have been elucidated and divergence between extant species revealed to be the result of chromosomal rearrangements.

However, differences between species as a result of karyotype variation offer purely a retrospective viewpoint: what evidence is there for speciation events occurring as a direct result of chromosomal reshuffling, rather than as a function of species divergence over time? Two different models have been proposed for the role of chromosome rearrangements in speciation events: e.g. the “hybrid sterility” and “suppressed recombination” model [367]. In the “hybrid sterility” model, chromosome rearrangements aid in the reproductive isolation of overlapping populations through reduced fertility in individuals heterozygous for these chromosome rearrangements [367,368]. In the “suppressed recombination” model, suppression of recombination over inversion regions may favor accumulation of locally-adapted alleles within this region, aiding in genetic differentiation of geographically-overlapping populations [368,369]. Chromosome rearrangements distinguishing the karyotypes of two closely related taxa that subsequently hybridize (homoploid hybridization) may also lead directly to genetic isolation of the hybrids from their parent species if backcrosses are subsequently sterile [370]. The role of chromosome inversions in speciation via reproductive isolation has been studied in fruitfly, mosquitoes and butterflies [371] and in models for speciation [372], but is rarely put forward as a primary driving force for speciation in most genera [361]. Meiotic mechanisms such as reduction of recombination between diverged homologous chromosomes have occasionally been investigated in relation to speciation [373], but overall far less experimental investigation has taken place into the role of chromosome changes in speciation in plants compared to in animals [66,374]. Recent research in *Helianthus* has provided some support for speciation via chromosomal rearrangements and recombination suppression, with genomic regions associated with particular chromosome rearrangements between species also associated with restricted gene flow between these species [375], thereby somewhat contradicting previous research [376]. However, chromosome rearrangements have also been more directly linked to speciation via reproductive isolation in this genus [377]. Dysploidy has also recently been proposed as the most probable cause of speciation in the genus *Carex* [378] although evidence against this citing lack of allelic differentiation between karyotypically diverse *Carex* lines has also been obtained [379]. As such, further research is required to determine the exact role of karyotype change, and in particular the genetic effects of chromosome rearrangements in facilitating speciation events [67].

In the angiosperms, which are now known to have several basal polyploidy events [21], genome redundancy provided by historic polyploidization events can allow for much greater karyotypic variation within species without subsequent loss of fertility [9], as observed in (for example) translocation heterozygotes in allopolyploid *Brassica napus* [380]. If fertility of a translocation heterozygote, i.e. the F_1 between two genotypes differing by a chromosomal translocation, is not significantly reduced, these changes may rarely result in reproductive isolation, at least in polyploid plants. Chromosome rearrangements are the least disruptive to the genome and to absolute gene dosage of all ploidy-related mechanisms for speciation, and differentiate the karyotypes of most extant species. Hence, it is possible that chromosome change plays a cryptic or accessory role in the majority of speciation events.

4.6. Aneuploidy and speciation

Aneuploidy has often been dismissed as a potential factor in speciation and karyotype evolution [381], due to the destabilizing effect on gene expression caused by duplication or deletion of some chromosomal regions of the genome but not others. This may result in altered dosages for genes involved in the same pathways or networks, to potentially detrimental effect [382]. However, aneuploidy may also confer beneficial effects: improved growth and proliferation is often a consequence of aneuploidy in tumors [383] and yeast [384], even if examples in more complex taxa are sparse. Aneuploidy is also frequently observed in nature, generally in plants with polyploid genomes or lineages. Examples include *Tragopogon* [188], *Rutidosis* grasses [385], *Malus* [386] and in the complex *Hieracium* and *Pilosella* lineages [387]. Recently, evidence for complex hybridization and accompanying aneuploidy leading to a new, established species was also obtained in the *Cardamine* genus (Asteraceae) [388]. Aneuploidy can also be an intermediate stage in the establishment of novel euploid karyotypes: *Arabidopsis* triploids give rise to aneuploid progeny that stabilize at either the diploid or tetraploid level after a few generations [77]. Moreover, research in *Malus* also suggests that in some cases aneuploid gametes or cytotypes may have an advantage over euploid gametes or cytotypes, contributing to increased heterozygosity and genetic variation [386]. The higher genome redundancy offered by polyploid genomes may allow greater tolerance of chromosome loss compared to diploid genomes. The prevalence of aneuploidy in interspecific hybrids and polyploids also suggests that this phenomenon may occasionally contribute to the establishment of new karyotypes, as suggested by the few examples of speciation via aneuploidy so far obtained in plants.

4.7. Cryptic aneuploidy in allopolyploids: homoeologous chromosome substitutions

A form of cryptic aneuploidy in new allopolyploids has recently been discovered. This form of aneuploidy, which has been observed in young allopolyploid *Tragopogon miscellus* [188,389] and in resynthesized *Brassica napus* [152] involves loss and gain of closely related homeologs, but with retention of overall homeolog dosage balance. As an example of this dosage compensation effect, for closely related whole-chromosome homeologs A1 and B1, a given plant may retain the expected A1-A1 and B1-B1 homologous chromosomes, or instead have a chromosome complement of A1-A1-A1-A1 (nullisomy–tetrasomy) or A1-B1-B1-B1 (monosomy–trisomy); overall chromosome number and "dosage" of homeologs is retained despite cryptic aneuploidy. The mechanistic basis for this effect is probably related to poor genetic regulation of homeolog recognition, i.e. incorrect separation of homologs and homeologs by the cell machinery during meiosis [390], coupled with selection for gametes maintaining the correct copy number of homologous and homeologous chromosomes [152]. The formation of tetravalents during meiosis I and subsequent tetrasomic inheritance (distributed inheritance of 0–4 chromosomes from the tetravalent) has been documented in a number of autopolyploid species, including blueberry [391], *Lotus corniculatus* [392], *Heucheria grossulariifolia* [393], *Papsalum simplex* [394] and potato [395]. Partial or intermediate tetrasomic inheritance can also occur (occasional formation of tetravalents), particularly in hybrid species [396]. This dosage compensation mechanism, which rearranges chromosome karyotype without the loss of gene information accompanying other forms of aneuploidy, has been postulated to lead to speciation after karyotype stabilization [32].

4.8. Dispensable, "B" and sex chromosomes

In some species, specific individuals can contain one or more chromosomes that are additional to the species karyotype; that is, not every member of the species will contain these additional chromosomes. These additional chromosomes fall into several broad categories. "B" chromosomes are generally defined as chromosomes that are not present in every individual of a species, do not constitute a duplicate of an existing chromosome in the standard species karyotype ("A" chromosome set), do not recombine with other chromosomes at meiosis and are inherited in a non-Mendelian or irregular fashion (reviewed by Jones and Houben [397]). Although B chromosomes do derive from A chromosomes, the common consensus is that B chromosomes do not generally facilitate evolution or drive speciation events; and in fact are generally detrimental "parasites" on the host genome [398]. Heteromorphic sex chromosomes, generally defined as chromosomes which contain sex-determination loci that do not recombine at meiosis [399], also provide an example of a very specific form of karyotypic variation within species. Interestingly, the evolution of sex chromosomes may comprise a special case of evolution via chromosome rearrangements, whereby sex chromosomes evolve via suppression of recombination (reviewed by Charlesworth et al. [399]). Other kinds of "dispensable" chromosomes that do not fit into the above categories are common in fungi (reviewed by Covert [400]), but have yet to be identified in plants. Further investigation of these phenomena in relation to speciation and with the aid of genome resequencing technologies may lend further weight to the role of dispensable chromosomes in plant speciation.

4.9. Environmental stress can trigger ploidy change

The environment may play a major role in dictating whether and how chromosome rearrangement and even aneuploidy events can lead to speciation. Firstly, positive or negative selection for particular karyotype changes may occur as a result of environmental factors. These karyotype changes may be either novel, or pre-existing within populations: minor chromosome rearrangements within species are now known to be common as resequencing becomes increasingly accessible, with insertion/deletion (indel) and translocation variation observed between accessions or cultivars in maize [401], rice [402] and *Brassica* [380] and with other species predicted to show similar trends. Environmental selective pressure may operate on karyotypic variation within a species in the same way as on more conventional allelic variation, with a similar potential end point of speciation and reproductive isolation with sufficient divergence. Secondly, karyotype change may also be induced by environmental effects, particularly by affecting non-homologous recombination frequencies [334]. In plants, homologous recombination is known to be affected by both biotic and abiotic factors [403], including ionising and UV radiation [404], temperature and day/night duration [405]. Specific environmental effects on non-homologous recombination rates are largely unknown. However, the hypothetical conservation of mechanisms resulting in both homologous and non-homologous recombination [350] suggests that non-homologous recombination should be similarly affected by environmental conditions, particularly stress. Possibly, karyotype change may be faster under stressful environmental conditions, which would facilitate generation of novel genetic diversity and perhaps even speciation as an escape mechanism. Future studies may lend weight to this speculation.

5. Conclusions

Understanding the cytological mechanisms underlying plant speciation events is critical in building correct models for karyotype and genome evolution and informing phylogenetic analysis. Cytological mechanisms underlie and tie together the complex relationship between individual plant genomes, populations and species. As well, knowledge of the effect of environmental variables on underlying molecular mechanisms contributing to chromosome and ploidy change over generations will help us understand and predict how speciation occurs in response to environmental change. Despite increasingly widespread awareness and recognition of the role of whole-genome duplication events and hybridization as critical processes shaping the flow of plant genome evolution [406], there is still a great deal of uncertainty as to the mechanistic processes underlying these phenomena.

In this review we provide a detailed overview of what we now know about sexual and somatic polyploidization in plants, as well as recent developments in understanding the role of dysploidy and aneuploidy in speciation. We cover the three main cytological mechanisms responsible for meiotic restitution: alterations in spindle fibre dynamics, defects in meiotic cell plate formation and omission of the first or second meiotic division; then go on to discuss meiotic restitution in interspecific F1 hybrids as an evolutionary mechanism for allopolyploid speciation, and how this is affected by the degree of relationship between the parental genomes. We discuss genetic control of meiotic restitution events and speculate on how stress-induced meiotic restitution can drive sexual polyploidization under adverse conditions. Up to date information on molecular and cytological mechanisms and stress responses in promotion of somatic polyploidization, aneuploidy, dysploidy and chromosome rearrangements are discussed, and we show how these lesser-known processes may contribute to speciation.

Not only are mechanistic constraints on karyotype and genome evolution often disregarded, as put forward by Schubert and Lysak [334], but terminology such as "whole genome duplication

events" is often used to gloss over our lack of knowledge of the potentially long and complex processes involved in speciation via chromosome change, polyploidy and interspecific hybridization. Greater awareness of the types and underlying mechanisms that are responsible for these speciation "events" may aid researchers at the cutting edge of genomics and phylogenetics to entertain more complex hypotheses and identify genomic clues relating to the evolutionary history of extant species. For example, chromosomal rearrangements and fragment loss commonly co-occur with meiotic restitution mechanisms in hybridization events between closely-related species. Hence, cryptic allopolyploidy may be the cause of any "bursts" of chromosome rearrangements observed in genomes over evolutionary timescales. Divergence times and subsequent allopolyploidization events between species may also be assessed in light of knowledge related to chance of genomic rearrangements vs. ease of meiotic control in allopolyploids with greater divergence between subgenomes [407]. Greater attention may also be paid to the potential role hybridization without accompanying genome doubling and of chromosome rearrangements in providing functionally adaptive variation and facilitating speciation via specialization to different ecological niches within a population [69,368].

Meiotic restitution, aneuploidy, chromosome rearrangements and somatic doubling all constitute cellular mechanisms that may originate ploidy change, particularly in response to stress. These mechanisms may all be co-opted as a means for adaptive speciation in response to changing environments, either by providing novel combinations of parent genetic variation as a resource for further environmental selection or by producing transgressive phenotypes through generation of novel genetic, genomic and epigenetic variation. Interestingly, a great deal of accumulated evidence is now suggesting that the meiotic and mitotic cell cycles are indeed highly vulnerable to environmental factors, as we discussed previously. Hence, these mechanisms may facilitate diversification and even allow "emergency speciation" in response to environmental stresses. Further research linking molecular mechanisms to cytological behavior to genome evolution and speciation at the population and environmental level will be needed to fully elucidate the complex interactions leading to these proposed effects.

With the advent of high-throughput molecular genetics and recent advances in DNA sequencing technologies, the time is ripe for experimental investigations into the cellular and molecular origin of plant speciation events [408]. Genomic signatures of cryptic hybridization or genomic introgression events [409], complex evolutionary histories of extant species [388] and the underlying bases for meiotic stabilization in novel polyploid species [31,410] may all become amenable to interrogation. Coupled with recent advances in our understanding of the molecular and cytological mechanisms underlying ploidy change, and the effects of the environment on these important speciation processes, the future holds promise for a deep understanding of how speciation processes shaped the world around us.

References

[1] B. Bremer, K. Bremer, M.W. Chase, M.F. Fay, J.L. Reveal, D.E. Soltis, P.S. Soltis, P.F. Stevens, A.A. Anderberg, M.J. Moore, R.G. Olmstead, P.J. Rudall, K.J. Sytsma, D.C. Tank, K. Wurdack, J.Q.Y. Xiang, S. Zmarzty, A.P. Grp, An update of the Angiosperm Phylogeny Group classification for the orders and families of flowering plants: APG III, Bot. J. Linn. Soc. 161 (2009) 105–121.

[2] R.M. Bateman, P.R. Crane, W.A. DiMichele, P.R. Kenrick, N.P. Rowe, T. Speck, W.E. Stein, Early evolution of land plants: phylogeny, physiology, and ecology of the primary terrestrial radiation, Annu. Rev. Ecol. Syst. 29 (1998) 263–292.

[3] L.E. Graham, M.E. Cook, J.S. Busse, The origin of plants: body plan changes contributing to a major evolutionary radiation, Proc. Natl. Acad. Sci. U. S. A. 97 (2000) 4535–4540.

[4] P.S. Soltis, D.E. Soltis, The origin and diversification of angiosperms, Am. J. Bot. 91 (2004) 1614–1626.

