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The role of mitochondria in plant development and stress tolerance

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Abstract

Eukaryotic cells require orchestrated communication between nuclear and organellar genomes, perturbations in which are linked to stress response and disease in both animals and plants. In addition to mitochondria, which are found across eukaryotes, plant cells contain a second organelle, the plastid. Signaling both among the organelles (cytoplasmic) and between the cytoplasm and the nucleus (i.e. nuclear-cytoplasmic interactions (NCI)) is essential for proper cellular function. A deeper understanding of NCI and its impact on development, stress response, and long-term health is needed in both animal and plant systems. Here we focus on the role of plant mitochondria in development and stress response. We compare and contrast features of plant and animal

mitochondrial genomes (mtDNA), particularly highlighting the large and highly dynamic nature of plant mtDNA. Plant-based tools are powerful yet underutilized resources for enhancing our fundamental understanding of NCI. These tools also have great potential for improving crop production. Across taxa, mitochondria are most abundant in cells that have high energy or nutrient demands including at key developmental time points. Although plant mitochondria act as integrators of signals involved in both development and stress response pathways, little is known about plant mtDNA diversity and its impact on these processes. In humans, there are strong correlations between particular mitotypes (and mtDNA mutations) and developmental differences (or disease). We propose that future work in plants should focus on defining mitotypes more carefully and investigating their functional implications as well as improving techniques to facilitate this research.

Abbreviations

Cytoplasmic male sterility (CMS), non-chromosomal stripe (NCS), reactive oxygen species (ROS), oxidative phosphorylation (OXPHOS), alternative oxidase (AOX), mitochondria electron transport chain (mETC), pulsed-field gel electrophoresis (PFGE), mitochondrial DNA (mtDNA), nuclear-cytoplasmic interaction (NCI)

Keywords

Plant organelle, chondriome, mitochondrial genome, nuclear-cytoplasmic interaction, alloplasmic, cytoplasmic male sterility, stress, development

1 Introduction

Proper cellular function requires orchestrated communication among cellular compartments and the ability of the cell to sense and respond to its environment. Eukaryotic cells are distinguished from prokaryotes by compartmentalization of the nuclear genome and the presence of organelles that maintain their own genomes. Organelles are essential for energy production, nutrient sensing, metabolism, and stress response. Orchestrated signaling among organelles and between the organelles and the nucleus (in both directions) impacts global gene expression and drives cellular functions. Therefore, organelles, especially mitochondria, are often referred to as “signaling hubs”

within the cell. In this review, we focus on the role of plant mitochondria in development and stress response. We highlight the unique features of plant mitochondria and the dynamics of these specialized organelles and their genomes throughout the cell cycle, development, and under stressful conditions.

1.1 Endosymbiotic origin of organelles

Although historically debated, the endosymbiotic theory, which states that mitochondria and plastids were once free-living alphaproteobacteria and cyanobacteria, respectively, is now widely accepted (reviewed in (ARCHIBALD 2015)). The exact origins of each organelle and the mechanism by which eukaryotic cells formed and evolved is still an active area of research with multiple competing theories (reviewed in (MARTIN *et al.* 2015; ARCHIBALD 2015)). However, it is widely accepted that mitochondria were acquired first, followed by a second endosymbiosis event to establish plastids (reviewed in (GREEN 2011)). As a result of endosymbiosis, plant cells contain three distinct genetic “compartments”: the nucleus, mitochondria, and plastids (with chloroplasts being the most prominent specialized representatives, acting in photosynthesis). While most of the cellular genetic content is housed in the nuclear genome, mitochondria and plastids have retained independent genomes. However, only a fraction of their original genetic content (core sets of essential genes involved in oxidative phosphorylation and photosynthesis) remains within the organelles – a majority was transferred from the founding mitochondrial and plastid genomes to the nucleus. There is even evidence for genetic transfer between the chloroplast and mitochondrial genomes, and the process of gene transfer and organellar evolution is ongoing (reviewed in (PALMER *et al.* 2000)).

1.2 Plastome size, structure, and genetic content

Although subsequent sections of this article will focus primarily on plant mitochondria, we would be remiss to ignore the chloroplast. The plastid genome (plastome) size and gene content is relatively conserved across land plants (reviewed in (GREEN 2011)). On average, the plastome size is between ~100-200 kb with few exceptions above or below that range. Land plants have an even narrower window with analyzed species falling in the ~120-160 kb range. In the genomes analyzed thus far, most contain between 90-100

genes, and include a relatively invariable number of tRNA and protein coding genes. The differences in gene content across taxa comes predominantly from copy number variation (CNV) of the rRNA genes, which are encoded within the inverted repeats and therefore copied a different number of times depending on species. The core set of protein coding genes includes essential components for photosynthesis-related complexes and pathways such as photosystem I & II, the cytochrome complex, and ATP synthesis pathway. Historically, the plastid genome was thought to assemble as a single circle with one set of inverted repeats. However, recent studies in maize and other monocots (OLDENBURG and BENDICH 2015), as well as photosynthetic protists such as *Chromera velia* (JANOUSKOVEC *et al.* 2013), have uncovered evidence for both linear structures, and multiple rings or mini-circles as in some dinoflagellates (ZHANG *et al.* 1999). While there has been a recent influx of available whole organellar genome sequences, particularly mitochondrial sequences, information on protist organellar genomes is lagging behind other taxa and could use more attention (reviewed in (SMITH 2015)), especially given that the aforementioned examples suggest a more complicated view of plastid genome biology.

1.3 Plant mitochondrial genome size, structure, and genetic content

In contrast to the described plant plastid genomes and animal mitochondrial genomes, which are highly conserved across taxa and for the most part maintain a conservative size (<20 kb on a single circular chromosome), plants have extremely variable mitochondrial genome sizes and structures (SMITH and KEELING 2015). Of the species analyzed thus far, plant mitochondrial genomes range from 200 kb, such as in *Brassica napus* (HANDA 2003), to >10 Mb, such as in some recently analyzed *Silene* species (WU *et al.* 2015a). The gene content is also variable, with around 25-40 protein encoding genes, including a core set of genes involved in oxidative phosphorylation (OXPHOS), and a more than ten-fold range in the number of tRNA genes from 2 tRNAs in *Silene conica* to 26 tRNAs in *Cucurbita pepo* (SLOAN *et al.* 2012). Of course, functional plant mitochondria need far more than those core mitochondria-encoded genes. For instance, around 800 proteins have been identified in Arabidopsis mitochondria (KRUFT *et al.* 2001), most of them encoded in the nucleus as a result of still ongoing integration events of mitochondrial

DNA or RNA into the nuclear genome (reviewed in (HENZE and MARTIN 2001)). Although there are now functional mitochondrial genes encoded in the nucleus, there are also mtDNA integration sites in the nucleus that have not acquired the necessary regulatory elements (i.e. promoter elements and mitochondrial targeting sequences) and are decaying (HUANG *et al.* 2005). Two further points are of particular interest regarding mtDNA and mitochondrial genes in the plant nucleus: (i) the amount and sites of nuclear integrated mtDNA can vary even among different inbred lines of the same species, as for example reported in maize (LOUGH *et al.* 2008), and (ii) in some plants more than half of the nuclear-encoded genes with mitochondrial functions are PPR (Pentatricopeptide repeat) proteins which are involved in modulating mitochondrial RNA expression (reviewed in (SAHA *et al.* 2007)).

In both plants and animals, mitochondrial genomes have been canonically mapped as single “master circle” structures (reviewed in (SLOAN 2013)). However, nearly two decades ago, pulsed-field gel electrophoresis (PFGE) studies demonstrated that although some fungal mitochondrial genomes map as circular, linear chromosomes predominate (JACOBS *et al.* 1996; GERHOLD *et al.* 2010). Assembling plant mitochondrial genomes according to the “master circle” model has prevailed until recently, but accumulating evidence supports a much more complex view of these molecules (reviewed in (SLOAN 2013)). Plant mitochondrial genomes are considered highly “multipartite” due to the extensive number of inverted and direct repeats, the former of which theoretically allows for major recombination events leading to structural rearrangements. While many genomes can often be assembled to a “master circle,” the *in vivo* structures remain somewhat mysterious - sub-genomic rings and more complex looped, linear, and branched structures are predicted and have been experimentally suggested by electron microscopy (BAILEY-SERRES *et al.* 1987) and PFGE (BENDICH 1996; JACOBS *et al.* 1996). In addition, several species have now been shown to carry multi-chromosome mitochondrial genomes. These genomes do not share repeats that allow them to be assembled into a single chromosome, much less a master circle. For example, cucumber (*Cucumis sativus*) has three circular chromosomes, including a large 1.6 Mb chromosome and two smaller circles (ALVERSON *et al.* 2011). Even more extreme cases are found in

Silene species such as *Silene noctiflora*, which has a large >7 Mb genome that is subdivided into more than 50 circular chromosomes (WU *et al.* 2015b).