[5] A.A. Agrawal, M. Fishbein, R. Halitschke, A.P. Hastings, D.L. Rabosky, S. Rasmann, Evidence for adaptive radiation from a phylogenetic study of plant defenses, Proc. Natl. Acad. Sci. U. S. A. 106 (2009) 18067–18072.

[6] M.D. Bennett, I.J. Leitch, Angiosperm DNA C-values database, release 4.0, 2003, http://www.rbgkew.org.uk/cval/homepage.html

[7] E.A. Kellogg, J.L. Bennetzen, The evolution of nuclear genome structure in seed plants, Am. J. Bot. 91 (2004) 1709–1725.

[8] M.D. Bennett, I.J. Leitch, Nuclear DNA amounts in angiosperms: targets, trends and tomorrow, Ann. Bot. 107 (2011) 467–590.

[9] A.R. Leitch, I.J. Leitch, Genomic plasticity and the diversity of polyploid plants, Science 320 (2008) 481–483.

[10] T.P. Michael, Plant genome size variation: bloating and purging DNA, Brief. Funct. Genomics (2014), http://dx.doi.org/10.1093/bfgp/elu1005 (in press).

[11] J.A. Fawcett, S. Maere, Y. Van de Peer, Plants with double genomes might have had a better chance to survive the Cretaceous-Tertiary extinction event, Proc. Natl. Acad. Sci. U. S. A. 106 (2009) 5737–5742.

[12] M. Ha, E.D. Kim, Z.J. Chen, Duplicate genes increase expression diversity in closely related species and allopolyploids, Proc. Natl. Acad. Sci. U. S. A. 106 (2009) 2295–2300.

[13] K. Vanneste, G. Baele, S. Maere, Y. Van de Peer, Analysis of 41 plant genomes supports a wave of successful genome duplications in association with the Cretaceous–Paleogene boundary, Genome Res. 24 (2014) 1334–1347.

[14] M.E. Schranz, S. Mohammadin, P.P. Edger, Ancient whole genome duplications, novelty and diversification: the WGD Radiation Lag-Time Model, Curr. Opin. Plant Biol. 15 (2012) 147–153.

[15] S. Magadum, U. Banerjee, P. Murugan, D. Gangapur, R. Ravikesavan, Gene duplication as a major force in evolution, J. Genet. 92 (2013) 155–161.

[16] A.H. Paterson, J.E. Bowers, B.A. Chapman, Ancient polyploidization predating divergence of the cereals, and its consequences for comparative genomics, Proc. Natl. Acad. Sci. U. S. A. 101 (2004) 9903–9908.

[17] L.Y. Cui, P.K. Wall, J.H. Leebens-Mack, B.G. Lindsay, D.E. Soltis, J.J. Doyle, P.S. Soltis, J.E. Carlson, K. Arumuganathan, A. Barakat, V.A. Albert, H. Ma, C.W. dePamphilis, Widespread genome duplications throughout the history of flowering plants, Genome Res. 16 (2006) 738–749.

[18] O. Jaillon, J.M. Aury, B. Noel, A. Policriti, C. Clepet, A. Casagrande, N. Choisne, S. Aubourg, N. Vitulo, C. Jubin, A. Vezzi, F. Legeai, P. Hugueney, C. Dasilva, D. Horner, E. Mica, D. Jublot, J. Poulain, C. Bruyere, A. Billault, B. Segurens, M. Gouyvenoux, E. Ugarte, F. Cattonaro, V. Anthouard, V. Vico, C. Del Fabbro, M. Alaux, G. Di Gaspero, V. Dumas, N. Felice, S. Paillard, I. Juman, M. Moroldo, S. Scalabrin, A. Canaguier, I. Le Clainche, G. Malacrida, E. Durand, G. Pesole, V. Laucou, P. Chatelet, D. Merdinoglu, M. Delledonne, M. Pezzotti, A. Lecharny, C. Scarpelli, F. Artiguenave, M.E. Pe, G. Valle, M. Morgante, M. Caboche, A.F. Adam-Blondon, J. Weissenbach, F. Quetier, P. Wincker, F.-I. Public, The grapevine genome sequence suggests ancestral hexaploidization in major angiosperm phyla, Nature 449 (2007) 463–467.

[19] H.B. Tang, X.Y. Wang, J.E. Bowers, R. Ming, M. Alam, A.H. Paterson, Unraveling ancient hexaploidy through multiply-aligned angiosperm gene maps, Genome Res. 18 (2008) 1944–1954.

[20] Y. Van de Peer, J.A. Fawcett, S. Proost, L. Sterck, K. Vandepoele, The flowering world: a tale of duplications, Trends Plant Sci. 14 (2009) 680–688.

[21] Y.N. Jiao, N.J. Wickett, S. Ayyampalayam, A.S. Chanderbali, L. Landherr, P.E. Ralph, L.P. Tomsho, Y. Hu, H.Y. Liang, P.S. Soltis, D.E. Soltis, S.W. Clifton, S.E. Schlarbaum, S.C. Schuster, H. Ma, J. Leebens-Mack, C.W. dePamphilis, Ancestral polyploidy in seed plants and angiosperms, Nature 473 (2011) 97–100.

[22] Y.N. Jiao, J. Leebens-Mack, S. Ayyampalayam, J.E. Bowers, M.R. McKain, J. McNeal, M. Rolf, D.R. Ruzicka, E. Wafula, N.J. Wickett, X.L. Wu, Y. Zhang, J. Wang, Y.T. Zhang, E.J. Carpenter, M.K. Deyholos, T.M. Kutchan, A.S. Chanderbali, P.S. Soltis, D.W. Stevenson, R. McCombie, J.C. Pires, G.K.S. Wong, D.E. Soltis, C.W. dePamphilis, A genome triplication associated with early diversification of the core eudicots, Genome Biol. 13 (2012).

[23] M.E. Schranz, T.C. Osborn, Novel flowering time variation in the resynthesized polyploid *Brassica napus*, J. Hered. 91 (2000) 242–246.

[24] H. Cohen, A. Fait, N. Tel-Zur, Morphological, cytological and metabolic consequences of autopolyploidization in Hylocereus (Cactaceae) species, BMC Plant Biol. 13 (2013).

[25] L. Comai, The advantages and disadvantages of being polyploid, Nat. Rev. Genet. 6 (2005) 836–846.

[26] K.L. Adams, J.F. Wendel, Polyploidy and genome evolution in plants, Curr. Opin. Plant Biol. 8 (2005) 135–141.

[27] Z.J. Chen, N. Ni, Mechanisms of genomic rearrangements and gene expression changes in plant polyploids, Bioessays 28 (2006) 240–252.

[28] K.M. Song, P. Lu, K.L. Tang, T.C. Osborn, Rapid genome change in synthetic polyploids of Brassica and its implications for polyploid evolution, Proc. Natl. Acad. Sci. U. S. A. 92 (1995) 7719–7723.

[29] S.D. Nicolas, G. Le Mignon, F. Eber, O. Coriton, H. Monod, V. Clouet, V. Huteau, A. Lostanlen, R. Delourme, B. Chalhoub, C.D. Ryder, A.M. Chevre, E. Jenczewski, Homeologous recombination plays a major role in chromosome rearrangements that occur during meiosis of *Brassica napus* haploids, Genetics 175 (2007) 487–503.

[30] R.T. Gaeta, J.C. Pires, Homoeologous recombination in allopolyploids: the polyploid ratchet, N. Phytol. 186 (2010) 18–28.

[31] L. Grandont, E. Jenczewski, A. Lloyd, Meiosis and its deviations in polyploid plants, Cytogenet. Genome Res. 140 (2013) 171–184.

[32] M.J. Lipman, M. Chester, P.S. Soltis, D.E. Soltis, Natural hybrids between Tragopogon Mirus and T. Miscellus (Asteraceae): a new perspective on karyotypic