2 Investigating the mitochondrial contribution to development and stress response

2.1 Utilizing plants as a model to investigate nuclear-cytoplasmic interactions (NCI)

Throughout evolutionary history, most of the mitochondrial and plastid genetic content was transferred to the nuclear genome. Therefore, cellular functions now rely on thousands of nuclear-encoded proteins to be properly synthesized in the cytosol and then targeted to and imported by the appropriate organelles (reviewed in (MURCHA *et al.* 2014)). Because of the unique relationship among the organelles and the nucleus, the three genomes have undergone co-evolution to remain compatible. Beside the co-evolved matching genomes, proper communication between the nucleus and organelles and between the two organelles, especially coordinated gene expression, is essential for cellular functions (reviewed in (WOODSON and CHORY 2008)). Plants are well poised yet underutilized for answering questions related to NCI and the role of mitochondria in development and stress response.

Over time, both nuclear- and mitochondrial-encoded genes for mitochondrial proteins have evolved, and divergent sequences can be categorized into different haplogroups. In humans, the mitochondria and nuclear genomes are thought to have co-evolved under selective pressure to adapt to particular environmental niches, particularly in response to thermal stress, resulting in different mitochondrial haplogroups or “mitotypes” (reviewed in (WALLACE 2005)). This hypothesis is supported based on the distribution of distinct mitotypes worldwide, which mirrors human migration out of Africa to distinct climatic zones. Interestingly, the evolutionary benefit of having particular mitotypes is now challenged by modern day stressors such as disease pressures (reviewed in (WALLACE 2005)). Correlations have been made between having a particular mitotype and disease progression. For instance, having haplogroup B2 is correlated with an enhanced risk of developing cervical cancer if exposed to HPV (GUARDADO-ESTRADA *et al.* 2012). On the other hand, having haplogroup H, which has higher ATP and reactive oxygen species (ROS) production than other European haplogroups, is correlated with a slower

187 progression of HIV to AIDS in infected patients (GUZMÁN-FULGENCIO *et al.* 2013).

188
189 Of course, the nuclear genome content cannot be controlled for or fixed in human
190 experiments. Therefore, correlations between mitotypes and phenotypes can be made, but
191 assigning functional relationships between specific nuclear and mitochondrial gene
192 combinations in the context of abundant nuclear genome polymorphisms remains
193 challenging. To avoid the confounding influence of nuclear genome differences, cybrid
194 (cytoplasm hybrid) cell lines can be used. In mammalian systems, NCI manipulation
195 significantly impacts cellular respiration and stress response. For instance, mouse cybrid
196 cell lines carrying different mtDNA types show differences in OXPHOS performance
197 and ROS levels (MORENO-LOSHUERTOS *et al.* 2006). By using human cybrid cell lines, it
198 was also demonstrated that haplogroups have different basal levels of OXPHOS function
199 which may explain why some haplogroups have higher prevalence of specific diseases
200 whereas others show a protective effect (GÓMEZ-DURÁN *et al.* 2010). In model systems,
201 alternative strategies such as extensive backcrossing can be employed to pair the nuclear
202 genome with a variety of mitotypes. Recent examples from *Drosophila* (HOEKSTRA *et al.*
203 2013) and budding yeast (PALIWAL *et al.* 2014) support the idea that mitotypes adapt to
204 particular environments, perhaps rather quickly, and that nuclear-cytoplasmic crosstalk is
205 impacted not only by compatibility among the genomes but also by environmental
206 conditions.

207
208 In plants, much less is known about the number of mitotypes per species or across related
209 taxa and their individual adaptive advantages. Where mitotyping has been performed,
210 there remains a lack of experiments to investigate the functional differences among
211 mitotypes. However, there is evidence for high levels of diversity in mitotypes within a
212 single species. For instance, in the multi-chromosome mitochondrial genome harboring
213 *Silene* species, distinct populations of the same species may have different chromosome
214 numbers and stoichiometry (WU *et al.* 2015b). Other species do not appear to have as
215 much diversity on a species level, but diversity is present among closely related species
216 of the same genus. For instance, analysis in maize (ALLEN 2005) and wheat (WANG *et al.*
217 2000) has demonstrated a lack of cytoplasmic diversity in domesticated species although

diversity persists in their wild relatives.

2.2 Specific tools available to study NCI in plants

2.2.1 Cybrid plants and alloplasmic lines

There are a number of tools available to study NCI in plants. For instance, cytoplasmic and nuclear genomes can be mismatched or mixed in a number of ways including the generation of “cybrid” (cytoplasm hybrid) plants or “alloplasmic” (alien cytoplasm) lines. A relatively quick strategy for studying NCI is to generate “cybrid” plants, which is often through somatic fusion/protoplast fusion and regeneration methods (Figure 1a,b). These fusions can be symmetric (Figure 1a) or asymmetric (Figure 1b), with symmetric fusions containing nuclear and cytoplasmic genome content from both parents. In contrast, elimination or inhibition of the nuclear or cytoplasm genomes prior to protoplast fusion (by radiation or chemical treatment) results in asymmetric fusions that primarily contain the nucleus from one species and cytoplasm from the other. Protoplast fusions have been used extensively in diverse plants. Studies in cybrids can lead to a better understanding of homologous recombination upon organellar fusion, as in analysis of chimeric genomes in cybrids of *Nicotiana* (SANCHEZ-PUERTA *et al.* 2015) and *Brassica* (OSHIMA *et al.* 2010). Notably, the breeding potential of cybrids has been reported for diverse traits including improved disease resistance in *Solanum* (PERL *et al.* 1991) and for generating seedless citrus fruits (XU *et al.* 2006).

An alternative technique for mismatching nuclear and cytoplasmic genomes is the generation of alloplasmic lines (Figure 1c). This method takes advantage of cross-compatibility of related plant species (and sometimes even across genera) to mismatch cytoplasms and nuclear genomes. Recurrent backcrossing with the cytoplasmic donor always used as the female¹ results in lines with relatively “pure” nuclear genome content from the pollen parent mismatched with the cytoplasm from divergent female lineages (Figure 1c). This system is advantageous for studying specific NCI because multiple

¹ In most cases the cytoplasm and therefore the organelles are maternally inherited, enabling this backcrossing scheme. In some cases however, the cytoplasm is paternally inherited or organelles are bi-parentally inherited, requiring adjustment of the crossing scheme accordingly. See Section 3.2 ‘Transmission of Organelles’ for more detail.

cytoplasm types can be mismatched with the same nuclear genome (Figure 1d), but it takes time to generate the advanced backcross lines.

The largest known collection of alloplasmic lines was generated in wheat by three groups several decades ago by mismatching cytoplasm from >40 *Triticum* and *Aegilops* species with the nuclear genomes of several domesticated wheat species and cultivars. This resulted in hundreds of wheat alloplasmic lines (reviewed in (TSUNEWAKI 1980)). Due to two rounds of polyploidization events before domestication, a majority of cultivated wheat has been reduced to one cytoplasm type. Yet, restriction fragment length polymorphism (RFLP) analysis detected over 20 distinct cytoplasm types in alloplasmic lines containing cytoplasm from 33 distinct wild relatives (WANG *et al.* 2000). In another approach, measurement of 21 developmental traits found positive correlations between phenotype and cytoplasm type (TSUNEWAKI *et al.* 2002). For the most part, these correlations were consistent with the genetic differences described previously (WANG *et al.* 2000). However, a few inconsistent cases were identified that group into two error types, namely: (i) no phenotypic differences were observed although the cytoplasm types are genetically distinct, and (ii) phenotypic differences were observed although the cytoplasm types were identical (TSUNEWAKI *et al.* 2002). The first error type may be explained if the genetic differences are not in coding or regulatory regions and therefore do not alter gene expression or if the molecular changes simply do not alter the measured phenotypes. The second error type may be explained by limitations in the sensitivity of the genotyping assay (RFLP did not detect certain genotypic differences) or by differences in NCI in the alloplasmic lines (i.e. different nuclear fragments from the cytoplasm donors remain after backcrossing).

A breadth of phenotypes are observed in the alloplasmic lines ranging from negative (e.g. reduced fertility and poor grain quality) to positive (e.g. increased biomass and improved disease resistance) effects of the nuclear-cytoplasmic mismatching (WASHINGTON and MAAN 1974; PANAYOTOV 1980; TSUNEWAKI *et al.* 1996, 2002). Alloplasmic lines from wider crosses (between genera) such as introduction of the barley cytoplasm into wheat are also possible (AKSYONOVA *et al.* 2005). Molecular analyses are only beginning to

address the functional implications of the alloplasmic condition. For instance, comparative analysis of the mitochondrial genomes among a wheat alloplasmic line and its two parents indicated accelerated evolution of the mtDNA under the alloplasmic condition resulting in SNP variation and structural rearrangements (NOYSZEWSKI *et al.* 2014). Detailed analysis of one particular mitochondrial gene essential to ATP production, *ATP synthase subunit 6 (ATP6)*, revealed moderate SNP variation, but much more substantial structural and presence/absence variation across the *Triticum-Aegilops* complex, suggesting rearrangements as the predominate evolutionary driving force in the mitochondria (SOLTANI *et al.* 2014). Whole genome sequencing of mitochondria and plastids from diverse species are necessary to further our understanding of cytoplasmic evolution and the functional significance of specific genomic changes remain under investigation. However, functional evidence from studies in similar wheat alloplasmic lines indicates that gene expression and metabolism are significantly impacted under the alloplasmic condition with the greatest changes resulting from the widest crosses (CROSATTI *et al.* 2013).