changes following hybridization at the polyploid level, Am. J. Bot. 100 (2013) 2016–2022.
[33] H. Shaked, K. Kashkush, H. Ozkan, M. Feldman, A.A. Levy, Sequence elimination and cytosine methylation are rapid and reproducible responses of the genome to wide hybridization and allopolyploidy in wheat, Plant Cell 13 (2001) 1749–1759.
[34] K. Kashkush, M. Feldman, A.A. Levy, Gene loss, silencing and activation in a newly synthesized wheat allotetraploid, Genetics 160 (2002) 1651–1659.
[35] A. Madlung, R.W. Masuelli, B. Watson, S.H. Reynolds, J. Davison, L. Comai, Remodeling of DNA methylation and phenotypic and transcriptional changes in synthetic Arabidopsis allotetraploids, Plant Physiol. 129 (2002) 733–746.
[36] A. Madlung, A.P. Tyagi, B. Watson, H.M. Jiang, T. Kagochi, R.W. Doerge, R. Martienssen, L. Comai, Genomic changes in synthetic Arabidopsis polyploids, Plant J. 41 (2005) 221–230.
[37] Z.J. Chen, Genetic and epigenetic mechanisms for gene expression and phenotypic variation in plant polyploids, Annu. Rev. Plant Biol. 58 (2007) 377–406.
[38] R.T. Gaeta, J.C. Pires, F. Iniguez-Luy, E. Leon, T.C. Osborn, Genomic changes in resynthesized Brassica napus and their effect on gene expression and phenotype, Plant Cell 19 (2007) 3403–3417.
[39] K.L. Adams, J.F. Wendel, Novel patterns of gene expression in polyploid plants, Trends Genet. 21 (2005) 539–543.
[40] L. Comai, A.P. Tyagi, K. Winter, R. Holmes-Davis, S.H. Reynolds, Y. Stevens, B. Byers, Phenotypic instability and rapid gene silencing in newly formed Arabidopsis allotetraploids, Plant Cell 12 (2000) 1551–1567.
[41] J.L. Wang, L. Tian, A. Madlung, H.S. Lee, M. Chen, J.J. Lee, B. Watson, T. Kagochi, L. Comai, Z.J. Chen, Stochastic and epigenetic changes of gene expression in Arabidopsis polyploids, Genetics 167 (2004) 1961–1973.
[42] R.M. Stupar, P.B. Bhaskar, B.S. Yandell, W.A. Rensink, A.L. Hart, S. Ouyang, R.E. Veilleux, J.S. Busse, R.J. Erhardt, C.R. Buell, J.M. Jiang, Phenotypic and transcriptomic changes associated with potato autopolyploidization, Genetics 176 (2007) 2055–2067.
[43] I. Mayrose, S.H. Zhan, C.J. Rothfels, K. Magnuson-Ford, M.S. Barker, L.H. Rieseberg, S.P. Otto, Recently formed polyploid plants diversify at lower rates, Science 333 (2011) 1257.
[44] J. Koh, P.S. Soltis, D.E. Soltis, Homeolog loss and expression changes in natural populations of the recently and repeatedly formed allotetraploid *Tragopogon mirus* (Asteraceae), BMC Genomics 11 (2010).
[45] M. Semon, K.H. Wolfe, Consequences of genome duplication, Curr. Opin. Genet. Dev. 17 (2007) 505–512.
[46] J.A. Tate, P. Joshi, K.A. Soltis, P.S. Soltis, D.E. Soltis, On the road to diploidization? Homoeolog loss in independently formed populations of the allopolyploid *Tragopogon miscellus* (Asteraceae), BMC Plant Biol. 9 (2009).
[47] P.G. Zhang, S.Z. Huang, A.L. Pin, K.L. Adams, Extensive divergence in alternative splicing patterns after gene and genome duplication during the evolutionary history of Arabidopsis, Mol. Biol. Evol. 27 (2010) 1686–1697.
[48] C. Hu, S.Y. Lin, W.T. Chi, Y.Y. Charng, Recent gene duplication and subfunctionalization produced a mitochondrial GrpE, the nucleotide exchange factor of the Hsp70 complex, specialized in thermotolerance to chronic heat stress in Arabidopsis, Plant Physiol. 158 (2012) 747–758.
[49] G.X. Xu, C.C. Guo, H.Y. Shan, H.Z. Kong, Divergence of duplicate genes in exon–intron structure, Proc. Natl. Acad. Sci. U. S. A. 109 (2012) 1187–1192.
[50] R. De Smet, K.L. Adams, K. Vandepoele, M.C.E. van Montagu, S. Maere, Y. Van de Peer, Convergent gene loss following gene and genome duplications creates single-copy families in flowering plants, Proc. Natl. Acad. Sci. U. S. A. 110 (2013) 2898–2903.
[51] A. Roulin, P.L. Auer, M. Libault, J. Schlueter, A. Farmer, G. May, G. Stacey, R.W. Doerge, S.A. Jackson, The fate of duplicated genes in a polyploid plant genome, Plant J. 73 (2013) 143–153.
[52] D.E. Soltis, P.S. Soltis, Polyploidy: recurrent formation and genome evolution, Trends Ecol. Evol. 14 (1999) 348–352.
[53] J.J. Doyle, L.E. Flagel, A.H. Paterson, R.A. Rapp, D.E. Soltis, P.S. Soltis, J.F. Wendel, Evolutionary genetics of genome merger and doubling in plants, Annu. Rev. Genet. 42 (2008) 443–461.
[54] J. Ramsey, D.W. Schemske, Pathways, mechanisms, and rates of polyploid formation in flowering plants, Annu. Rev. Ecol. Syst. 29 (1998) 467–501.
[55] R.C. Jackson, Polyploidy and diploidy – new perspectives on chromosome-pairing and its evolutionary implications, Am. J. Bot. 69 (1982) 1512–1523.
[56] E. Benavente, J. Orellana, Chromosome differentiation and pairing behavior of polyploids – an assessment on preferential metaphase-i associations in colchicine-induced autotetraploid hybrids within the genus Secale, Genetics 128 (1991) 433–442.
[57] J. Sybenga, Chromosome pairing affinity and quadrivalent formation in polyploids: do segmental allopolyploids exist? Genome 39 (1996) 1176–1184.
[58] A. Widmer, M. Baltisberger, Molecular evidence for allopolyploid speciation and a single origin of the narrow endemic *Draba ladina* (Brassicaceae), Am. J. Bot. 86 (1999) 1282–1289.
[59] B.K. Mable, 'Why polyploidy is rarer in animals than in plants': myths and mechanisms, Biol. J. Linn. Soc. 82 (2004) 453–466.
[60] K.Y. Lim, R. Matyasek, A. Kovarik, A. Leitch, Parental origin and genome evolution in the allopolyploid *Iris versicolor*, Ann. Bot. 100 (2007) 219–224.
[61] M. Töpel, M. Lundberg, T. Eriksson, B. Eriksen, Molecular data and ploidal levels indicate several putative allopolyploidization events in the genus Potentilla (Rosaceae), PLoS Curr. 16 (2011).
[62] R. Velasco, A. Zharkikh, J. Affourtit, A. Dhingra, A. Cestaro, A. Kalyanaraman, P. Fontana, S.K. Bhatnagar, M. Troggio, D. Pruss, S. Salvi, M. Pindo, P. Baldi, S. Castelletti, M. Cavaiuolo, G. Coppola, F. Costa, V. Cova, A. Dal Ri, V. Goremykin, M. Komjanc, S. Longhi, P. Magnago, G. Malacarne, M. Malnoy, D. Micheletti, M. Moretto, M. Perazzolli, A. Si-Ammour, S. Vezzulli, E. Zini, G. Eldredge, L.M. Fitzgerald, N. Gutin, J. Lanchbury, T. Macalma, J.T. Mitchell, J. Reid, B. Wardell, C. Kodira, Z. Chen, B. Desany, F. Niazi, M. Palmer, T. Koepke, D. Jiwan, S. Schaeffer, V. Krishnan, C. Wu, V.T. Chu, S.T. King, J. Vick, Q. Tao, A. Mraz, A. Stormo, K. Stormo, R. Bogden, D. Ederle, A. Stella, A. Vecchietti, M.M. Kater, S. Masiero, P. Lasserre, Y. Lespinasse, A.C. Allan, V. Bus, D. Chagne, R.N. Crowhurst, A.P. Gleave, E. Lavezzo, J.A. Fawcett, S. Proost, P. Rouze, L. Sterck, S. Toppo, B. Lazzari, R.P. Hellens, C.E. Durel, A. Gutin, R.E. Bumgarner, S.E. Gardiner, M. Skolnick, M. Egholm, Y. Van de Peer, F. Salamini, R. Viola, The genome of the domesticated apple (*Malus* x *domestica* Borkh.), Nat. Genet. 42 (2010) 833.
[63] G.I. Lavia, A.M. Ortiz, G. Robledo, A. Fernandez, G. Seijo, Origin of triploid *Arachis pintoi* (Leguminosae) by autopolyploidy evidenced by FISH and meiotic behaviour, Ann. Bot. 108 (2011) 103–111.
[64] G.Y. Lu, F. Zhang, P.Y. Zheng, Y. Cheng, F.L. Liu, G.P. Fu, X.K. Zhang, Relationship among yield components and selection criteria for yield improvement in early rapeseed (*Brassica napus* L.), Agric. Sci. China 10 (2011) 997–1003.
[65] Y. Wang, S.L. Sun, B. Liu, H. Wang, J. Deng, Y.C. Liao, Q. Wang, F. Cheng, X.W. Wang, J. Wu, A sequence-based genetic linkage map as a reference for *Brassica rapa* pseudochromosome assembly, BMC Genomics 12 (2011).
[66] W.R. Rice, E.E. Hostert, Laboratory experiments on speciation – what have we learned in 40 years, Evolution 47 (1993) 1637–1653.
[67] R. Faria, S. Renaut, J. Galindo, C. Pinho, J. Melo-Ferreira, M. Melo, F. Jones, W. Salzburger, D. Schluter, R. Butlin, Advances in ecological speciation: an integrative approach, Mol. Ecol. 23 (2014) 513–521.
[68] J. Mallet, Hybrid speciation, Nat. Rev. 446 (2007) 279–283.
[69] R. Abbott, D. Albach, S. Ansell, J.W. Arntzen, S.J.E. Baird, N. Bierne, J.W. Boughman, A. Brelsford, C.A. Buerkle, R. Buggs, R.K. Butlin, U. Dieckmann, F. Eroukhmanoff, A. Grill, S.H. Cahan, J.S. Hermansen, G. Hewitt, A.G. Hudson, C. Jiggins, J. Jones, B. Keller, T. Marczewski, J. Mallet, P. Martinez-Rodriguez, M. Most, S. Mullen, R. Nichols, A.W. Nolte, C. Parisod, K. Pfennig, A.M. Rice, M.G. Ritchie, B. Seifert, C.M. Smadja, R. Stelkens, J.M. Szymura, R. Vainola, J.B.W. Wolf, D. Zinner, Hybridization and speciation, J. Evol. Biol. 26 (2013) 229–246.
[70] R. Veilleux, Diploid and polyploid gametes in crop plants: mechanisms of formation and utilization in plant breeding, Plant Breed. Rev. 3 (1985) 252–288.
[71] F. Bretagnolle, J.D. Thompson, Tansley review No. 78. Gametes with the somatic (sic) chromosome number: mechanisms of their formation and role in the evolution of autopolypoid plants, N. Phytol. 129 (1995) 1–22.
[72] L. Brownfield, C. Kohler, Unreduced gamete formation in plants: mechanisms and prospects, J. Exp. Bot. 62 (2011) 1659–1668.
[73] N. De Storme, D. Geelen, Sexual polyploidization in plants: cytological mechanisms and molecular regulation, N. Phytol. 198 (2013) 670–684.
[74] R.J. Scott, M. Spielman, J. Bailey, H.G. Dickinson, Parent-of-origin effects on seed development in *Arabidopsis thaliana*, Development 125 (1998) 3329–3341.
[75] C. Simioni, M.T. Schifino-Wittmann, M. Dall'Agnol, Sexual polyploidization in red clover, Sci. Agr. 63 (2006) 26–31.
[76] C. Kohler, O.M. Scheid, A. Erilova, The impact of the triploid block on the origin and evolution of polyploid plants, Trends Genet. 26 (2010) 142–148.
[77] I.M. Henry, B.P. Dilkes, K. Young, B. Watson, H. Wu, L. Comai, Aneuploidy and genetic variation in the *Arabidopsis thaliana* triploid response, Genetics 170 (2005) 1979–1988.
[78] E.L. Camadro, Cytological mechanisms of 2n microspore formation in garden Asparagus, Hortscience 27 (1992) 831–832.
[79] N. Shamina, N. Dorogova, N. Goncharov, A. Orlova, S. Trunova, Abnormalities of spindle and cytokine behavior leading to the formation of meiotic restitution nuclei in intergeneric cereal hybrids, Cell Biol. Int. 23 (1999) 863–870.
[80] N.V. Shamina, O.G. Silkova, E.G. Seriukova, Monopolar spindles in meiosis of intergeneric cereal hybrids, Cell Biol. Int. 27 (2003) 657–664.
[81] S. Andreuzza, I. Siddiqi, Spindle positioning, meiotic nonreduction, and polyploidy in plants, PLoS Genet. 4 (2008).
[82] I. d'Erfurth, S. Jolivet, N. Froger, O. Catrice, M. Novatchkova, M. Simon, E. Jenczewski, R. Mercier, Mutations in *AtPS1* (*Arabidopsis thaliana Parallel Spindle 1*) lead to the production of diploid pollen grains, PLoS Genet. 4 (2008).
[83] N. De Storme, D. Geelen, The Arabidopsis mutant jason produces unreduced first division restitution male gametes through a parallel/fused spindle mechanism in meiosis II, Plant Physiol. 155 (2011) 1403–1415.
[84] C. Conicella, G. Genualdo, A. Errico, L. Frusciante, L.M. Monti, Meiotic restitution mechanisms and 2n pollen formation in a *Solanum tuberosum* dihaploid and in dihaploid x wild species hybrids, Plant Breed. 115 (1996) 157–161.
[85] C. Conicella, A. Capo, M. Cammareri, A. Errico, N. Shamina, L.M. Monti, Elucidation of meiotic nuclear restitution mechanisms in potato through analysis of microtubular cytoskeleton, Euphytica 133 (2003) 107–115.
[86] L.A.B. Lopez-Lavalle, G. Orjeda, Occurrence and cytological mechanism of 2n pollen formation in a tetraploid accession of *Ipomoea batatas* (sweet potato), J. Hered. 93 (2002) 185–192.
[87] J.F. Zhang, Z.Z. Wei, D. Li, B.L. Li, Using SSR markers to study the mechanism of 2n pollen formation in *Populus* x *euramericana* (Dode) Guinier and *P.* x *popularis*, Ann. For. Sci. 66 (2009).
[88] Y.H. Li, Y.A. Shen, C. Cai, C.C. Zhong, L. Zhu, M. Yuan, H.Y. Ren, The type II Arabidopsis Formin14 interacts with microtubules and microfilaments to regulate cell division, Plant Cell 22 (2010) 2710–2726.
[89] M. Iwanaga, S.J. Peloquin, Origin and evolution of cultivated tetraploid potatoes via 2n-gametes, Theor. Appl. Genet. 61 (1982) 161–169.