Beyond wheat, there are few published alloplasmic collections to draw from. However, a thorough study performed in maize-teosinte alloplasmics (or “cytolines”) also found a range of phenotypic effects resulting from the alloplasmic condition and demonstrates that the more divergent the nuclear-cytoplasmic pairing, the greater the impact on developmental phenotypes (ALLEN 2005). Fifty-eight developmental characteristics were directly measured or calculated, including traits related to growth rate, fertility, yield, and seed quality. In alloplasmic lines containing more divergent cytoplasm from the *Luxuriantes* section, nearly all traits were significantly impacted, while phenotypic changes were modestly impacted for only a couple of traits in lines with more closely related cytoplasm from the *Zea* section. The literature is scattered with a few additional reports of alloplasmic lines such as in cruciferous crops (reviewed in (KANEKO and BANG 2014)) and carrot (STEINBORN *et al.* 1995).

2.2.2 CMS and NCS mutants

Another approach to investigate NCI is to examine the impact of particular cytoplasmic

mutants. Two classes of mitochondrial mutants have been identified in plants, cytoplasmic male sterility (CMS) and nonchromosomal stripe (NCS) mutants, which both impair normal NCI. CMS has been widely studied in crop species due to its utility for generating hybrid seeds for agriculture. CMS is primarily thought to be a result of mtDNA structural rearrangements. In many cases, CMS has been traced to the introduction of sterility-associated open reading frames (ORFs) and/or generation of chimeric ORFs in the mtDNA. These changes may result from spontaneous mutations or as a result of the alloplasmic or cybrid conditions in crosses between distinct populations or species with differing cytotypes (CHEN and LIU 2014). Often, essential mtDNA genes involved in the electron transport chain are affected in these genomic disruptions. For instance, altered *atp6* structure is implicated in CMS maize (DEWEY *et al.* 1991), rice (IWABUCHI *et al.* 1993; HU *et al.* 2013), and *Brassica sp.* (WANG *et al.* 1995; GEDDY *et al.* 2005; JING *et al.* 2012). Mutations in the *COX2* and *COX3* genes, encoding cytochrome c oxidase subunits, are connected to CMS in multiple species including maize (DEWEY *et al.* 1991), rice (HU *et al.* 2013), pepper (FUNG *et al.* 2004; Ji *et al.* 2013), and beet (DUCOS *et al.* 2001). Alternatively, it is also possible that point mutations or other mechanisms may lead to CMS. For instance, disrupted RNA editing of the mitochondrial *ATP6* gene in sorghum is also linked to CMS (KEMPKEN *et al.* 1998).

Nonchromosomal stripe (NCS) mutants of maize are also attributed to deleterious mitochondrial DNA mutations. Unlike CMS mutants, which are primarily detrimental during pollen development, NCS mutants have a more widespread impact on growth and development. While named for the green striping observed on leaves, other aspects of growth and development are impacted including plant stature and yield (SHUMWAY and BAUMAN 1967; NEWTON and COE 1986). The leaf striping phenotype observed in these mutants is due to reduced photosynthetic rate in the affected tissue, which is linked to mitochondrial heteroplasmy and chimeric sectoring of defective mitochondria and chloroplasts (light stripes) versus an admixture of defective and wild type organelles (normal green coloration) (GU *et al.* 1993). NCS mutants are due to deletions in mitochondrial genes that impact different aspects of the electron transport chain including NADH dehydrogenase (MARIENFELD and NEWTON 1994), cytochrome oxidase 2 (LAUER

et al. 1990; NEWTON *et al.* 1990), and a ribosomal protein (HUNT and NEWTON 1991; NEWTON *et al.* 1996). Newton and colleagues systematically demonstrated that both CMS and NCS mutants increase nuclear expression of alternative oxidase (*AOX*) genes, but in different patterns and to different degrees depending on the mtDNA mutation type (KARPOVA *et al.* 2002). Further investigations will be useful for teasing apart the genetic pathways involved in nuclear-cytoplasmic crosstalk.

2.2.3 "Recombination surveillance" mutants

Another approach for elucidating NCI is to investigate the impact of mutations in nuclear-encoded "recombination surveillance" mutants on organellar genome integrity and function. Plant mitochondria experience lower nucleotide substitution rates than in mammals and fungi, but extensive repetitive elements in their genomes facilitate more complex and dynamic structural rearrangements. Typically, organellar recombination is regulated by several nuclear-encoded genes including the *MutS HOMOLOG1 (MSH1)*, *RECA* homologs, and multiple *OSB* and *WHY* genes, both of which encode ssDNA binding proteins (ARRIETA-MONTIEL *et al.* 2009). Of all these factors, *MSH1* is the most studied in plants. It localizes to both mitochondria and chloroplasts in *Arabidopsis*, and a functional role for *MSH1* in both organelles appears conserved in diverse plant species (SANDHU *et al.* 2007; XU *et al.* 2011). The dynamic recombination of plant mitochondrial genomes is under the control of DNA double-strand break repair pathways and genome rearrangements are rampant in *msh1* mutants (DAVILA *et al.* 2011). Moreover, *Arabidopsis msh1 recA3* double mutants exhibit substantial structural rearrangements in mtDNA, which is correlated with altered expression of stress response genes and enhanced thermotolerance (SHEDGE *et al.* 2010).

Manipulation of *MSH1* activity is being harnessed in several crop systems as a powerful tool to study the functional implications of altered mitochondrial genome structure on plant development and productivity. RNAi suppression of *MSH1* results in a number of developmental phenotypes and stress response (XU *et al.* 2012a). Developmental phenotypes include dwarfism and other changes in plant architecture, altered leaf morphology, and variations in flowering time. These phenotypic changes have been

linked to heritable non-genetic (presumably epigenetic) changes also referred to as *MSH1*-developmental reprogramming (*MSH1*-dr) (XU *et al.* 2012a; DE LA ROSA SANTAMARIA *et al.* 2014; VIRDI *et al.* 2015; YANG *et al.* 2015). Work in sorghum *MSH1*-dr lines has demonstrated that the developmental changes induced by *MSH1* RNAi suppression are maintained after loss of the transgene and recovery of wild type *MSH1* transcript levels, even after several generations of selfing (DE LA ROSA SANTAMARIA *et al.* 2014). The *MSH1*-dr phenotype was lost in the F1 generation of a cross with wild type; however, phenotypic variation was regained in the F2 generation. Selection for yield traits in subsequent generations resulted in significant increases in grain yield and above ground biomass compared to a wild type inbred line. Likewise, selection in tomato *MSH1*-dr derived lines from crosses with wild type resulted in significant fruit yield increases by the F3 or F4 generations grown under both greenhouse and field conditions (YANG *et al.* 2015). Importantly, while fruit size decreased, the transition to flowering was faster, overall flower and fruit set was greater, and fruit ripened faster, without compromising fruit sugar content. Combined, these recent examples demonstrate a significant and heritable role of mitochondrial genome structure in plant development that may be harnessed to improve crop production.

Transgenic knockdown of *MSH1* can also induce CMS, as first demonstrated in tobacco and tomato (SANDHU *et al.* 2007). As described in previous sections, CMS is often the result of mitochondrial genome rearrangements leading to altered expression of novel ORFs. In some cases, suppression of CMS-associated ORFs by substoichiometric shifting is one mechanism for maintaining or restoring fertility. Combination lines with CMS and fertility restorer genes are powerful breeding tools for hybrid seed production and are currently used in crops such as maize. Interestingly, the CMS phenotype is stable over multiple generations in *MSH1* knockdown derived lines, even after loss of the transgene, which has promising implications for plant breeding efforts, particularly for production of hybrid seeds in crops where stable CMS lines are not yet available. Moreover, altered activity of *MSH1* in *Brassica juncea* is correlated to the substoichiometric shifting of the CMS-associated ORF220. Notably, increased ORF220 copy number was found in male sterile RNAi-*MSH1* lines, which provides a new approach to manipulate CMS for

agronomic use (ZHAO *et al.* 2015).

3 Mitochondria dynamics in plants

The already mentioned mtDNA size variability and the phenomenon of substoichiometric shifting are characteristic of plants and can manifest phenotypically. In addition, physical attributes of mitochondria such as size, shape and connectivity can also change drastically albeit with few reported implications on their functional relevance. Although the functional consequences of structural heterogeneity of mitochondrial bodies and their genomes remains partly unresolved, observations discussed in this section and others can be summarized by the following conclusions: (i) The canonical view of a single “master circle” does not capture the range of structural diversity and dynamics of plant mitochondrial genomes; (ii) mitochondria frequently fuse and divide which ensures mixing of all components, including genomes; and (iii) mitochondria are frequently localized at cellular areas of active processes where they can quickly and directly supply energy in the form of ATP, participate in biosynthesis of special lipids and proteins, or act as signaling hub.

3.1 Morphological & spatial dynamics during cell cycle and development

Mitochondria from higher plants are pleomorphic, existing as up to several hundreds of discrete organelles of spherical, sausage-like or tubular shape (STICKENS and VERBELEN 1996; LOGAN and LEAVER 2000; SHEAHAN *et al.* 2004). The collective mitochondrial bodies are known as the chondriome. In addition, mitochondria-like structures, which might be precursors or degradation products of mitochondria, were discovered by electron microscopy and cellular fractionation, and are termed spherical inclusions (SI) and slow sedimenting (ss) mitochondria, respectively (DICKINSON and LI 1988; DAI *et al.* 1998). SIs were mainly detected in EM studies of meiosis where their number peaks during meiosis II, and were hypothesized to be degenerating mitochondria (BELL and MÜHLETHALER 1964; DICKINSON and POTTER 1978; BIRD *et al.* 1983). The ss-mitochondria were found by centrifugation-mediated cell fractionation and shown to contain mtDNA, some mitochondrial proteins, reduced membranes and no respiratory activity (DAI *et al.* 1998).

Plant mitochondria are not distributed evenly throughout the cytoplasm. Instead, they aggregate in distinct locations and may change location throughout the cell cycle or dependent on cell type. During meiosis, many plants possess a characteristic aggregation of mitochondria not reported in animals, namely the organelle band. This separates the spindles in meiosis II when no cytokinesis takes place at the end of meiosis I (BROWN and LEMMON 2001; CHEN *et al.* 2005). Instead, mitochondria and vesicles form a distinct organelle band at the division plane (the position of the future cell wall), thus separating the two sets of spindles, which is essential for proper chromosome segregation (BROWNFIELD *et al.* 2015). Aggregations of mitochondria are also found around the nucleus and around the few chloroplasts in the hypocotyl (LOGAN and LEAVER 2000; SHEAHAN *et al.* 2004). These examples demonstrate that plant mitochondria accumulate around cellular areas with high energy need as in other organisms (reviewed in (BEREITER-HAHN and VÖTH 1994; LOGAN 2006a)), suggesting a micro-compartmentalization to overcome diffusion limitations of ATP (FRIEDRICH 1985).

The plant chondriome is highly dynamic and changes within seconds, with motions ranging from small continuous oscillations to large-scale movements (LOGAN and LEAVER 2000). Due to frequent fusion and fission which accomplish complete mixing of a cell's mitochondria in 1-2 hours (ARIMURA *et al.* 2004), the plant chondriome has been described as a “dynamic syncytium”, or more recently as a “discontinuous whole” (LONSDALE *et al.* 1988; LOGAN 2006b). In contrast to mammals, mitochondrial movement in plants depends mainly on actin, not microtubules ((OLYSLAEGERS and VERBELEN 1998; VAN GESTEL *et al.* 2002; SHEAHAN *et al.* 2004) and reviewed in (YAFFE 1999)). Mitochondrial fusion requires microtubules in plants, but neither actin nor microtubules are required in mammals (ISHIHARA *et al.* 2003; MATTENBERGER *et al.* 2003; SHEAHAN *et al.* 2005).

Increased dynamics and production of new mitochondria occur mainly in meristems and before need, e.g. cell elongation or growth ((FUJIE *et al.* 1994) and reviewed in (McCABE *et al.* 2000)). The amount of mitochondria increases several fold in developing flowers

(HUANG *et al.* 1994), possibly due to the demonstrated increase in male and female sporogeneous cells and tapetum layer of anthers (WARMKE and LEE 1978; DICKINSON and LI 1988). As in mammals, the plant mitochondria cycle is – at least partly - coordinated with the cell cycle, doubling the number of mitochondria before mitosis (SHEAHAN *et al.* 2004, 2005; ZOTTINI *et al.* 2006), and alternating appearances from a spherical shape in dividing cells to tubular shapes in cells preparing for division or undergoing expansion (STICKENS and VERBELEN 1996; SHEAHAN *et al.* 2004). Similar to yeast, there is also frequent fusion throughout the cell cycle (SHEAHAN *et al.* 2005). In special cases, large reticulate networks form, like (i) by “massive mitochondrial fusion” after fusion of dedifferentiated protoplasts (SHEAHAN *et al.* 2005), or (ii) in pollen (MCCONCHIE *et al.* 1987; MOGENSEN *et al.* 1990), or (iii) in the shoot apical meristem where a large tentaculate cage-like mitochondrion wraps around the nucleus (SEGUÍ-SIMARRO *et al.* 2008; SEGUÍ-SIMARRO and STAEHELIN 2009). Giant mitochondria, some of them reticulum-like, can also occur in an anoxic environment, e.g. in tobacco cell cultures under a nitrogen atmosphere (VAN GESTEL and VERBELEN 2002), or be triggered by disruption of the mitochondrial electron transport chain (mETC) (RAKHMATULLINA *et al.* 2015). A special cycle of de- and redifferentiation of mitochondria can be observed during male meiosis, with rapid organellar DNA synthesis and small round pro-mitochondria during prophase I and mitochondria redifferentiation in microspores after the tetrad stage (BAL and DE 1961; BIRD *et al.* 1983; DICKINSON and LI 1988).

3.2 Transmission of organelles

Physical mixing of mitochondria to facilitate homogenizing genome content is a sophisticated means to ensure viability for the next generation. Plants have evolved different mechanisms for transmitting mitochondria and plastids from one generation to the next. Evidence for maternal, paternal, and bi-parental inheritance of these two organellar types exists across the kingdom Plantae (reviewed in (NAGATA 2010); Figure 2). As in humans, plant mitochondria are typically maternally inherited. Likewise, plastids are usually inherited maternally together with the mitochondria. However, in some special cases, both mitochondria and chloroplasts are paternally transmitted to offspring such as in redwood trees (NEALE *et al.* 1989). There are some interesting cases

of bi-parental inheritance as well, e.g. in cucumber where the mitochondria are maternally inherited, while the plastids are paternally inherited (HAVEY 1997). Across the eukaryotes, multiple mechanisms control organellar transmission from mother to daughter cells during cell division and from one generation to the next during embryogenesis (reviewed in (SATO and SATO 2013; GREINER *et al.* 2015)). For instance, it is known for some plant species (e.g. as in some members of the Solanaceae family) that plastids can be excluded or degraded from the pollen generative cell early in pollen development, while for others like wheat, the plastids persist until fertilization of the egg cell and are excluded upon fertilization (reviewed in (GREINER *et al.* 2015)). Paternal leakage of both organelle types have been observed in several plant species (reviewed in (XU 2005; GREINER *et al.* 2015)). Interestingly, interspecific crosses, for instance between rye and wheat (SINIAVSKAIA *et al.* 2004) and barley and wheat (AKSYONOVA *et al.* 2005), may increase the odds of paternal leakage. Whether this is linked to the specific mechanism of parent-of-origin exclusion of organelles in the *Triticum* and related cereals is unresolved. It will be interesting to see how often hybridization impacts this inheritance pattern of increased paternal leakage in more diverse plant species, and also if this may correlate with overcoming crossing barriers. Nonetheless, it is important to keep in mind that mechanisms controlling the inheritance of organelles impact the genetic diversity available in a population over time and may have functional consequences.

3.3 Genome dynamics - heteroplasmy & substoichiometric shifting in development and stress response

Mitochondrial genomes consist of dynamic molecules that may undergo structural changes throughout the cell cycle in somatic cells or during the transmission to the next generation during embryogenesis. Individual eukaryotic cells contain many – up to thousands – of mitochondria, and in the case of plants also plastids. Not all organelles within an organism have identical genomic sequences, and in fact an admixture of organellar genomes can be found within a single cell (reviewed in (WOLOSZYNSKA 2010)). Each organelle can also have multiple copies of the genome or, as the other extreme, an incomplete genome (SATO *et al.* 1993; ARIMURA *et al.* 2004). Maintaining different subtypes is known as heteroplasmy. Different subtypes may exist at different

levels (stoichiometries) within a given tissue type or even at the cellular level within a single individual (reviewed in (WOLOSZYNSKA 2010)). Subtypes may thrive under varying conditions and therefore it is possible for the levels of each subtype to shift over time and/or environmental condition, a phenomenon known as substoichiometric shifting. In plants, this phenomenon was first described in maize when comparing structure and copy number of a conserved gene, *atpA*, in male sterile versus fertile cytoplasms (SMALL *et al.* 1987). While this phenomenon is still poorly understood, evidence in several additional species including bean (JANSKA 1998), rice (BENTOLILA and STEFANOV 2012), and *Brassica napus* (CHEN *et al.* 2011) has demonstrated that stoichiometric shifting of plant mitotypes can lead to developmental defects such as male sterility. Even under normal conditions, the relative abundance of each chromosome in multi-chromosome species may vary significantly as has been observed in cucumber (ALVERSON *et al.* 2011) and in *Silene noctiflora* populations, which differ in the number of chromosomes (WU *et al.* 2015a). The functional significance of these stoichiometries is an active area of research.