[90] K. Watanabe, S.J. Peloquin, Occurrence of 2n pollen and Ps gene – frequencies in cultivated groups and their related wild-species in tuber-bearing Solanums, Theor. Appl. Genet. 78 (1989) 329–336.
[91] D. Mok, S.J. Peloquin, Three mechanisms of 2n pollen formation in diploid potatoes, Can. J. Genet. Cytol. 17 (1975) 217–225.
[92] Z.H. Zhang, X.Y. Kang, Cytological characteristics of numerically unreduced pollen production in *Populus tomentosa* Carr, Euphytica 173 (2010) 151–159.
[93] T.W. Pfeiffer, E.T. Bingham, Abnormal meiosis in alfalfa, Medicago-Sativa – cytology of 2n egg and 4n pollen formation, Can. J. Genet. Cytol. 25 (1983) 107–112.
[94] M. Spielman, D. Preuss, F.L. Li, W.E. Browne, R.J. Scott, H.G. Dickinson, TETRASPORE is required for male meiotic cytokinesis in *Arabidopsis thaliana*, Development 124 (1997) 2645–2657.
[95] M.S. Pagliarini, S.Y. Takayama, P.M. de Freitas, L.R. Carraro, E.V. Adamowski, N. Silva, L.A.R. Batista, Failure of cytokinesis and 2n gamete formation in Brazilian accessions of *Paspalum*, Euphytica 108 (1999) 129–135.
[96] N. De Storme, G.P. Copenhaver, D. Geelen, Production of diploid male gametes in Arabidopsis by cold-induced destabilization of postmeiotic radial microtubule arrays, Plant Physiol. 160 (2012) 1–19.
[97] K.R. Boldrini, M.S. Pagliarini, C.B. Do Valle, Abnormal timing of cytokinesis in microsporogenesis in *Brachiaria humidicola* (Poaceae: Paniceae), J. Genet. 85 (2006) 225–228.
[98] M.N. Premachandran, R. Lalitha, Microsporogenesis in a fertile *Saccharum officinarum* x *Erianthus arundinaceus* hybrid with floral abnormalities, J. Sugarcane Res. 2 (2012) 11–17.
[99] V.M. Gómez-Rodríguez, B. Rodríguez-Garay, R. Barba-Gonzalez, Meiotic restitution mechanisms involved in the formation of 2n pollen in *Agave tequilana* Weber and *Agave angustifolia* Haw, SpringerPlus 1 (2012) 1–7.
[100] C.Y. Yang, M. Spielman, J.P. Coles, Y. Li, S. Ghelani, V. Bourdon, R.C. Brown, B.E. Lemmon, R.J. Scott, H.G. Dickinson, TETRASPORE encodes a kinesin required for male meiotic cytokinesis in Arabidopsis, Plant J. 34 (2003) 229–240.
[101] Q.N. Zeng, J.G. Chen, B.E. Ellis, AtMPK4 is required for male-specific meiotic cytokinesis in Arabidopsis, Plant J. 67 (2011) 895–906.
[102] N.V. Shamina, O.A. Shatskaya, Two novel meiotic restitution mechanisms in haploid maize (*Zea mays* L.), Russ. J. Genet. 47 (2011) 438–445.
[103] L.C. Enns, M.M. Kanaoka, K.U. Torii, L. Comai, K. Okada, R.E. Cleland, Two callose synthases, GSL1 and GSL5, play an essential and redundant role in plant and pollen development and in fertility, Plant Mol. Biol. 58 (2005) 333–349.
[104] T. Krupnova, Y.D. Stierhof, U. Hiller, G. Strompen, S. Muller, The microtubule-associated kinase-like protein RUNKEL functions in somatic and syncytial cytokinesis, Plant J. 74 (2013) 781–791.
[105] S.A. Oh, V. Bourdon, H.G. Dickinson, D. Twell, S.K. Park, Arabidopsis fused kinase TWO-IN-ONE dominantly inhibits male meiotic cytokinesis, Plant Reprod. 27 (2014) 7–17.
[106] M. Juranic, K.O. Srilunchang, N.G. Krohn, D. Leljak-Levanic, S. Sprunck, T. Dresselhaus, Germline-specific MATH-BTB substrate adaptor MAB1 regulates spindle length and nuclei identity in maize, Plant Cell 24 (2012) 4974–4991.
[107] S. Tavoletti, Cytological mechanisms of 2n egg formation in a diploid genotype of *Medicago Sativa* subsp. Falcata, Euphytica 75 (1994) 1–8.
[108] M. Ravi, M.P.A. Marimuthu, I. Siddiqi, Gamete formation without meiosis in Arabidopsis, Nature 451 (2008) 1121–1124.
[109] I. d'Erfurth, S. Jolivet, N. Froger, O. Catrice, M. Novatchkova, R. Mercier, Turning meiosis into mitosis, PLoS Biol. 7 (2009).
[110] M. Singh, S. Goel, R.B. Meeley, C. Dantec, H. Parrinello, C. Michaud, O. Leblanc, D. Grimanelli, Production of viable gametes without meiosis in maize deficient for an ARGONAUTE protein, Plant Cell 23 (2011) 443–458.
[111] G. Barcaccia, Alfalfa reproductive mutants: cytological mechanisms, molecular markers and candidate genes for apomeiosis, in: D. Rosellini, F. Veronesi (Eds.), Breeding and Seed Production for Conventional and Organic Agriculture. Proceedings of the XXVI Meeting of the EUCARPIA Fodder Crops and Amenity Grasses Section, XVI Meeting of the EUCARPIA *Medicago* spp. Group, Perugia, Italy, 2–7 September, 2006, pp. 270–274.
[112] P. Ozias-Akins, Apomixis: developmental characteristics and genetics, Crit. Rev. Plant Sci. 25 (2006) 199–214.
[113] D. Grimanelli, O. Leblanc, E. Espinosa, E. Perotti, D.G. De Leon, Y. Savidan, Mapping diplosporous apomixis in tetraploid Tripsacum: one gene or several genes? Heredity 80 (1998) 33–39.
[114] P.J. van Dijk, J.M.T. Bakx-Schotman, Formation of unreduced megaspores (diplospory) in apomictic dandelions (*Taraxacum officinale*, s.l) is controlled by a sex-specific dominant locus, Genetics 166 (2004) 483–492.
[115] R.D. Noyes, Inheritance of apomeiosis (diplospory) in fleabanes (Erigeron: Asteraceae), Heredity 94 (2005) 193–198.
[116] I. d'Erfurth, L. Cromer, S. Jolivet, C. Girard, C. Horlow, Y.J. Sun, J.P.C. To, L.E. Berchowitz, G.P. Copenhaver, R. Mercier, The CYCLIN-A CYCA1;2/TAM is required for the meiosis I to meiosis II transition and cooperates with OSD1 for the prophase to first meiotic division transition, PLoS Genet. 6 (2010).
[117] D.M. Stelly, S.J. Peloquin, Formation of 2n megagametophytes in diploid tuber-bearing solanums, Am. J. Bot. 73 (1986) 1351–1363.
[118] C. Conicella, A. Barone, A. Delgiudice, L. Frusciante, L.M. Monti, Cytological evidences of SDR-FDR mixture in the formation of 2n eggs in a potato diploid clone, Theor. Appl. Genet. 81 (1991) 59–63.
[119] C. Ferris, R.S. Callow, A.J. Gray, Mixed first and second division restitution in male meiosis of *Hierochloë odorata* (L.) Beauv (Holy Grass), Heredity 69 (1991) 21–31.
[120] K.B. Lim, T.M. Shen, R. Barba-Gonzalez, M.S. Ramanna, J.M. Van Tuyl, Occurrence of SDR 2N-gametes in *Lilium* hybrids, Breed. Sci. 54 (2004) 13–18.
[121] A.M. Koltunow, Apomixis – embryo sacs and embryos formed without meiosis or fertilization in ovules, Plant Cell 5 (1993) 1425–1437.
[122] A.M. Koltunow, U. Grossniklaus, Apomixis: a developmental perspective, Annu. Rev. Plant Biol. 54 (2003) 547–574.
[123] E. Hörandl, D. Hojsgaard, The evolution of apomixis in angiosperms: a reappraisal, Plant Biosyst. 146 (2012) 681–693.
[124] H.M. Liu, R.J. Dyer, Z.Y. Guo, Z. Meng, J.H. Li, H. Schneider, The evolutionary dynamics of apomixis in ferns: a case study from polystichoid ferns, J. Bot. (2012) 1–11.
[125] R.A. Bicknell, A.M. Koltunow, Understanding apomixis: recent advances and remaining conundrums, Plant Cell 16 (2004) S228–S245.
[126] M.B. Burgess, K.R. Cushman, E.T. Doucette, N. Talent, C.T. Frye, C.S. Campbell, Effects of apomixis and polyploidy on diversification and geographic distribution in Amelanchier (Rosaceae), Am. J. Bot. 101 (2014) 1375–1387.
[127] A.C. Cosendai, J. Rodewald, E. Horandl, Origin and distribution of autopolyploids via apomixis in the alpine species *Ranunculus kuepferi* (Ranunculaceae), Taxon 60 (2011) 355–364.
[128] J.T. Lovell, O.M. Aliyu, M. Mau, M.E. Schranz, M. Koch, C. Kiefer, B.H. Song, T. Mitchell-Olds, T.F. Sharbel, On the origin and evolution of apomixis in Boechera, Plant Reprod. 26 (2013) 309–315.
[129] S.P. Otto, J. Whitton, Polyploidy: incidence and evolution, Annu. Rev. Genet. 34 (2000) 401–437.
[130] P. Ozias-Akins, P.J. van Dijk, Mendelian genetics of apomixis in plants, Annu. Rev. Genet. 41 (2007) 509–537.
[131] M.L. Voigt-Zielinski, M. Piwczynski, T.F. Sharbel, Differential effects of polyploidy and diploidy on fitness of apomictic Boechera, Sex. Plant Reprod. 25 (2012) 97–109.
[132] J.-P. Vielle-Calzada, E. Hernández-Lagana, D. Rodríguez-Leal, I. Rodríguez-Arévalo, G. León-Martínez, U. Abad-Vivero, E. Demesa-Arévalo, A. Armenta-Medina, C. Alvarez-Mejía, Reproductive versatility and the epigenetic control of female gametogenesis, Advance 77 (2012) 17–21.
[133] E. Wijnker, A. Schnittger, Control of the meiotic cell division program in plants, Plant Reprod. 26 (2013) 143–158.
[134] E. Iwata, S. Ikeda, S. Matsunaga, M. Kurata, Y. Yoshioka, M.C. Criqui, P. Genschik, M. Ito, GIGAS CELL1, a novel negative regulator of the anaphase-promoting complex/cyclosome, is required for proper mitotic progression and cell fate determination in Arabidopsis, Plant Cell 23 (2011) 4382–4393.
[135] L. Cromer, J. Heyman, S. Touati, H. Harashima, E. Araou, C. Girard, C. Horlow, K. Wassmann, A. Schnittger, L. De Veylder, R. Mercier, OSD1 Promotes meiotic progression via APC/C inhibition and forms a regulatory network with TDM and CYCA1;2/TAM, PLoS Genet. 8 (2012).
[136] P. Bulankova, N. Riehs-Kearnan, M.K. Nowack, A. Schnittger, K. Riha, Meiotic progression in Arabidopsis is governed by complex regulatory interactions between SMG7, TDM1, and the meiosis I-specific cyclin TAM, Plant Cell 22 (2010) 3791–3803.
[137] I. Siddiqi, G. Ganesh, U. Grossniklaus, V. Subbiah, The dyad gene is required for progression through female meiosis in Arabidopsis, Development 127 (2000) 197–207.
[138] B. Agashe, C.K. Prasad, I. Siddiqi, Identification and analysis of DYAD: a gene required for meiotic chromosome organisation and female meiotic progression in Arabidopsis, Development 129 (2002) 3935–3943.
[139] J.M. Corral, H. Vogel, O.M. Aliyu, G. Hensel, T. Thiel, J. Kumlehn, T.F. Sharbel, A conserved apomixis-specific polymorphism is correlated with exclusive exonuclease expression in premeiotic ovules of apomictic Boechera species, Plant Physiol. 163 (2013) 1660–1672.
[140] M. Garcia-Aguilar, C. Michaud, O. Leblanc, D. Grimanelli, Inactivation of a DNA methylation pathway in maize reproductive organs results in apomixis-like phenotypes, Plant Cell 22 (2010) 3249–3267.
[141] V. Olmedo-Monfil, N. Duran-Figueroa, M. Arteaga-Vazquez, E. Demesa-Arevalo, D. Autran, D. Grimanelli, R.K. Slotkin, R.A. Martienssen, J.P. Vielle-Calzada, Control of female gamete formation by a small RNA pathway in Arabidopsis, Nature 464 (2010) 628–632.
[142] R. Mercier, D. Vezon, E. Bullier, J.C. Motamayor, A. Sellier, F. Lefevre, G. Pelletier, C. Horlow, SWITCH1 (SWI1): a novel protein required for the establishment of sister chromatid cohesion and for bivalent formation at meiosis, Genes Dev. 15 (2001) 1859–1871.
[143] R. Mercier, S.J. Armstrong, C. Horlow, N.P. Jackson, C.A. Makaroff, D. Vezon, G. Pelletier, G.H. Jones, F.C.H. Franklin, The meiotic protein SWI1 is required for axial element formation and recombination initiation in Arabidopsis, Development 130 (2003) 3309–3318.
[144] K.A. Boateng, X. Yang, F. Dong, H.A. Owen, C.A. Makaroff, SWI1 is required for meiotic chromosome remodeling events, Mol. Plant 1 (2008) 620–633.
[145] H. Ozkan, A.A. Levy, M. Feldman, Allopolyploidy-induced rapid genome evolution in the wheat (Aegilops-Triticum) group, Plant Cell 13 (2001) 1735–1747.
[146] O. Paun, M.F. Fay, D.E. Soltis, M.W. Chase, Genetic and epigenetic alterations after hybridization and genome doubling, Taxon 56 (2007) 649–656.
[147] B. Liu, C.M. Xu, N. Zhao, B. Qi, J.N. Kimatu, J.S. Pang, F.P. Han, Rapid genomic changes in polyploid wheat and related species: implications for genome evolution and genetic improvement, J. Genet. Genomics 36 (2009) 519–528.
[148] Y.Z. Dong, Z.L. Liu, X.H. Shan, T. Qiu, M.Y. He, B. Liu, Allopolyploidy in wheat induces rapid and heritable alterations in DNA methylation patterns of cellular genes and mobile elements, Russ. J. Genet. 41 (2005) 890–896.
[149] B. Yaakov, K. Kashkush, Massive alterations of the methylation patterns around DNA transposons in the first four generations of a newly formed wheat allohexaploid, Genome 54 (2011) 42–49.

[150] M. Feldman, B. Liu, G. Segal, S. Abbo, A.A. Levy, J.M. Vega, Rapid elimination of low-copy DNA sequences in polyploid wheat: a possible mechanism for differentiation of homoeologous chromosomes, Genetics 147 (1997) 1381–1387.
[151] I. Mestiri, V. Chague, A.M. Tanguy, C. Huneau, V. Huteau, H. Belcram, O. Coriton, B. Chalhoub, J. Jahier, Newly synthesized wheat allohexaploids display progenitor-dependent meiotic stability and aneuploidy but structural genomic additivity, N. Phytol. 186 (2010) 86–101.
[152] Z.Y. Xiong, R.T. Gaeta, J.C. Pires, Homoeologous shuffling and chromosome compensation maintain genome balance in resynthesized allopolyploid *Brassica napus*, Proc. Natl. Acad. Sci. U. S. A. 108 (2011) 7908–7913.
[153] B.R. Baum, M. Feldman, Elimination of 5S DNA unit classes in newly formed allopolyploids of the genera Aegilops and Triticum, Genome 53 (2010) 430–438.
[154] J.R. Harlan, J.M.J. De Wet, On O. Winge and a prayer: the origins of polyploidy, Bot. Rev. 41 (1975) 361–690.
[155] L.L. Li, M. Jean, F. Belzile, The impact of sequence divergence and DNA mismatch repair on homeologous recombination in Arabidopsis, Plant J. 45 (2006) 908–916.
[156] C.G. Bozza, W.P. Pawlowski, The cytogenetics of homologous chromosome pairing in meiosis in plants, Cytogenet. Genome Res. 120 (2008) 313–319.
[157] Y.I. Lee, F.C. Chang, M.C. Chung, Chromosome pairing affinities in interspecific hybrids reflect phylogenetic distances among lady's slipper orchids (Paphiopedilum), Ann. Bot. 108 (2011) 113–121.
[158] F.M. Abbasi, A.H. Shah, F. Perveen, M. Afzal, M. Sajid, R. Masood, F. Nawaz, Genomic affinity between *Oryza sativa* and *Oryza brachyantha* as revealed by in situ hybridization and chromosome pairing, Afr. J. Biotechnol. 9 (2010) 3068–3072.
[159] M.S. Sohoo, K.S. Gill, Asynapsis in cluster bean (Cyamopsis-Tetragonoloba (L) Taub), Theor. Appl. Genet. 46 (1975) 411–412.
[160] M. Pradillo, E. Lopez, C. Romero, E. Sanchez-Moran, N. Cunado, J.L. Santos, An analysis of univalent segregation in meiotic mutants of *Arabidopsis thaliana*: a possible role for synaptonemal complex, Genetics 175 (2007) 505–511.
[161] M. Ravi, S.W.L. Chan, Haploid plants produced by centromere-mediated genome elimination, Nature 464 (2010) 615–618.
[162] X.W. Cai, S.S. Xu, Meiosis-driven genome variation in plants, Curr. Genomics 8 (2007) 151–161.
[163] E.B. Wagenaar, Meiotic restitution and the origin of polyploidy. I. Influence of genotype on polyploid seedset in a *Triticum crassum* x *T. turgidum* hybrid, Can. J. Genet. Cytol. 10 (1968) 836–843.
[164] P.P. Jauhar, M. Dogramaci-Altuntepe, T.S. Peterson, A.B. Almouslem, Seedset on synthetic haploids of durum wheat: cytological and molecular investigations, Crop Sci. 40 (2000) 1742–1749.
[165] V.K. Tiwari, N. Rawat, K. Neelam, G.S. Randhawa, K. Singh, P. Chhuneja, H.S. Dhaliwal, Development of *Triticum turgidum* subsp. *durum* – *Aegilops longissima* amphiploids with high iron and zinc content through unreduced gamete formation in F-1 hybrids, Genome 51 (2008) 757–766.
[166] Y.W. Yang, L.Q. Zhang, Y. Yen, Y.L. Zheng, D.C. Liu, Cytological evidence on meiotic restitution in pentaploid F1 hybrids between synthetic hexaploid wheat and *Aegilops variabilis*, Caryologia 63 (2010) 354–358.
[167] M.S. Ramanna, A.G.J. Kuipers, E. Jacobsen, Occurrence of numerically unreduced (2n) gametes in Alstroemeria interspecific hybrids and their significance for sexual polyploidisation, Euphytica 133 (2003) 95–106.
[168] P.P. Jauhar, Meiotic restitution in wheat polyhaploids (Amphihaploids): a potent evolutionary force, J. Hered. 98 (2007) 188–193.
[169] L.Q. Zhang, Y. Yen, Y.L. Zheng, D.C. Liu, Meiotic restriction in emmer wheat is controlled by one or more nuclear genes that continue to function in derived lines, Sex. Plant Reprod. 20 (2007) 159–166.
[170] L.Q. Zhang, Q.J. Chen, Z.W. Yuan, Z.G. Xiang, Y.L. Zheng, D.C. Liu, Production of aneuhaploid and euhaploid sporocytes by meiotic restitution in fertile hybrids between durum wheat Langdon chromosome substitution lines and *Aegilops tauschii*, J. Genet. Genomics 35 (2008) 617–623.
[171] M.Y. Chung, J.D. Chung, M. Ramanna, J.M. van Tuyl, K.B. Lim, Production of polyploids and unreduced gametes in *Lilium auratum* x *L. henryi* hybrid, Int. J. Biol. Sci. 9 (2013) 693–701.
[172] O.G. Silkova, A.I. Shchapova, L.A. Kravtsova, Mechanisms of meiotic restitution and their genetic regulation in wheat-rye polyhaploids, Russ. J. Genet. 39 (2003) 1271–1280.
[173] O.G. Silkova, A.I. Shchapova, V.K. Shumny, Meiotic restitution in amphihaploids in the tribe Triticeae, Russ. J. Genet. 47 (2011) 383–393.
[174] K.B. Lim, M.S. Ramanna, J.H. de Jong, E. Jacobsen, J.M. van Tuyl, Indeterminate Meiotic Restitution (IMR): a novel type of meiotic nuclear restitution mechanism detected in interspecific lily hybrids by GISH, Theor. Appl. Genet. 103 (2001) 219–230.
[175] X.W. Cai, S.S. Xu, X.W. Zhu, Mechanism of haploidy-dependent unreductional meiotic cell division in polyploid wheat, Chromosoma 119 (2010) 275–285.
[176] Y. Matsuoka, S. Nasuda, Durum wheat as a candidate for the unknown female progenitor of bread wheat: an empirical study with a highly fertile F-1 hybrid with *Aegilops tauschii* Coss, Theor. Appl. Genet. 109 (2004) 1710–1717.
[177] L.Y. Yan, X.Z. Zhang, G.J. Liu, Occurrence of unreduced gametes and ploidy restoration in haploid *Capsicum annuum* L., J. Hortic. Sci. Biotechnol. 75 (2000) 195–197.
[178] R.G. Kynast, D.W. Davis, R.L. Phillips, H.W. Rines, Gamete formation via meiotic nuclear restitution generates fertile amphiploid F1 (oat x maize) plants, Sex. Plant Reprod. 25 (2012) 111–122.
[179] L. Comai, A.P. Tyagi, M.A. Lysak, FISH analysis of meiosis in Arabidopsis allopolyploids, Chromosome Res. 11 (2003) 217–226.
[180] L.Q. Zhang, D.C. Liu, Y.L. Zheng, Z.H. Yan, S.F. Dai, Y.F. Li, Q. Jiang, Y.Q. Ye, Y. Yen, Frequent occurrence of unreduced gametes in *Triticum turgidum–Aegilops tauschii* hybrids, Euphytica 172 (2010) 285–294.
[181] A. Tayale, C. Parisod, Natural pathways to polyploidy in plants and consequences for genome reorganization, Cytogenet. Genome Res. 140 (2013) 79–96.
[182] L. Avivi, The effect of genes controlling different degrees of homoeologous pairing on quadrivalent frequency in induced autotetraploid lines of *Triticum longissimum*, Can. J. Genet. Cytol. 18 (1976) 357–364.
[183] S. Oleszczuk, A.J. Lukaszewski, The origin of unusual chromosome constitutions among newly formed allopolyploids, Am. J. Bot. 101 (2014) 1–9.
[184] G.L. Stebbins, Types of polyploids: their classification and significance, Adv. Genet. 1 (1947) 403–429.
[185] B.S. Baut, J.F. Doebley, DNA sequence evidence for the segmental allotetraploid origin of maize, Proc. Natl. Acad. Sci. U. S. A. 94 (1997) 6809–6814.
[186] T. Boff, M.T. Schifino-Wittmann, Segmental allopolyploidy and paleopolyploidy in species of Leucaena Benth: evidence from meiotic behaviour analysis, Hereditas 138 (2003) 27–35.
[187] W. Shinohara, Y. Ushio, A. Seo, N. Nakato, M. Kono, H. Kudoh, H. Tobe, N. Murakami, Evidence for hybrid origin and segmental allopolyploidy in eutetraploid and aneutetraploid *Lepisorus thunbergianus* (Polypodiaceae), Syst. Bot. 35 (2010) 20–29.
[188] M. Chester, J.P. Gallagher, V.V. Symonds, A.V.C. da Silva, E.V. Mavrodiev, A.R. Leitch, P.S. Soltis, D.E. Soltis, Extensive chromosomal variation in a recently formed natural allopolyploid species, *Tragopogon miscellus* (Asteraceae), Proc. Natl. Acad. Sci. U. S. A. 109 (2012) 1176–1181.
[189] K.L. Hunter, J.L. Betancourt, B.R. Riddle, T.R. Van Devender, K.L. Cole, W.G. Spaulding, Ploidy race distributions since the last glacial maximum in the North American desert shrub, Larrea tridentata, Global Ecol. Biogeogr. 10 (2001) 521–533.
[190] C.J. Wang, L.Q. Zhang, S.F. Dai, Y.L. Zheng, H.G. Zhang, D.C. Liu, Formation of unreduced gametes is impeded by homologous chromosome pairing in tetraploid *Triticum turgidum* x *Aegilops tauschii* hybrids, Euphytica 175 (2010) 323–329.
[191] O.G. Silkova, I.G. Adonina, E.A. Krivosheina, A.I. Shchapova, V.K. Shumny, Chromosome pairing in meiosis of partially fertile wheat/rye hybrids, Plant Reprod. 26 (2013) 33–41.
[192] P.P. Jauhar, A.B. Almouslem, T.S. Peterson, L.R. Joppa, Inter- and intragenomic chromosome pairing in haploids of durum wheat, J. Hered. 90 (1999) 437–445.
[193] D. Pignone, Non-reductional meiosis in a*Triticum turgidum* × *Aegilops longissima* hybrid and in backcrosses of its amphidiploid with *T. turgidum* (Poaceae), Plant Syst. Evol. 187 (1993) 127–134.
[194] J.L. Gerton, R.S. Hawley, Homologous chromosome interactions in meiosis: diversity amidst conservation, Nat. Rev. Genet. 6 (2005) 477–487.
[195] D.Q. Ding, T. Haraguchi, Y. Hiraoka, From meiosis to postmeiotic events: alignment and recognition of homologous chromosomes in meiosis, FEBS J. 277 (2010) 565–570.
[196] P. Prieto, P. Shaw, G. Moore, Homologue recognition during meiosis is associated with a change in chromatin conformation, Nat. Cell Biol. 6 (2004) 906–908.
[197] I. Colas, P. Shaw, P. Prieto, M. Wanous, W. Spielmeyer, R. Mago, G. Moore, Effective chromosome pairing requires chromatin remodeling at the onset of meiosis, Proc. Natl. Acad. Sci. U. S. A. 105 (2008) 6075–6080.
[198] B. Maestra, J.H. de Jong, K. Shepherd, T. Naranjo, Chromosome arrangement and behaviour of two rye homologous telosomes at the onset of meiosis in disomic wheat-5RL addition lines with and without the Ph1 locus, Chromosome Res. 10 (2002) 655–667.
[199] P. Prieto, G. Moore, S. Reader, Control of conformation changes associated with homologue recognition during meiosis, Theor. Appl. Genet. 111 (2005) 505–510.
[200] E. Knight, E. Greer, T. Draeger, V. Thole, S. Reader, P. Shaw, G. Moore, Inducing chromosome pairing through premature condensation: analysis of wheat interspecific hybrids, Funct. Integr. Genomics 10 (2010) 603–608.
[201] M. Rezaei, A. Arzani, B.E. Sayed-Tabatabaei, Meiotic behaviour of tetraploid wheats (*Triticum turgidum* L.) and their synthetic hexaploid wheat derivates influenced by meiotic restitution and heat stress, J. Genet. 89 (2010) 401–407.
[202] E. Corredor, T. Naranjo, Effect of colchicine and telocentric chromosome conformation on centromere and telomere dynamics at meiotic prophase I in wheat-rye additions, Chromosome Res. 15 (2007) 231–245.
[203] F. Ressurreição, A. Barão, W. Viegas, M. Delgado, Haploid independent unreductional meiosis in hexaploid wheat, in: A. Swan (Ed.), Meiosis – Molecular Mechanisms and Cytogenetic Diversity, InTech, Rijeka, 2012.
[204] M.W. Bayliss, R. Riley, An analysis of temperature-dependent asynapsis in *Triticum aestivum*, Genet. Res. 20 (1972).
[205] A. Queiroz, T. Mellosampayo, W.S. Viegas, Identification of low-temperature stabilizing genes, controlling chromosome synapsis or recombination, in short arms of chromosomes from the homoeologous group-5 of *Triticum–Aestivum*, Hereditas 115 (1991) 37–41.
[206] M. Tanaka, Newly synthesized amphidiploids from the hybrids emmer wheats x *Aegilops squarrosa* varieties, Wheat Inform. Serv. 8 (1961).
[207] S.J. Xu, Y.S. Dong, Fertility and meiotic mechanisms of hybrids between chromosome autoduplication tetraploid wheats and *Aegilops* species, Genome 35 (1992) 379–384.