During cell division, organelles with different admixtures of their genetic content may distribute unevenly into the daughter cells. Deleterious mtDNA alleles starting at low levels in a particular cell or tissue type may or may not reach a pathogenic threshold over time due to the uneven propagation and/or distribution of mitochondria. In humans, deleterious mtDNA alleles appear to be common (ELLIOTT *et al.* 2008; YE *et al.* 2014) and have been linked to numerous diseases and aging (reviewed in (WALLACE 2005; CHAN 2006)). Therefore, understanding the mechanisms controlling the replication and distribution of mitotypes is essential for human health.

Plant mitochondrial genomic content is not always divided equally as mitochondria propagate via fission (ARIMURA *et al.* 2004). However, as mentioned before, massive fusion of plant mitochondria is observed before fission occurs, which likely mixes the genomic contents well (SHEAHAN *et al.* 2005). In addition, novel sequences are generated, mainly attributed to homologous recombination during the fusion of mitochondria. Abundant opportunities for recombination and thus diversification of

mtDNA exist according to the already discussed “discontinuous whole” hypothesis which proposes frequent transient fusion of small mitochondria (LOGAN 2006a; b). Another aspect adding to the immense mtDNA variability in plants was reported in Amborella, considered as one of the most ancient extant flowering plants, which harbors an extreme example of mitochondrial horizontal gene transfer (RICE *et al.* 2013). This species has taken up entire genomes from other algal and plant species resulting in more “foreign” than native mitochondrial sequences. It is hypothesized that uptake of mitochondria, followed by fusion and subsequent whole-genome recombination events led to its large and complex genome. Even in more moderately sized plant mtDNA, such as in maize, a majority of the mtDNA is of unknown origin (CLIFTON *et al.* 2004). Moreover, there has also been significant movement of plastid genome content into the mitochondrial genome in plants (reviewed in (PALMER *et al.* 2000)).

4 Roles of mitochondria in development

4.1 Mitochondria as key players in development

Mitochondria are key factors in regulating metabolism, programmed cell death (PCD) and intracellular signaling ((EPSTEIN *et al.* 2001) and reviewed in (RAVAGNAN *et al.* 2002; GALLUZZI *et al.* 2012)), all of which are integral parts of plant growth and development. Prominent examples include homeotic changes in flower morphology caused by mitochondria defects, as seen in some CMS and alloplasmic lines (GERSTEL *et al.* 1978; BERETERBIDE *et al.* 2002; LINKE *et al.* 2003; LEINO *et al.* 2003; ZUBKO *et al.* 2003; TEIXEIRA *et al.* 2005; NIKOVA and VLADOVA 2014). Defects in flower morphology might be due to retrograde signaling targeting nuclear floral developmental regulators ((ZUBKO *et al.* 2001; MURAI *et al.* 2002; BERETERBIDE *et al.* 2002; GEDDY *et al.* 2005) and reviewed in (ZUBKO 2004; LINKE and BÖRNER 2005)), or due to affected ATP-dependent proteolysis of floral developmental regulators (TEIXEIRA *et al.* 2005).

Reproductive tissues seem to be most severely impaired by mitochondrial defects, although vegetative development can also be affected manifesting in slow growth and stunted stature. Mutants of plant mitochondrial polymerase IB for example show a cell-cycle related phenotype with reduced growth and less cell expansion (PARENT *et al.*

2011; CUPP and NIELSEN 2013). Mutations causing mitochondrial defects do not necessarily have to be in mitochondrial-encoded genes, but can also reside in nuclear-encoded genes with mitochondrial function (CHRISTENSEN *et al.* 2002; BERTHOMÉ *et al.* 2008). An example is female sterility caused by mutation in the nuclear encoded gene Hüllenlos (HLL), encoding a mitochondrial ribosomal subunit (SKINNER *et al.* 2001).

4.2 Mitochondrial respiration in plant development

One likely reason for the sensitivity of plant reproductive tissues to mitochondrial defects is that they exhibit the highest respiratory rates, e.g. during seed germination, pollen development, and fruit ripening (MACKENZIE *et al.* 1994). This is reminiscent of human mitochondrial defects: due to heteroplasmy, pathogenic mutations can be tolerated when occurring as a minor portion of the mitochondria population of a cell. However, these defective mitotypes can manifest in severe human diseases when a higher energy demand exists, which is the case in certain cell types like cardiomyocytes (heart muscles) or neurons in the brain (reviewed in (DE CASTRO *et al.* 2010; LIMONGELLI *et al.* 2012)). Diseases in infancy or early childhood can also arise from defective mitochondrial function, mainly due to mutations in nuclear-encoded genes (reviewed in (NOGUEIRA *et al.* 2014)).

In plants, it was found that a high respiratory demand, i.e. high energy and metabolic requirements, might be reflected in and met by an increased number or area of mitochondria and specific increase of e.g. components of the mETC (WARMKE and LEE 1978; HUANG *et al.* 1994; ZABALETA *et al.* 1998). The root apical meristem of Arabidopsis and other plants increases in mitochondria number around 3-fold, in parallel with an increase in respiratory rate (YEMM 1965; KUROIWA *et al.* 1992; FUJIE *et al.* 1993). Pollen respire ~10x faster (DICKINSON 1965, 1966), and the number of mitochondria is already increased 20- to- 40-fold in meiotic and tapetal cells (WARMKE and LEE 1978; LEE and WARMKE 1979).

Not surprisingly, expression levels of mitochondrial genes are elevated in male gametogenesis, but this is not strictly due to a higher number of mitochondria since it

could be shown that components of the mETC are specifically increased (SMART *et al.* 1994; DUKOWIC-SCHULZE *et al.* 2014a; b). Pollen abortion in CMS plants strengthens the dependency of male gametogenesis on energy production and biosynthetic function of mitochondria (reviewed in (HANSON and FOLKERTS 1992; MACKENZIE *et al.* 1994)). Another high energy-requiring developmental process is seed germination and respiration, and mETC and energy metabolism are activated accordingly (EHRENSHAFT and BRAMBL 1990; DEKKERS *et al.* 2013).

What is unique for plant respiration, and described in more detail in section 5, is the existence of additional components to bypass complex I, III and IV of the mETC, namely by NAD(P)H-dehydrogenases (NAD(P)H-DHs) and alternative oxidase (AOX) in addition to Uncoupling Proteins (UCPs) which are also present in animals (reviewed in (JARMUSZKIEWICZ 2001; JARMUSZKIEWICZ *et al.* 2004; RASMUSSEN *et al.* 2004)). All bypasses lead to lower ATP production since they do not contribute to the proton gradient – their importance rather lies in ROS prevention, thus functioning as antioxidants ((UMBACH *et al.* 2005) and reviewed in (RASMUSSEN *et al.* 2004)).

AOX is especially intriguing since it has five family members with tissue- and developmental stage-specific expression for different purposes, e.g. stress, senescence, seed maturation and flower development (SAISHO *et al.* 2001; THIRKETTLE-WATTS *et al.* 2003; CLIFTON *et al.* 2006). Fruit ripening has been shown to involve both AOX as well as UCP (ALMEIDA *et al.* 1999; CONSIDINE *et al.* 2001; XU *et al.* 2012b). In addition, AOX is crucial for the temperature increase in reproductive tissues of thermogenic plants (reviewed in (VANLERBERGHE and MCINTOSH 1997; ZHU *et al.* 2011)), which facilitates the emission of volatile molecules to attract pollinators, and might also support adaptation to cold climate, as well as optimal pollen germination and pollen tube growth (SEYMOUR *et al.* 2003, 2009; ONDA *et al.* 2008; SEYMOUR and GIBERNAU 2008).

4.3 Regulation of growth and development

AOX and canonical mitochondrial respiratory proteins can be bound and regulated by thioredoxins (RHOADS *et al.* 1998; GELHAYE *et al.* 2004). Beside redox signaling, carbon

status and especially citrate are involved in regulating AOX synthesis and activity (VANLERBERGHE and MCLINTOSH 1996; SIEDOW and UMBACH 2000). TCP transcription factors participate in the regulation of genes encoding mETC components, and are linked to growth and development, making them likely key players in integrating mitochondrial gene expression with the demand in growth (MARTÍN-TRILLO and CUBAS 2010). In addition to functioning in normal growth, mitochondria also act in differentiation, e.g. via their involvement in redox homeostasis and redox signaling. A prominent example for the connection between ROS and developmental regulation, albeit nuclear-encoded, is the transcription factor UPBEAT1 (UPB1), which has been described as a key regulator for the transition from proliferation to differentiation in root growth (TSUKAGOSHI *et al.* 2010). Mutants of *upb1* show disturbed ROS balance regulation and distribution, resulting in alteration of meristem size and root length (TSUKAGOSHI *et al.* 2010).

While ROS have long been interpreted as mere oxidative stress, their rather important role as signaling components has now been acknowledged (reviewed in (FOYER and NOCTOR 2005; MØLLER and SWEETLOVE 2010)). ROS are particularly important as signals in reproductive development, i.e. pollen germination, pollen tube growth and fertilization (MARTIN *et al.* 2013; LASSIG *et al.* 2014). The oxidative burst released from synergid cells to signal pollen tube arrival is especially intriguing (MARTIN *et al.* 2013). ROS are also produced during germination in multiple species, where they have been proposed as a signal for release from seed dormancy (ORACZ *et al.* 2009; LEYMARIE *et al.* 2012; ISHIBASHI *et al.* 2013). Furthermore, redox status was suggested to be a cell fate determinant in anthers as well as in roots (JIANG *et al.* 2006; KELLIHER and WALBOT 2012).

Mitochondria are also key factors in cell death signaling (reviewed in (LAM 2004; BRAS *et al.* 2005; LOGAN 2006b)). ROS bursts are needed for pollen maturation, e.g. PCD of the tapetum layer (XIE *et al.* 2014; AN *et al.* 2015), and timely and targeted PCD is integral for both male and female gametogenesis ((PAPINI *et al.* 1999, 2011) and reviewed in (NICK 2011)). Mitochondria even change their shape in PCD from sausage-like to spherical in leaves, followed by swelling ((YOSHINAGA *et al.* 2005) and reviewed

in (LOGAN 2006b)), possibly indicating further involvement.

5 Roles of mitochondria in stress response

5.1 Overview of stressors and stress responses in plants

Abiotic and biotic stresses severely limit plant productivity and the range of potentially arable land. Abiotic stresses are defined as poor environmental and growing conditions and can include drought, salinity, cold, anoxia, and pollution (CRAMER *et al.* 2011; MICKELBART *et al.* 2015), whereas biotic stresses are caused by other living organisms and consist of herbivory and pathogen attack (FUJITA *et al.* 2006; ATKINSON and URWIN 2012). The first step in mitigating the deleterious effects of stress is timely detection and response, and stress exposure activates signal cascades that lead to extensive modulation of transcriptional networks that are specific to the type of stress encountered. Post-transcriptional and -translational modifications also induce rapid functional changes in already existing molecules (MAZZUCOTELLI *et al.* 2008). Furthermore, epigenetic processes, such as small RNAs and DNA methylation, play a role in the transgenerational memory of stress (KINOSHITA and SEKI 2014). While all of the aforementioned mechanisms are of great importance to stress response, mitochondria play a crucial role by acting as sensors of environmental perturbation when metabolic pathways are disrupted. Mitochondria also function in translating stress perception into energy-deficiency signals and then re-establishing metabolic balance (PU *et al.* 2015). Upon stress exposure, respiration and photosynthesis flux change and an energy deficit signal is produced which leads to global changes in organellar and nuclear gene expression (HUNER *et al.* 1998; WILSON *et al.* 2006).

A common feature of abiotic and biotic stress in both plants and animals is the rapid generation of excess ROS, much of which is generated in mitochondria and chloroplasts (MØLLER 2001; WALLACE 2005; MUJAHID *et al.* 2006; SPOONER and YILMAZ 2011; KIKUSATO and TOYOMIZU 2013; COLOMBATTI *et al.* 2014). In general, stress can alter the rate at which certain mETC complexes donate or accept electrons. These changes to electron flow ultimately lead to over-reduction of certain mETC sites which causes electron leakage to O₂ and subsequent ROS production (JACOBY *et al.* 2011). The mETC

Complexes I and III are the primary sites of mitochondrial ROS production, similar to animals (MØLLER 2001), but recent work indicates that Complex II generated ROS is also required for biotic and abiotic stress responses (GLEASON *et al.* 2011). ROS include superoxide, hydrogen peroxide, and hydroxyl radicals which oxidize cellular constituents and compromise organellar membrane integrity (MØLLER and SWEETLOVE 2010; SUZUKI *et al.* 2012; GOLLDACK *et al.* 2014). Oxidative stress due to uncontrolled ROS production can lead to PCD in response to pathogens (COLOMBATTI *et al.* 2014) and during abiotic stress (PETROV *et al.* 2015), as well as to redox imbalances and inhibition of photosynthesis (GOLLDACK *et al.* 2014). Typically, excess ROS will be scavenged by antioxidants and enzymes that aid in detoxification such as catalase and metal-containing superoxide dismutases (SODs) (LÁZARO *et al.* 2013). In plants, alternative oxidase (AOX) plays a key role during stress by lowering ROS production from the mETC by preventing excessive reduction of the mitochondrial ubiquinone pool (PASTORE *et al.* 2007; VANLERBERGHE 2013; SCHERTL and BRAUN 2014). Other mitochondrial proteins can also dissipate excess energy flow through the mETC during stress, including an ATP-sensitive potassium channel and uncoupling protein (UCP) (PASTORE *et al.* 2007). Plant UCP dissipates the transmembrane potential generated by respiration as heat, therefore lessening production of ROS, similar to the function of yeast mitochondrial unspecific channels (MUCs) and mammalian permeability transition pores (PTPs) (GUERRERO-CASTILLO *et al.* 2011). Although uncontrolled ROS production is injurious to organisms, organellar ROS generation during stress can also serve as a mitochondrial retrograde (mitochondria to nucleus) signal resulting in expression changes of ROS responsive regulatory genes involved in tolerance to various stresses (LÁZARO *et al.* 2013). Transmission of ROS signals likely occurs via detection of the subcellular location of ROS production, damaged and oxidized cellular components, and hormones. Together, these signals ensure coordination of cytoplasmic and nuclear stress responses (GECHEV *et al.* 2006; MØLLER and SWEETLOVE 2010; SUZUKI *et al.* 2012).

An emerging pattern in the literature indicates that abiotic stress tolerant species and cultivars frequently show enhanced gene expression and protein abundance of mitochondrial localized antioxidant defense genes when compared to less tolerant

cultivars. The same trend is evident for biotic stress tolerant cultivars, although there is a dearth of literature on the topic. Additionally, gene expression induction and enzymatic activity of these proteins is typically dependent on mitochondrial ROS production, providing further evidence for the role of mitochondria in abiotic and biotic stress tolerance. Moreover, genetic diversity in mitochondrial DNA polymorphisms (haplogroups or mitotypes) in animals and plants influences stress response and physiology. Below, we discuss the different strategies that plant mitochondria employ to combat stress and also the implications of mitochondrial genetic diversity for stress response.

5.2 Excess energy dissipation to prevent ROS production and promote stress tolerance

Plant mitochondria have numerous methods of controlling excess energy that flows through the mETC during stress and thus are able to lessen ROS production. In addition to the aforementioned role of AOXs in thermogenesis in floral organs (VANLERBERGHE and MCINTOSH 1997), AOX is also crucial for preventing excess electrons leaking to oxygen when there is over-reduction of ubiquinone during times of excess energy demand, such as stress. In general, nuclear-encoded *AOX* expression is induced by many different biotic and abiotic stress conditions and represents a classic example of mitochondrial retrograde regulation (MRR) (HANQING *et al.* 2010; NG *et al.* 2014). The plant hormone salicylic acid (SA) is produced in response to biotic stress and is known to disrupt mitochondrial function (AMIRSADEGHI *et al.* 2007). Although little is known about the role of AOX during biotic stress, the literature is in agreement that increased SA levels after infection with bacteria or virus can lead to strong induction of *AOX* transcripts which accompanies PCD during the hypersensitive response (HR) (CHIVASA 1998; SIMONS 1999; LACOMME and ROBY 1999; VIDAL *et al.* 2007). However, following an initial induction to combat oxidative stress, *AOX* expression is suppressed in many plant species which likely allows for accumulation of adequate ROS amounts for the oxidative burst that triggers PCD during HR (AMIRSADEGHI *et al.* 2007; HANQING *et al.* 2010). Both chewing herbivores and piercing-sucking insects induce expression of *AOX*, and downregulation of *AOX* using RNA interference (RNAi) leads to more damage

caused by piercing-sucking insects but not rapidly-feeding herbivores (ZHANG *et al.* 2012). Similarly, virus-resistant tobacco cultivars show a stronger induction of *AOX* expression than virus-susceptible cultivars (CHIVASA 1998). Likewise, in *Arabidopsis* cell cultures treated with a bacterial virulence factor, most ROS is of mitochondrial origin (KRAUSE and DURNER 2004).