[208] S.J. Xu, L.R. Joppa, Mechanisms and inheritance of first division restitution in hybrids of wheat, rye, and *Aegilops–Squarrosa*, Genome 38 (1995) 607–615.
[209] S.J. Xu, L.R. Joppa, First-division restitution in hybrids of *Langdon durum* disomic substitution lines with rye and *Aegilops squarrosa*, Plant Breed. 119 (2000) 233–241.
[210] Y. Matsuoka, S. Takumi, T. Kawahara, Natural variation for the fertile triploid F1 hybrid formation in allohexaploid wheat speciation, Theor. Appl. Genet. 115 (2007) 509–518.
[211] D.C. Liu, X.J. Lan, Z.J. Yang, Y.L. Zheng, Y.M. Wei, Y.H. Zhou, A unique *Aegilops tauschii* genotype needles to immature embryo culture in cross with wheat, Acta Bot. Sin. 44 (2002) 708–713.
[212] Y. Matsuoka, S. Nasuda, Y. Ashida, M. Nitta, H. Tsujimoto, S. Takumi, T. Kawahara, Genetic basis for spontaneous hybrid genome doubling during allopolyploid speciation of common wheat shown by natural variation analyses of the paternal species, PLoS One 8 (2013).
[213] B.D. Dou, B.W. Hou, H.M. Xu, X.Y. Lou, X.F. Chi, J.B. Yang, F. Wang, Z.F. Ni, Q.X. Sun, Efficient mapping of a female sterile gene in wheat (*Triticum aestivum* L.), Genet. Res. 91 (2009) 337–343.
[214] M. Okamoto, Asynapsis effect of chromosome V, Wheat Inform. Serv. 5 (1957).
[215] R. Riley, V. Chapman, G. Kimber, Genetic control of chromosome pairing in intergeneric hybrids with wheat, Nature 183 (1959) 1244–1246.
[216] T. Mello-Sampayo, Genetic regulation of meiotic chromosome pairing by chromosome 3D of *Triticum aestivum*, Nat. New Biol. 230 (1971) 22–23.
[217] T. Sutton, R. Whitford, U. Baumann, C.M. Dong, J.A. Able, P. Langridge, The Ph2 pairing homoeologous locus of wheat (*Triticum aestivum*): identification of candidate meiotic genes using a comparative genetics approach, Plant J. 36 (2003) 443–456.
[218] E.R. Sears, Genetic control of chromosome pairing in common wheat, Annu. Rev. Genet. 10 (1976) 31–51.
[219] E.R. Sears, An induced mutant with homoeologous pairing in common wheat, Can. J. Genet. Cytol. 19 (1977) 585–593.
[220] P.P. Jauhar, M. Dogramaci, T.S. Peterson, Synthesis and cytological characterization of trigeneric hybrids of durum wheat with and without Ph1, Genome 47 (2004) 1173–1181.
[221] S. Griffiths, R. Sharp, T.N. Foote, I. Bertin, M. Wanous, S. Reader, I. Colas, G. Moore, Molecular characterization of *Ph1* as a major chromosome pairing locus in polyploid wheat, Nature 439 (2006) 749–752.
[222] N. Al-Kaff, E. Knight, I. Bertin, T. Foote, N. Hart, S. Griffiths, G. Moore, Detailed dissection of the chromosomal region containing the Ph1 locus in wheat *Triticum aestivum*: with deletion mutants and expression profiling, Ann. Bot. 101 (2008) 863–872.
[223] F.K. Yousafzai, N. Al-Kaff, G. Moore, Structural and functional relationship between the Ph1 locus protein 5B2 in wheat and CDK2 in mammals, Funct. Integr. Genomics 10 (2010) 157–166.
[224] J.H. Doonan, G. Kitsios, Functional evolution of cyclin-dependent kinases, Mol. Biotechnol. 42 (2009) 14–29.
[225] S.A. Boden, P. Langridge, G. Spangenberg, J.A. Able, TaASY1 promotes homologous chromosome interactions and is affected by deletion of Ph1, Plant J. 57 (2009) 487–497.
[226] E. Greer, A.C. Martin, A. Pendle, I. Colas, A.M.E. Jones, G. Moore, P. Shaw, The Ph1 locus suppresses Cdk2-type activity during premeiosis and meiosis in wheat, Plant Cell 24 (2012) 152–162.
[227] M. Martinez, N. Cunado, N. Carcelen, C. Romero, The Ph1 and Ph2 loci play different roles in the synaptic behaviour of hexaploid wheat *Triticum aestivum*, Theor. Appl. Genet. 103 (2001) 398–405.
[228] O.G. Silkova, O.B. Dobrovolskaya, A.I. Shchapova, V.K. Shumny, Features of the regulation of meiotic restitution in androgenic haploids of wheat-rye substitution lines 2R(2D)(1), 2R(2D)(3), and 6R(6A) (*Triticum aestivum* L., cultivar Saratovskaya 29/*Secale cereale* L., cultivar Onokhoiskaya), Russ. J. Genet. 45 (2009) 1062–1066.
[229] O.G. Silkova, A.I. Shchapova, V.K. Shumny, Role of rye chromosome 2R from wheat-rye substitution line 2R(2D)(1) (*Triticum aestivum* L. cv. Saratovskaya 29–*Secale cereale* L. cv. Onokhoiskaya) in genetic regulation of meiotic restitution in wheat-rye polyhaploids, Russ. J. Genet. 43 (2007) 805–814.
[230] J.D. Hollister, B.J. Arnold, E. Svedin, K.S. Xue, B.P. Dilkes, K. Bomblies, Genetic adaptation associated with genome-doubling in autotetraploid *Arabidopsis arenosa*, Plos Genet. 8 (2012).
[231] I.M. Henry, B.P. Dilkes, A. Tyagi, J. Gao, B. Christensen, L. Comai, The BOY NAMED SUE quantitative trait locus confers increased meiotic stability to an adapted natural allopolyploid of *Arabidopsis*, Plant Cell 26 (2014) 181–194.
[232] M. Okamoto, N. Inomata, Possibility of 5B-like effect in diploid species, Wheat Inform. Serv. 38 (1974) 15–16.
[233] J.G. Waines, A model for the origin of diploidizing mechanisms in polyploid species, Am. Nat. 110 (1976) 415–430.
[234] M. Feldman, A.A. Levy, Genome evolution due to allopolyploidization in wheat, Genetics 192 (2012) 763–774.
[235] K.B. Lim, M.S. Ramanna, J.M. Van Tuyl, Homoeologous recombination in interspecific hybrids of *Lilium*, Korean J. Breed. 35 (2003) 8–12.
[236] L. Yant, J.D. Hollister, K.M. Wright, B.J. Arnold, J.D. Higgins, F.C.H. Franklin, K. Bomblies, Meiotic adaptation to genome duplication in *Arabidopsis arenosa*, Curr. Biol. 23 (2013) 2151–2156.
[237] N. De Storme, D. Geelen, The impact of environmental stress on male reproductive development in plants: biological processes and molecular mechanisms, Plant Cell Environ. 37 (2014) 1–18.
[238] R.W. Parish, H.A. Phan, S. Iacuone, S.F. Li, Tapetal development and abiotic stress: a centre of vulnerability, Funct. Plant Biol. 39 (2012) 553–559.
[239] G. Barcaccia, S. Tavoletti, M. Falcinelli, F. Veronesi, Environmental influences on the frequency and viability of meiotic and apomeiotic cells of a diploid mutant of alfalfa, Crop Sci. 37 (1997) 70–76.
[240] Y. Pecrix, G. Rallo, H. Folzer, M. Cigna, S. Gudin, M. Le Bris, Polyploidization mechanisms: temperature environment can induce diploid gamete formation in *Rosa* sp., J. Exp. Bot. 62 (2011) 3587–3597.
[241] K. Watanabe, S.J. Peloquin, Cytological basis of 2n pollen formation in a wide-range of 2x, 4x, and 6x taxa from tuber-bearing *Solanum* species, Genome 36 (1993) 8–13.
[242] L.C. Stephens, Formation of unreduced pollen by an *Impatiens hawkeri* x *platypetala* interspecific hybrid, Hereditas 128 (1998) 251–255.
[243] V. Negri, G. Lemmi, Effect of selection and temperature stress on the production of 2n gametes in *Lotus tenuis*, Plant Breed. 117 (1998) 345–349.
[244] X.Z. Zhang, G.J. Liu, L.Y. Yan, Y.B. Zhao, R.F. Chang, L.P. Wu, Creating triploid germplasm via induced 2n pollen in *Capsicum annuum* L., J. Hortic. Sci. Biotechnol. 78 (2003) 84–88.
[245] J. Wang, D.L. Li, X.Y. Kang, Induction of unreduced megaspores with high temperature during megasporogenesis in *Populus*, Ann. For. Sci. 69 (2012) 59–67.
[246] M. Lu, P.D. Zhang, X.Y. Kang, Induction of 2n female gametes in *Populus adenopoda* Maxim by high temperature exposure during female gametophyte development, Breed. Sci. 63 (2013) 96–103.
[247] H.O. Lee, J.M. Davidson, R.J. Duronio, Endoreplication: polyploidy with purpose, Genes Dev. 23 (2009) 2461–2477.
[248] J. Loidl, Effects of elevated temperatures on meiotic chromosome synapsis, Chromosoma 97 (1989) 449–458.
[249] K. Church, D.E. Wimber, Meiosis in the grasshopper: chiasma frequency after elevated temperature and X-rays, Can. J. Genet. Cytol. 11 (1969) 209–216.
[250] J.D. Higgins, R.M. Perry, A. Barakate, L. Ramsay, R. Waugh, C. Halpin, S.J. Armstrong, F.C.H. Franklin, Spatiotemporal asymmetry of the meiotic program underlies the predominantly distal distribution of meiotic crossovers in barley, Plant Cell 24 (2012) 4096–4109.
[251] M.A. Janicke, L. Lasko, R. Oldenbourg, J.R. LaFountain, Chromosome malorientations after meiosis II arrest cause nondisjunction, Mol. Biol. Cell 18 (2007) 1645–1656.
[252] A. Ressayre, C. Raquin, A. Mignot, B. Godelle, P.H. Gouyon, Correlated variation in microtubule distribution, callose deposition during male post-meiotic cytokinesis, and pollen aperture number across *Nicotiana* species (solanaceae), Am. J. Bot. 89 (2002) 393–400.
[253] A. Yamada, R. Tao, A. Sugiura, Influence of low temperature before flowering on the occurrence of unreduced pollen in Japanese persimmon (*Diospyros kaki* Thunb.), Hortscience 40 (2005) 24–28.
[254] A.S. Mason, M.N. Nelson, G.J. Yan, W.A. Cowling, Production of viable male unreduced gametes in *Brassica* interspecific hybrids is genotype specific and stimulated by cold temperatures, BMC Plant Biol. 11 (2011).
[255] A. Stefani, N. Colonna, The influence of temperature on meiosis and microspore development in *Dasypyrum villosum* P. Candargy, Cytologia 61 (1996) 277–283.
[256] C. Xu, Z. Liu, L. Zhang, C. Zhao, S. Yuan, F. Zhang, Organization of actin cytoskeleton during meiosis I in a wheat thermo-sensitive genic male sterile line, Protoplasma (2012).
[257] Z.H. Tang, L.P. Zhang, D. Yang, C.P. Zhao, Y.L. Zheng, Cold stress contributes to aberrant cytokinesis during male meiosis I in a wheat thermosensitive genic male sterile line, Plant Cell Environ. 34 (2011) 389–405.
[258] S. Breton, D. Brown, Cold-induced microtubule disruption and relocalization of membrane proteins in kidney epithelial cells, J. Am. Soc. Nephrol. 9 (1998) 155–166.
[259] Q.Y. Wang, P. Nick, Cold acclimation can induce microtubular cold stability in a manner distinct from abscisic acid, Plant Cell Physiol. 42 (2001) 999–1005.
[260] A. Hochwagen, G. Wrobel, M. Cartron, P. Demougin, C. Niederhauser-Wiederkehr, M.G. Boselli, M. Primig, A. Amon, Novel response to microtubule perturbation in meiosis, Mol. Cell. Biol. 25 (2005) 4767–4781.
[261] K.O. Pope, K.H. Baines, A.C. Ocampo, B.A. Ivanov, Impact winter and the Cretaceous-Tertiary extinctions – results of a Chicxulub Asteroid Impact Model, Earth Planet. Sci. Lett. 128 (1994) 719–725.
[262] V. Vajda, J.I. Raine, C.J. Hollis, Indication of global deforestation at the Creataceous-Tertiary boundary by New Zealand fern spike, Science 294 (2001) 1700–1702.
[263] P. Wilf, K.R. Johnson, Land plant extinction at the end of the cretaceous: a quantitative analysis of the North Dakota megafloral record, Paleobiology 30 (2004) 347–368.
[264] P.C.J. Donoghue, M.A. Purnell, Genome duplication, extinction and vertebrate evolution, Trends Ecol. Evol. 20 (2005) 312–319.
[265] S.H. Xiao, M. Laflamme, On the eve of animal radiation: phylogeny, ecology and evolution of the Ediacara biota, Trends Ecol. Evol. 24 (2009) 31–40.
[266] D.A. Levin, Polyploidy and novelty in flowering plants, Am. Nat. 122 (1983) 1–25.
[267] J.D. Thompson, R. Lumaret, The evolutionary dynamics of polyploid plants – origins, establishment and persistence, Trends Ecol. Evol. 7 (1992) 302–307.
[268] D.E. Soltis, P.S. Soltis, The dynamic nature of polyploid genomes, Proc. Natl. Acad. Sci. U. S. A. 92 (1995) 8089–8091.
[269] M. Hegarty, S. Hiscock, Polyploidy: doubling up for evolutionary success, Curr. Biol. 17 (2007) R927–R929.
[270] Y. Van de Peer, S. Maere, A. Meyer, The evolutionary significance of ancient genome duplications, Nat. Rev. Genet. 10 (2009) 725–732 (Opinion).