Additionally, during abiotic stress exposure (including drought, salt, and heat) in rice, *OsAOX1a/b* induction requires mitochondrial-generated superoxide, further demonstrating the role of MRR in abiotic stress tolerance and response (LI *et al.* 2013). Exposure to increased metal concentrations also causes increased ROS generation and mitochondria play a key role in its detoxification (KEUNEN *et al.* 2011). For example, in both pea roots exposed to lead (Pb) (MALECKA *et al.* 2009) and *Arabidopsis* protoplasts exposed to aluminum (Al) (LI and XING 2011), expression of *AOX* increases indicating its crucial role in reducing oxidative stress caused by metals. Likewise, mitochondria from drought tolerant durum wheat also have highly active AOX, UCP, and an ATP-sensitive potassium channel (PASTORE *et al.* 2007). Furthermore, perturbation of ROS signaling, MRR, and reduced *AOX* expression induction was found in *Arabidopsis thaliana* overexpressing the nuclear encoded ROS-inducible transcription factor *AtWRKY15*, which lead to increased susceptibility to osmotic stressors such as salt stress (VANDERAUWERA *et al.* 2012).

The transmembrane potential-dissipating UCP and the excess redox energy-dissipating AOX both lead to a reduction in ROS production caused by a decrease in the efficiency of oxidative phosphorylation (NOGUEIRA *et al.* 2005). Similar to what is seen for *AOX*, *UCP* expression is frequently induced by both biotic and abiotic stresses (NOGUEIRA *et al.* 2005). In many plant species, overexpression of either *AOX* or *UCP* can lead to increased abiotic stress tolerance by greatly diminishing ROS production during stress (BRANDALISE *et al.* 2003; SMITH *et al.* 2009; VAN AKEN *et al.* 2009; VANLERBERGHE 2013). For example, overexpression of *A. thaliana AtUCP1* in tobacco leads to increased drought and salt tolerance (BEGCY *et al.* 2011) and overexpression of *AOX* in *A. thaliana* protoplasts improves Al tolerance (LI and XING 2011). *AOX* overexpression in tobacco

also leads to smaller HR lesions than wild-type plants, suggesting that AOX mitigates excessive PCD during HR (ORDOG *et al.* 2002). Taken together, the literature indicates that dissipation of excess energy, and hence decreased ROS production, leads to enhanced stress tolerance.

5.3 Regulation of energy production as a stress tolerance measure

As stress responses are energetically demanding, ATP production and energy management during stress is crucial for an effective response. *MtATP6* is a nuclear gene which encodes a 6-kDa subunit of the mitochondrial F₁F₀-ATP synthase complex and expression of this gene is upregulated during salt stress in *Arabidopsis* (ZHANG *et al.* 2008), wheat (MOGHADAM *et al.* 2013), and rice (ZHANG *et al.* 2006). In wheat, both salt tolerant cultivated and wild species have higher and more prolonged expression of *MtATP6* during stress than salt sensitive cultivars (MOGHADAM *et al.* 2013). *MtATP6* from the biodiesel crop *Jatropha curcas* was independently identified as conferring salt tolerance using a heterologous screening system in yeast (ESWARAN *et al.* 2010). Aluminum tolerance in wheat is also associated with increased activity of the mitochondrial F₁F₀-ATP synthase complex (HAMILTON *et al.* 2001). In *Arabidopsis*, overexpression of the mitochondrial AAA-type ATPase *AtOM66* leads to increases in both drought tolerance and pathogen resistance, demonstrating the dual function of mitochondria in response to both abiotic and biotic stresses (ZHANG *et al.* 2014). Ectopic overexpression of *RMtATP6* and *AtMtATP6* in tobacco (ZHANG *et al.* 2006) and yeast (ZHANG *et al.* 2008), respectively, also increases tolerance to salt stress. Taken together, the literature suggests that expression of nuclear encoded *MtATP6* can act as an anterograde signal to regulate energy metabolism during stress.

5.4 Scavenging ROS and alleviating ROS damage to cope with stress

Mitochondrial antioxidants and detoxification enzymes play crucial roles in stress tolerance by mitigating the effects of excess ROS production. The mETC components are particularly damaged by ROS and adequate protection is vital to maintain ATP production during stress (JACOBY *et al.* 2011). Manganese-containing SOD (MnSOD) is typically localized to mitochondria and its expression and activity levels are generally

altered by biotic and abiotic stress (Y WANG, M WISNIEWSKI 2007; HANQING *et al.* 2010). In *A. thaliana*, salt stressed plants display both up- and down-regulation of *SOD* gene expression, depending on the ecotype background and the type of *SOD* examined (ATTIA *et al.* 2008). Importantly, overexpression of mitochondrial *MnSOD* in many species, including *Arabidopsis* (WANG *et al.* 2004), tomato (Y WANG, M WISNIEWSKI 2007), and poplar (WANG *et al.* 2010a), increases salt tolerance. Additionally, overexpression of *MnSOD* from an Al-resistant wheat cultivar in *Brassica napus* leads to a reduction in symptoms of Al injury (BASU *et al.* 2001). In wheat, mitochondrial proteomic analysis of salt-sensitive and salt-tolerant Australian cultivars found quantitative differences in abundance of proteins such as MnSOD and AOX, suggesting that differences in mitochondrial ROS defense pathways correlate with salinity tolerance (JACOBY *et al.* 2010). Another proteomic study of an amphidiploid line of wheat that combines the genome of bread wheat with the genome of the salt-tolerant wild wheatgrass *Lophopyrum elongatum* found increased abundance of mitochondrial enzymes, including MnSOD, that could account for the increased tolerance in the amphidiploid (JACOBY *et al.* 2013). In *Arabidopsis*, more than half of the salt stress induced proteins are localized to the mitochondria (HEAZLEWOOD *et al.* 2007; LÁZARO *et al.* 2013) and mitochondrial ROS defense-related protein abundance is increased in 95% of all published studies on salinity stress in diverse plant species (JACOBY *et al.* 2011). In general, *MnSOD* expression and activity levels are positively correlated with salt tolerance and overexpression of *MnSOD* helps mitigate excess ROS production during stress. Moreover, activity levels of other antioxidant enzymes, including mitochondrial ascorbate peroxidase and glutathione reductase, are also increased in salt-tolerant varieties of tomato and wheat (SAIRAM and SRIVASTAVA 2002; MITTOVA *et al.* 2004; BOUTHOUR *et al.* 2015). In contrast to abiotic stress, ROS scavenging enzymes are initially downregulated during pathogen attack, similar to what is seen for AOX, to allow enough accumulation of ROS to trigger the oxidative burst that initiates PCD to prevent the spread of the invading pathogen (AMIRSADEGHI *et al.* 2007; KÜNSTLER *et al.* 2007; HANQING *et al.* 2010).

Proline performs as both an osmoprotectant, for which it is most commonly known, as

well as a ROS scavenger, and its catabolism occurs in the mitochondria (SZABADOS and SAVOURÉ 2010; JACOBY *et al.* 2011). Therefore, proline abundance is directly controlled by the mitochondria and plays a key role during abiotic stress. For example, durum wheat mitochondria show decreased catabolism of proline during salt stress and hence increased abundance of proline (FLAGELLA *et al.* 2006). Additionally, increased abundances of proline may also act as a convenient source of energy after stress as proline levels have been observed to rapidly decrease upon stress abatement (VERSLUES and SHARMA 2010; SZABADOS and SAVOURÉ 2010).

5.5 Implications of mitochondrial genetic diversity for stress response

A growing body of literature on mitochondria from different species indicates that these organelles evolve together with the cell and can drive adaptation to the environment and the unique stresses found in different geographic locations. As a result of this adaptation, there is extensive variation in the genome and protein composition of mitochondria. Much of the literature on how mitochondrial diversity can impact stress response and drive adaptation is from non-plant systems, such as human and yeast. For example, in the yeast *Saccharomyces cerevisiae* there is extensive variation in isolates from different environments (PALIWAL *et al.* 2014). Altering native NCI in these divergent *S. cerevisiae* isolates leads to changes in growth rate and ROS production (PALIWAL *et al.* 2014). Studies in other yeast species also demonstrate the importance of mitochondria in adaptation to the environment. For example, in contrast to non-marine yeast species, *Debaryomyces hansenii* (halophilic marine yeast) MUCs close in response to Na⁺ and K⁺ under high salinity conditions therefore increasing transmembrane potential and ATP synthesis (CABRERA-OREFICE *et al.* 2010). Consequently, we can infer that this shift in MUC function drove the adaptation of *D. hansenii* to saline environments.