[271] K.D. Crow, G.P. Wagner, What is the role of genome duplication in the evolution of complexity and diversity? Mol. Biol. Evol. 23 (2006) 887–892.
[272] S.P. Otto, The evolutionary consequences of polyploidy, Cell 131 (2007) 452–462.
[273] W.M. Kurschner, S.J. Batenburg, L. Mander, Aberrant Classopollis pollen reveals evidence for unreduced (2n) pollen in the conifer family Cheirolepidiaceae during the Triassic-Jurassic transition, Proc. R. Soc. B: Biol. Sci. 280 (2013).
[274] H. Visscher, C.V. Looy, M.E. Collinson, H. Brinkhuis, Cittert JHAVKV, W.M. Kurschner, M.A. Sephton, Environmental mutagenesis during the end-Permian ecological crisis, Proc. Natl. Acad. Sci. U. S. A. 101 (2004) 12952–12956.
[275] C.B. Foster, S.A. Afonin, Abnormal pollen grains: an outcome of deteriorating atmospheric conditions around the Permian – Triassic boundary, J. Geol. Soc. 162 (2005) 653–659.
[276] C. Brochmann, A.K. Brysting, I.G. Alsos, L. Borgen, H.H. Grundt, A.C. Scheen, R. Elven, Polyploidy in arctic plants, Biol. J. Linn. Soc. 82 (2004) 521–536.
[277] J. Ramsey, Polyploidy and ecological adaptation in wild yarrow, Proc. Natl. Acad. Sci. U. S. A. 108 (2011) 7096–7101.
[278] R.J. Abbott, C. Brochmann, History and evolution of the arctic flora: in the footsteps of Eric Hulten, Mol. Ecol. 12 (2003) 299–313.
[279] C. Parisod, R. Holderegger, C. Brochmann, Evolutionary consequences of autopolyploidy, N. Phytol. 186 (2010) 5–17.
[280] D.Y. Hashimoto-Freitas, N.M.A. Nassar, Cytogenetic and anatomic behavior of cytochimeras and total polyploids in cassava, Genet. Mol. Res. 12 (2013) 4879–4894.
[281] H.J.M. Bastiaanssen, P.M.M.M. van den Berg, P. Lindhout, E. Jacobsen, M.S. Ramanna, Postmeiotic restitution in 2n egg formation of diploid potato, Heredity 81 (1998) 20–27.
[282] D.T. Fox, R.J. Duronio, Endoreplication and polyploidy: insights into development and disease, Development 140 (2013) 3–12.
[283] J. Joubes, C. Chevalier, Endoreduplication in higher plants, Plant Mol. Biol. 43 (2000) 735–745.
[284] B.A. Larkins, B.P. Dilkes, R.A. Dante, C.M. Coelho, Y.M. Woo, Y. Liu, Investigating the hows and whys of DNA endoreduplication, J. Exp. Bot. 52 (2001) 183–192.
[285] B.A. Edgar, N. Zielke, C. Gutierrez, Endocycles: a recurrent evolutionary innovation for post-mitotic cell growth, Nat. Rev. Mol. Cell Biol. 15 (2014) 197–210.
[286] Z. Larson-Rabin, Z. Li, P.H. Masson, C.D. Day, FZR2/CCS52A1 expression is a determinant of endoreduplication and cell expansion in Arabidopsis, Plant Physiol. 149 (2009) 874–884.
[287] Z. Li, Z. Larson-Rabin, P.H. Masson, C.D. Day, FZR2/CCS52A1 mediated endoreduplication in Arabidopsis development, Plant Signal. Behav. 4 (2009) 451–453.
[288] F. D'Amato, Endopolyploidy as a factor in plant tissue development, Caryologia 17 (1964) 41–52.
[289] H. Weiss, J. Maluszynska, Molecular cytogenetic analysis of polyploidization in the anther tapetum of diploid and autotetraploid *Arabidopsis thaliana* plants, Ann. Bot. 87 (2001) 729–735.
[290] S.K. Sharma, S. Kumaria, P. Tandon, S.R. Rao, Endomitosis in tapetal cells of some Cymbidiums (Orchidaceae), Nucleus 55 (2012) 21–25.
[291] M. Weingartner, M.C. Criqui, T. Meszaros, P. Binarova, A.C. Schmit, A. Helfer, A. Derevier, M. Erhardt, L. Bogre, P. Genschik, Expression of a nondegradable cyclin B1 affects plant development and leads to endomitosis by inhibiting the formation of a phragmoplast, Plant Cell 16 (2004) 643–657.
[292] J.G. Torrey, Kinetin as trigger for mitosis in mature endomitotic plant cells, Exp. Cell Res. 23 (1961) 2p1–299p.
[293] G. Lavia, A. Fernandez, G. Marquez, Chromosome doubling in *Turnera ulmifolia* (Turneraceae) induced by regeneration of plants from in vitro cultured leaf explants, Plant Syst. Evol. 192 (1994) 41–48.
[294] Z.Q. Tang, D.L. Chen, Z.J. Song, Y.C. He, D.T. Cai, In vitro induction and identification of tetraploid plants of *Paulownia tomentosa*, Plant Cell Tissue Organ Cult. 102 (2010) 213–220.
[295] Z.Y. Yang, Z.X. Shen, H. Tetreault, L. Johnson, B. Friebe, T. Frazier, L.K. Huang, C. Burklew, X.Q. Zhang, B.Y. Zhao, Production of autopolyploid lowland switchgrass lines through in vitro chromosome doubling, Bioenergy Res. 7 (2014) 232–242.
[296] H. Dewar, K. Tanaka, K. Nasmyth, T.U. Tanaka, Tension between two kinetochores suffices for their bi-orientation on the mitotic spindle, Nature 428 (2004) 93–97.
[297] P.Y. Goh, J.V. Kilmartin, Ndc10 – a gene involved in chromosome segregation in Saccharomyces–Cerevisiae, J. Cell Biol. 121 (1993) 503–512.
[298] H.J. Chial, M. Winey, Mechanisms of genetic instability revealed by analysis of yeast spindle pole body duplication, Biol. Cell 91 (1999) 439–450.
[299] D. Dikovskaya, D. Schiffmann, I.P. Newton, A. Oakley, K. Kroboth, O. Sansom, T.J. Jamieson, V. Meniel, A. Clarke, I.S. Nathke, Loss of APC induces polyploidy as a result of a combination of defects in mitosis and apoptosis, J. Cell Biol. 176 (2007) 183–195.
[300] R. Henriques, Z. Magyar, A. Monardes, S. Khan, C. Zalejski, J. Orellana, L. Szabados, C. de la Torre, C. Koncz, L. Bogre, Arabidopsis S6 kinase mutants display chromosome instability and altered RBR1-E2F pathway activity, EMBO J. 29 (2010) 2979–2993.
[301] D.P.S. Verma, Cytokinesis and building of the cell plate in plants, Annu. Rev. Plant Physiol. Plant Mol. Biol. 52 (2001) 751–784.
[302] G. Jurgens, Cytokinesis in higher plants, Annu. Rev. Plant Biol. 56 (2005) 281–299.
[303] W. Lukowitz, U. Mayer, G. Jurgens, Cytokinesis in the Arabidopsis embryo involves the syntaxin-related KNOLLE gene product, Cell 84 (1996) 61–71.
[304] D.L. Klindworth, N.D. Williams, Characterization of a mitotic mutant of durum wheat, Chromosome Res. 9 (2001) 377–386.
[305] X.Y. Chen, L. Liu, E. Lee, X. Han, Y. Rim, H. Chu, S.W. Kim, F. Sack, J.Y. Kim, The Arabidopsis callose synthase gene GSL8 is required for cytokinesis and cell patterning, Plant Physiol. 150 (2009) 105–113.
[306] N. De Storme, J. De Schrijver, W. Van Criekinge, V. Wewer, P. Dormann, D. Geelen, GLUCAN SYNTHASE-LIKE8 and STEROL METHYLTRANSFERASE2 are required for ploidy consistency of the sexual reproduction system in Arabidopsis, Plant Cell 25 (2013) 387–403.
[307] N. De Storme, D. Geelen, Pre-meiotic endomitosis in the cytokinesis-defective tomato mutant pmcd1 generates tetraploid meiocytes and diploid gametes, J. Exp. Bot. 64 (2013) 2345–2358.
[308] F.F. Assaad, U. Mayer, G. Wanner, G. Jurgens, The KEULE gene is involved in cytokinesis in Arabidopsis, Mol. Gen. Genet. 253 (1996) 267–277.
[309] R. Sollner, G. Glasser, G. Wanner, C.R. Somerville, G. Jurgens, F.F. Assaad, Cytokinesis-defective mutants of Arabidopsis, Plant Physiol. 129 (2002) 678–690.
[310] E. Falistocco, N. Tosti, M. Falcinelli, Cytomixis in pollen mother cells of diploid Dactylis, one of the origins of 2n gametes, J. Hered. 86 (1995) 448–453.
[311] S.K. Lattoo, S. Khan, S. Bamotra, A.K. Dhar, Cytomixis impairs meiosis and influences reproductive success-in *Chlorophytum comosum* (Thunb) Jacq. – an additional strategy and possible implications, J. Biosci. 31 (2006) 629–637.
[312] V.K. Singhal, P. Kumar, Impact of cytomixis on meiosis, pollen viability and pollen size in wild populations of *Himalayan poppy* (*Meconopsis aculeata* Royle), J. Biosci. 33 (2008) 371–380.
[313] P. Kumar, V.K. Singhal, D. Kaur, S. Kaur, Cytomixis and associated meiotic abnormalities affecting pollen fertility in *Clematis orientalis*, Biol. Plant. 54 (2010) 181–184.
[314] V.K. Singhal, P.K. Rana, P. Kumar, D. Kaur, Persistent occurrence of meiotic abnormalities in a new hexaploid cytotype of *Thalictrum foetidum* from Indian cold deserts, Biologia (Bratisl.) 66 (2011) 458–464.
[315] J.Z. Guan, J.J. Wang, Z.H. Cheng, Y. Liu, Z.Y. Li, Cytomixis and meiotic abnormalities during microsporogenesis are responsible for male sterility and chromosome variations in *Houttuynia cordata*, Genet. Mol. Res. 11 (2012) 121–130.
[316] S.M. Ghaffari, Occurrence of diploid and polyploidy microspores in *Sorghum bicolor* (Poaceae) is the result of cytomixis, Afr. J. Biotechnol. 5 (2006) 1450–1453.
[317] G. Kumar, S. Naseem, Gamma-induced infraspecific polyploidy via cytomixis-mediated syncyte formation in *Papaver somniferum* (Papaveraceae), Turk. J. Bot. 37 (2013) 530–536.
[318] J.S. Kim, K. Oginuma, H. Tobe, Syncyte formation in the microsporangium of Chrysanthemum (Asteraceae): a pathway to infraspecific polyploidy, J. Plant Res. 122 (2009) 439–444.
[319] Y.V. Sidorchuk, E.V. Deineko, V.K. Shumny, Peculiarities of cytomixis in pollen mother cells of transgenic tobacco plants (*Nicotiana tabacum* L.) with mutant phenotype, Cell Tissue Biol. 1 (2007) 570–576.
[320] Z.Q. Song, X.F. Li, Cytomixis in pollen mother cells of *Salvia miltiorrhiza*, Caryologia 62 (2009) 213–219.
[321] D. Kaur, V.K. Singhal, Phenomenon of cytomixis and intraspecific polyploidy (2x, 4x) in *Spergularia diandra* (Guss.) Heldr. & Sart. in the cold desert regions of Kinnaur District (Himachal Pradesh), Cytologia (Tokyo) 77 (2012) 163–171.
[322] S.R. Mursalimov, Y.V. Sidorchuk, E.V. Deineko, New insights into cytomixis: specific cellular features and prevalence in higher plants, Planta 238 (2013) 415–423.
[323] A. Levan, Syncyte formation in the pollen mother cells of haploid *Phleum pratense*, Hereditas 27 (1941) 243–252.
[324] T. Lelley, A.A. Mahmoud, V. Lein, Genetics and cytology of unreduced gametes in cultivated rye (Secale-Cereale L.), Genome 29 (1987) 635–638.
[325] M. Sheidai, A.M. Saeed, B. Zehzad, Meiotic studies of some *Aegilops* (Poaceae) species and populations in Iran, Edinburgh J. Bot. 56 (1999) 405–419.
[326] M. Bellucci, C. Roscini, A. Mariani, Cytomixis in pollen mother cells of *Medicago sativa* L., J. Hered. 94 (2003) 512–516.
[327] M. Sheidai, P. Koobaz, B. Zehzad, Meiotic studies of some *Avena* species and populations, J. Sci. I. R. Iran 14 (2003) 121–131.
[328] M. Sheidai, E.S. Bagheri-Shabestarei, Cytomixis and unreduced pollen formation in some *Festuca* L. species of Iran, Caryologia 60 (2007) 364–371.
[329] G.A. Malallah, T.A. Attia, Cytomixis and its possible evolutionary role in a Kuwaiti population of *Diplotaxis harra* (Brassicaceae), Bot. J. Linn. Soc. 143 (2003) 169–175.
[330] G. Kumar, R. Tripathi, Induced cytomictic variations through abiotic stresses in grasspea (*Lathyrus sativus* L.), Indian J. Genet. Plant Breed. 68 (2008) 58–64.
[331] N.C. Merwine, H.W. Bennett, Syncytes in meiosis of polyploid sorghum, Crop Sci. 6 (1966) 155–157.
[332] V.K. Singhal, P.K. Rana, P. Kumar, Syncytes during male meiosis resulting in 2n pollen grain formation in Lindelofia longiflora var. falconeri, J. Syst. Evol. 49 (2011) 406–410.
[333] J. Ramsey, D.W. Schemske, Neopolyploidy in flowering plants, Annu. Rev. Ecol. Syst. 33 (2002) 589–639.
[334] I. Schubert, M.A. Lysak, Interpretation of karyotype evolution should consider chromosome structural constraints, Trends Genet. 27 (2011) 207–216.
[335] P.V. Jallepalli, D. Pellman, Aneuploidy in the balance, Science 317 (2007) 904–905.

[336] E.M. Torres, T. Sokolsky, C.M. Tucker, L.Y. Chan, M. Boselli, M.J. Dunham, A. Amon, Effects of aneuploidy on cellular physiology and cell division in haploid yeast, Science 317 (2007) 916–924.
[337] I.M. Henry, B.P. Dilkes, E.S. Miller, D. Burkart-Waco, L. Comai, Phenotypic consequences of aneuploidy in *Arabidopsis thaliana*, Genetics 186 (2010) 1231–1245.
[338] M. Ravi, F. Shibata, J.S. Ramahi, K. Nagaki, C.B. Chen, M. Murata, S.W.L. Chan, Meiosis-specific loading of the centromere-specific histone CENH3 in *Arabidopsis thaliana*, PLoS Genet. 7 (2011).
[339] T. Chiang, R.M. Schultz, M.A. Lampson, Meiotic origins of maternal age-related aneuploidy, Biol. Reprod. 86 (2012).
[340] C.L. Tiang, Y. He, W.P. Pawlowski, Chromosome organization and dynamics during interphase, mitosis, and meiosis in plants, Plant Physiol. 158 (2012) 26–34.
[341] M. Cifuentes, L. Grandont, G. Moore, A.M. Chèvre, E. Jenczewski, Genetic regulation of meiosis in polyploid species: new insights into an old question, N. Phytol. 186 (2010) 29–36.
[342] S.D. Nicolas, M. Leflon, Z. Liu, F. Eber, L. Chelysheva, O. Coriton, A.M. Chèvre, E. Jenczewski, Chromosome 'speed dating' during meiosis of polyploid *Brassica* hybrids and haploids, Cytogenet. Genome Res. 120 (2008) 331–338.
[343] A.S. Mason, J. Batley, P.E. Bayer, A. Hayward, W.A. Cowling, M.N. Nelson, High-resolution molecular karyotyping uncovers pairing between ancestrally related *Brassica* chromosomes, N. Phytol. 202 (2014) 964–974.
[344] A.S. Mason, V. Huteau, F. Eber, O. Coriton, G. Yan, M.N. Nelson, W.A. Cowling, A.-M. Chèvre, Genome structure affects the rate of autosyndesis and allosyndesis in AABC, BBAC and CCAB Brassica interspecific hybrids, Chromosome Res. 18 (2010) 655–666.
[345] A.S. Mason, M.N. Nelson, J. Takahira, W.A. Cowling, G. Moreira Alves, A. Chaudhuri, N. Chen, M.E. Ragu, J. Dalton-Morgan, O. Coriton, V. Huteau, F. Eber, A.-M. Chèvre, J. Batley, The fate of chromosomes and alleles in an allohexaploid *Brassica* population, Genetics 197 (2014) 273–283.
[346] L. Suay, D. Zhang, F. Eber, H. Jouy, M. Lodé, V. Huteau, O. Coriton, E. Szadkowski, M. Leflon, O.C. Martin, M. Falque, E. Jenczewski, S. Paillard, A.-M. Chèvre, Crossover rate between homologous chromosomes and interference are regulated by the addition of specific unpaired chromosomes in *Brassica*, N. Phytol. 201 (2014) 645–656.
[347] E. Jenczewski, F. Eber, A. Grimaud, S. Huet, M.O. Lucas, H. Monod, A.M. Chèvre, *PrBn*, a major gene controlling homeologous pairing in oilseed rape (*Brassica napus*) haploids, Genetics 164 (2003) 645–653.
[348] Z. Liu, K. Adamczyk, M. Manzanares-Dauleux, F. Eber, M.-O. Lucas, R. Delourme, A.M. Chèvre, E. Jenczewski, Mapping *PrBn* and other quantitative trait loci responsible for the control of homeologous chromosome pairing in oilseed rape (*Brassica napus* L.) haploids, Genetics 174 (2006) 1583–1596.
[349] S.D. Nicolas, M. Leflon, H. Monod, F. Eber, O. Coriton, V. Huteau, A.M. Chèvre, E. Jenczewski, Genetic regulation of meiotic cross-overs between related genomes in *Brassica napus* haploids and hybrids, Plant Cell 21 (2009) 373–385.
[350] L. Grandont, N. Cuñado, O. Coriton, V. Huteau, F. Eber, A.M. Chèvre, M. Grelon, L. Chelysheva, E. Jenczewski, Homoeologous chromosome sorting and progression of meiotic recombination in *Brassica napus*: ploidy does matter!, Plant Cell 26 (4) (2014) 1448–1463, http://dx.doi.org/10.1105/tpc.114.122788.
[351] S.W.L. Chan, In a battle between parental chromosomes, a failure to reload, Proc. Natl. Acad. Sci. U. S. A. 108 (2011) 13361–13362.
[352] D. Gernand, T. Rutten, A. Varshney, M. Rubtsova, S. Prodanovic, C. Bruss, J. Kumlehn, F. Matzk, A. Houben, Uniparental chromosome elimination at mitosis and interphase in wheat and pearl millet crosses involves micronucleus formation, progressive heterochromatinization, and DNA fragmentation, Plant Cell 17 (2005) 2431–2438.
[353] D.A. Laurie, M.D. Bennett, The timing of chromosome elimination in hexaploid wheat x maize crosses, Genome 32 (1989) 953–961.
[354] M.D. Bennett, R.A. Finch, I.R. Barclay, Time rate and mechanism of chromosome elimination in *Hordeum* hybrids, Chromosoma 54 (1976) 175–200.
[355] S. Henikoff, Y. Dalal, Centromeric chromatin: what makes it unique? Curr. Opin. Genet. Dev. 15 (2005) 177–184.
[356] M. Sanei, R. Pickering, K. Kumke, S. Nasuda, A. Houben, Loss of centromeric histone H3 (CENH3) from centromeres precedes uniparental chromosome elimination in interspecific barley hybrids, Proc. Natl. Acad. Sci. U. S. A. 108 (2011) E498–E505.
[357] K.M. Wright, J.C. Pires, A. Madlung, Mitotic instability in resynthesized and natural polyploids of the genus *Arabidopsis* (Brassicaceae), Am. J. Bot. 96 (2009) 1656–1664.
[358] A.M.A. Wolters, H.C.H. Schoenmakers, S. Kamstra, J. Vaneden, M. Koornneef, J.H. Dejong, Mitotic and meiotic irregularities in somatic hybrids of *Lycopersicon esculentum* and *Solanum tuberosum*, Genome 37 (1994) 726–735.
[359] C.B.S.R. Sharma, Chemically induced aneuploidy in higher plants, Mutagenesis 5 (1990) 105–125.
[360] F. Damato, Cytogenetics of plant cell and tissue cultures and their regenerates, Crit. Rev. Plant Sci. 3 (1985) 73–112.
[361] M.A. Ferguson-Smith, V. Trifonov, Mammalian karyotype evolution, Nat. Rev. Genet. 8 (2007) 950–962.
[362] M.A. Lysak, A. Berr, A. Pecinka, R. Schmidt, K. McBreen, I. Schubert, Mechanisms of chromosome number reduction in *Arabidopsis thaliana* and related Brassicaceae species, Proc. Natl. Acad. Sci. U. S. A. 103 (2006) 5224–5229.
[363] L.M. Yang, D.H. Koo, D.W. Li, T. Zhang, J.M. Jiang, F.S. Luan, S.S. Renner, E. Henaff, W. Sanseverino, J. Garcia-Mas, J. Casacuberta, D.A. Senalik, P.W. Simon, J.F. Chen, Y.Q. Weng, Next-generation sequencing, FISH mapping and synteny-based modeling reveal mechanisms of decreasing dysploidy in *Cucumis*, Plant J. 77 (2014) 16–30.
[364] F. Cheng, T. Mandakova, J. Wu, Q. Xie, M.A. Lysak, X.W. Wang, Deciphering the diploid ancestral genome of the mesohexaploid *Brassica rapa*, Plant Cell 25 (2013) 1541–1554.
[365] M.N. Nelson, I.A.P. Parkin, D.J. Lydiate, The mosaic of ancestral karyotype blocks in the *Sinapis alba* L. genome, Genome 54 (2011) 33–41.
[366] F. Murat, J.H. Xu, E. Tannier, M. Abrouk, N. Guilhot, C. Pont, J. Messing, J. Salse, Ancestral grass karyotype reconstruction unravels new mechanisms of genome shuffling as a source of plant evolution, Genome Res. 20 (2010) 1545–1557.
[367] R. Faria, A. Navarro, Chromosomal speciation revisited: rearranging theory with pieces of evidence, Trends Ecol. Evol. 25 (2010) 660–669.
[368] F.J. Ayala, M. Coluzzi, Chromosome speciation: humans, Drosophila, and mosquitoes, Proc. Natl. Acad. Sci. U. S. A. 102 (2005) 6535–6542.
[369] M. Kirkpatrick, N. Barton, Chromosome inversions, local adaptation and speciation, Genetics 173 (2006) 419–434.
[370] S.B. Yakimowski, L.H. Rieseberg, The role of homoploid hybridization in evolution: a century of studies synthesizing genetics and ecology, Am. J. Bot. 101 (2014) 1247–1258.
[371] M. Kirkpatrick, A. Kern, Where's the money? Inversions, genes, and the hunt for genomic targets of selection, Genetics 190 (2012) 1153–1155.
[372] R.F. Guerrero, F. Rousset, M. Kirkpatrick, Coalescent patterns for chromosomal inversions in divergent populations, Philos. Trans. R. Soc. B: Biol. Sci. 367 (2012) 430–438.
[373] M. Radman, R. Wagner, Mismatch recognition in chromosomal interactions and speciation, Chromosoma 102 (1993) 369–373.
[374] K. Jones, Robertsonian fusion and centric fission in karyotype evolution of higher plants, Bot. Rev. 64 (1998) 273–289.
[375] J.G. Barb, J.E. Bowers, S. Renaut, J.I. Rey, S.J. Knapp, L.H. Rieseberg, J.M. Burke, Chromosomal evolution and patterns of introgression in *Helianthus*, Genetics 197 (2014) 969–979.
[376] J.L. Strasburg, C. Scotti-Saintagne, I. Scotti, Z. Lai, L.H. Rieseberg, Genomic patterns of adaptive divergence between chromosomally differentiated sunflower species, Mol. Biol. Evol. 26 (2009) 1341–1355.
[377] Z. Lai, T. Nakazato, M. Salmaso, J.M. Burke, S.X. Tang, S.J. Knapp, L.H. Rieseberg, Extensive chromosomal repatterning and the evolution of sterility barriers in hybrid sunflower species, Genetics 171 (2005) 291–303.
[378] I. Lipnerova, P. Bures, L. Horova, P. Smarda, Evolution of genome size in Carex (Cyperaceae) in relation to chromosome number and genomic base composition, Ann. Bot. 111 (2013) 79–94.
[379] M. Escudero, J.A. Weber, A.L. Hipp, Species coherence in the face of karyotype diversification in holocentric organisms: the case of a cytogenetically variable sedge (*Carex scoparia*, Cyperaceae), Ann. Bot. 112 (2013) 515–526.
[380] T.C. Osborn, D.V. Butrulle, A.G. Sharpe, K.J. Pickering, I.A.P. Parkin, J.S. Parker, D.J. Lydiate, Detection and effects of a homeologous reciprocal transposition in *Brassica napus*, Genetics 165 (2003) 1569–1577.
[381] M. Guerra, Chromosome numbers in plant cytotaxonomy: concepts and implications, Cytogenet. Genome Res. 120 (2008) 339–350.
[382] M. Guo, D. Davis, J.A. Birchler, Dosage effects on gene expression in a maize ploidy series, Genetics 142 (1996) 1349–1355.
[383] J.M. Sheltzer, A. Amon, The aneuploidy paradox: costs and benefits of an incorrect karyotype, Trends Genet. 27 (2011) 446–453.
[384] T.R. Hughes, C.J. Roberts, H.Y. Dai, A.R. Jones, M.R. Meyer, D. Slade, J. Burchard, S. Dow, T.R. Ward, M.J. Kidd, S.H. Friend, M.J. Marton, Widespread aneuploidy revealed by DNA microarray expression profiling, Nat. Genet. 25 (2000) 333–337.
[385] B.G. Murray, A.G. Young, Widespread chromosome variation in the endangered grassland forb *Rutidosis leptorrhynchoides* F. Muell. (Asteraceae: Gnaphalieae), Ann. Bot. 87 (2001) 83–90.
[386] M.J. Considine, Y.Z. Wan, M.F. D'Antuono, Q. Zhou, M.Y. Han, H. Gao, M. Wang, Molecular genetic features of polyploidization and aneuploidization reveal unique patterns for genome duplication in diploid malus, PLoS One 7 (2012) 57–65.
[387] A. Krahulcova, F. Krahulec, H.M. Chapman, Variation in *Hieracium* subgen. Pilosella (Asteraceae): what do we know about its sources? Folia Geobot. 35 (2000) 319–338.
[388] T. Mandakova, A. Kovarik, J. Zozomova-Lihova, R. Shimizu-Inatsugi, K.K. Shimizu, K. Mummenhoff, K. Marhold, M.A. Lysak, The more the merrier: recent hybridization and polyploidy in *Cardamine*, Plant Cell 25 (2013) 3280–3295.
[389] M. Chester, M.J. Lipman, J.P. Gallagher, P.S. Soltis, D.E. Soltis, An assessment of karyotype restructuring in the neoallotetraploid *Tragopogon miscellus* (Asteraceae), Chromosome Res. 21 (2013) 75–85.
[390] F.K. Yousafzai, N. Al-Kaff, G. Moore, The molecular features of chromosome pairing at meiosis: the polyploid challenge using wheat as a reference, Funct. Integr. Genomics 10 (2010) 147–156.
[391] S.L. Krebs, J.F. Hancock, Tetrasomic inheritance of isoenzyme markers in the highbush blueberry, *Vaccinium corymbosum* L., Heredity 63 (1989) 11–18.
[392] R.G. Fjellstrom, P.R. Beuselinck, J.J. Steiner, RFLP marker analysis supports tetrasonic (sic) inheritance in *Lotus corniculatus* L., Theor. Appl. Genet. 102 (2001) 718–725.
[393] P.G. Wolf, P.S. Soltis, D.E. Soltis, Tetrasomic inheritance and chromosome-pairing behavior in the naturally-occurring autotetraploid *Heuchera grossulariifolia* (Saxifragaceae), Genome 32 (1989) 655–659.

[394] F. Pupilli, M.E. Caceres, C.L. Quarin, S. Arcioni, Segregation analysis of RFLP markers reveals a tetrasomic inheritance in apomictic *Paspalum simplex*, Genome 40 (1997) 822–828.
[395] A.O. Mendiburu, S.J. Peloquin, Significance of 2n gametes in potato breeding, Theor. Appl. Genet. 49 (1977) 53–61.
[396] M. Stift, C. Berenos, P. Kuperus, P.H. van Tienderen, Segregation models for disomic, tetrasomic and intermediate inheritance in tetraploids: a general procedure applied to *Rorippa* (Yellow cress) microsatellite data, Genetics 179 (2008) 2113–2123.
[397] N. Jones, A. Houben, B chromosomes in plants: escapees from the A chromosome genome? Trends Plant Sci. 8 (2003) 417–423.
[398] R.N. Jones, W. Viegas, A. Houben, A century of b chromosomes in plants: so what? Ann. Bot. 101 (2008) 767–775.
[399] D. Charlesworth, B. Charlesworth, G. Marais, Steps in the evolution of heteromorphic sex chromosomes, Heredity 95 (2005) 118–128.
[400] S.F. Covert, Supernumerary chromosomes in filamentous fungi, Curr. Genet. 33 (1998) 311–319.
[401] J.S. Lai, R.Q. Li, X. Xu, W.W. Jin, M.L. Xu, H.N. Zhao, Z.K. Xiang, W.B. Song, K. Ying, M. Zhang, Y.P. Jiao, P.X. Ni, J.G. Zhang, D. Li, X.S. Guo, K.X. Ye, M. Jian, B. Wang, H.S. Zheng, H.Q. Liang, X.Q. Zhang, S.C. Wang, S.J. Chen, J.S. Li, Y. Fu, N.M. Springer, H.M. Yang, J.A. Wang, J.R. Dai, P.S. Schnable, J. Wang, Genome-wide patterns of genetic variation among elite maize inbred lines, Nat. Genet. 42 (2010) 1027–1030.
[402] J.X. Ma, J.L. Bennetzen, Rapid recent growth and divergence of rice nuclear genomes, Proc. Natl. Acad. Sci. U. S. A. 101 (2004) 12404–12410.
[403] D. Schuermann, J. Molinier, O. Fritsch, B. Hohn, The dual nature of homologous recombination in plants, Trends Genet. 21 (2005) 172–181.
[404] A.B. Britt, DNA damage and repair in plants, Annu. Rev. Plant Physiol. Plant Mol. Biol. 47 (1996) 75–100.
[405] A. Boyko, J. Filkowski, I. Kovalchuk, Homologous recombination in plants is temperature and day-length dependent, Mutat. Res. 572 (2005) 73–83.
[406] D.E. Soltis, M.C. Segovia-Salcedo, I. Jordon-Thaden, L. Majure, N.M. Miles, E.V. Mavrodiev, W. Mei, M.B. Cortez, P.S. Soltis, M.A. Gitzendanner, Are polyploids really evolutionary dead-ends (again)? A critical reappraisal of Mayrose et al., N. Phytol. 202 (2014) 1105–1117.
[407] D.A. Levin, The timetable for allopolyploidy in flowering plants, Ann. Bot. 112 (2013) 1201–1208.
[408] N.J. Larkan, D.J. Lydiate, I.A.P. Parkin, M.N. Nelson, D.J. Epp, W.A. Cowling, S.R. Rimmer, M.H. Borhan, The *Brassica napus* blackleg resistance gene *LepR3* encodes a receptor-like protein triggered by the *Leptosphaeria maculans* effector AVRLM1, N. Phytol. 197 (2013) 595–605.
[409] R.J.A. Buggs, S. Renny-Byfield, M. Chester, I.E. Jordon-Thaden, L.F. Viccini, S. Chamala, A.R. Leitch, P.S. Schnable, W.B. Barbazuk, P.S. Soltis, D.E. Soltis, Next-generation sequencing and genome evolution in allopolyploids, Am. J. Bot. 99 (2012) 372–382.
[410] A.H. Lloyd, M. Ranoux, S. Vautrin, N. Glover, J. Fourment, D. Charif, F. Choulet, G. Lassalle, W. Marande, J. Tran, F. Granier, L. Pingault, A. Remay, C. Marquis, H. Belcram, B. Chalhoub, C. Feuillet, H. Bergès, P. Sourdille, E. Jenczewski, Meiotic gene evolution: can you teach a new dog new tricks? Mol. Biol. Evol. (2014), http://dx.doi.org/10.1093/molbev/msu1119 (in press).