In plants, hybridization can frequently result in organellar genome heteroplasmy and phenotypes such as CMS, as previously discussed in the review, and organellar genome variation within plant species was long thought to only occur in experimental plant populations. Interestingly, artificially induced rearrangements in mtDNA via *MSH1* disruption results in enhanced abiotic stress tolerance, suggesting that mitochondrial

sequence variation can directly impact stress responses (SHEDGE *et al.* 2010). However, it is now clear that natural plant populations show extensive mitochondrial variation, although most of the sequence variations do not result in obvious phenotypic differences (WOLOSZYNSKA 2010). However, it stands to reason that other naturally occurring mitotypes and sequence variations might also have adaptive significance, and this is indeed indicated by the reported mitotype variation in *Arabidopsis* ecotypes, which correlate with geographic origin (DAVILA *et al.* 2011). Moreover, naturally-occurring CMS in has been found in many different plant species with various mitotypes (CUGUEN *et al.* 1994; RONFORT *et al.* 1995; CHANG *et al.* 2011; CHEN *et al.* 2011). The most prominent example is the phenomenon of gynodioecy, which is the simultaneous existence of both hermaphroditic and male-sterile plants in a population. There, the benefits are dual – from an evolutionary perspective, outcrossing and thus higher genome variability is strengthened, and from a productivity view, female-only plants produce more seeds (BUDAR and PELLETIER 2001; DELPH *et al.* 2007; MA 2013). A recent study that profiled transcription of mitochondrial genes in natural populations of the gynodioecious species *Silene vulgaris* found that mitochondrial haplotypes were correlated with transcriptome variation (ELANSARY *et al.* 2010). This suggests that mitochondrial DNA sequence variation could directly influence transcription and therefore other traits, such as stress response. Additionally, metabolomics analysis of a reciprocal cross-derived recombinant inbred line (RIL) population of *Arabidopsis* revealed that variation in the organellar genome influenced the levels of over 80% of profiled metabolites (JOSEPH *et al.* 2013). Further exploration of currently available large sequencing datasets of natural plant populations, such as the 1001 genomes project of *Arabidopsis* accessions, could provide a deeper understanding of the extent and function of naturally occurring mitochondrial DNA variation in addition to nuclear genome variation (CAO *et al.* 2011).

As discussed in section 2.2.1, alloplasmics are a useful tool for understanding cytonuclear crosstalk and an extremely large collection exists in the *Triticum* genus which has proven useful for investigating the physiological effects of different cytoplasms (TSUNEWAKI *et al.* 2002). In many instances, substitution of native cytoplasm with those of wild relatives

can lead to improvements in stress tolerance. Alloplasmic lines have been found to differ from euplasmic controls for resistance to leaf rusts (WASHINGTON and MAAN 1974; SECHNYAK *et al.* 2010). Additionally, the alloplasmic condition can lead to substantial improvements in tolerance to abiotic stresses, such as salt and drought (WU *et al.* 1998; HOU *et al.* 2000; LIU *et al.* 2002, 2005; KHOLODOVA *et al.* 2007).

Another example demonstrating how mtDNA polymorphism could influence stress response comes from a recent study of *Drosophila* lines that contain a temperature-sensitive nuclear-mitochondrial incompatibility (HOEKSTRA *et al.* 2013). In this study it was shown that lines with the incompatibility, due to incompatible SNPs in a mt-tRNATyr and a nuclear-encoded tyrosyl-tRNA synthetase, had reduced growth rate and overall inferior performance in warmer temperatures but this was mitigated at cooler temperatures. Based on the results of this study, one can speculate that mitochondrial genetic diversity can drive the distribution of species, including humans, as populations may carry deleterious alleles that are hidden in environments with certain stressors (i.e. good air quality or cooler temperatures) and uncovered in others (i.e. air pollution or warmer temperatures) (HOEKSTRA *et al.* 2013). Indeed, many different mtDNA haplogroups with distinct geographical origins exist in humans but the physiological significance of these groups is just beginning to be understood (WALLACE 2005).

Similar to what is seen for *Drosophila*, mtDNA variants in humans also play a role in driving adaptation to different climates. When humans initially migrated from Africa into colder northern climates specific mtDNA variants were selected for. These mutations are in UCPs and result in a less tightly coupled respiratory chain, thereby increasing thermogenesis and decreasing ATP synthesis which is adaptive in cold environments (MISHMAR *et al.* 2003; WALLACE 2005; WALLACE and CHALKIA 2013; NISHIMURA and WATANUKI 2014). Mitochondrial genetic diversity can also drive the distribution of fish and other ectothermic animal species. Specifically, their distribution is limited by the thermal tolerance of their mitochondria. Less thermal tolerant species experience heart failure at warmer temperatures due to decreased stability of their mitochondria and lack of ATP synthesis (IFTIKAR *et al.* 2014). Hence, more tolerant species have thermal

protection conferred by the mitochondria. Although there is little evidence in plants that mitochondrial genetic diversity can drive species distribution, this is an area that warrants further investigation.

In addition to driving adaptation to climate, variations within the human mtDNA are also correlated with different disease states and haplogroups have different energetic capacities. For example, certain SNPs in the control region of the mtDNA, which controls DNA replication and hence mitochondrial biogenesis rates, are associated with metabolic phenotypes, such as obesity, and markers of oxidative stress (WENG *et al.* 2013). Human cybrid cell lines with mitochondria from either the H (European) or L haplogroups (African) have different ROS levels and OXPHOS efficiencies which may explain why different haplogroups show differential predispositions to certain diseases (KENNEY *et al.* 2014). Indeed, mitochondrial haplogroups and other mitochondrial DNA variations are associated with many complex diseases and cancers, such as Alzheimer's (VAN DER WALT *et al.* 2004), Parkinson's (PYLE *et al.* 2005), breast cancer (CANTER *et al.* 2005; BLEIN *et al.* 2015), and cervical cancer (GUARDADO-ESTRADA *et al.* 2012). Different haplogroups can also have protective effects against other environmental stresses, such as air pollution. Traffic-related air pollution is comprised of particles containing redox active transition metals and may cause increases in oxidative stress. A recent study found that mitochondrial haplogroup U (Eastern European/Asian) is protective for air pollution-related inflammation when compared to the European haplogroup H (WITTKOPP *et al.* 2013). This is likely because haplogroup U has lower ROS production compared to haplogroup H due to a more uncoupled respiratory chain. Importantly, mitochondrial mutations are also dependent on the genomic context in which they arose. In certain haplogroups, a mitochondrial variant in NADH dehydrogenase subunit 1 is associated with the degenerative disease Leber hereditary optic neuropathy. However, in other central Asian haplogroups this same mutation is significantly linked with high-altitude adaption (JI *et al.* 2012; WALLACE and CHALKIA 2013). These results suggest that although haplogroups may be protective in one environment and were adaptive to ancestral conditions (for example, where malaria is prevalent) they may also carry negative costs later in life or in modern environments (WALLACE and FAN 2010).

Cytoplasmic variation in plants can also have a profound impact on disease susceptibility. Until the early 1970s, the Texas cytoplasm (*cms-T*) was widely used in maize to speed up the process of hybrid seed production by circumventing manual detasseling. In *cms-T* lines, no pollen is produced due to the recombination-mediated formation of an aberrant open reading frame called *urf13-T* (LEVINGS 1990). However, it was later found that the presence of *urf13-T* causes hypersensitivity to the fungal disease Southern corn leaf blight. Therefore, *cms-T* lines are no longer used as a source of CMS in hybrid maize breeding. These results indicate that plant mtDNA sequence variation can directly alter stress responses similar to what is seen in humans. Future work should continue to explore the functional consequences of naturally occurring mitochondrial sequence variation in plants.

6 The roles of mitochondria in the integration of development and stress response

Though some effects of stress on plant development like stunted growth might be obvious, others like early flowering are at first counterintuitive. But early flowering is a common response for plants under stress, presumably to enable survival of the species by generating durable offspring. Mitochondria as a signaling hub are interconnected with or even involved in the regulation of both developmental reactions, and certain plant mitochondrial mutants can indeed show e.g. stunted growth and/or early flowering. Particularly, early flowering is a possible consequence of H₂O₂ production due to electron leakage by altered expression of mETC components (LI *et al.* 2014). Growth defects can also result when the stability of mitochondrial DNA is impaired, as in mutants of RECA and POLIB, defective in DNA repair or replication (PARENT *et al.* 2011; MILLER-MESSMER *et al.* 2012; CUPP and NIELSEN 2013).

6.1 Common regulation between stress and development

Mitochondria are canonically known as the “powerhouse” of the cell. Therefore, it is not surprising that key players integrate plant growth and stress responses via energy signaling and modulating. SnRK1 (Sucrose-non-fermenting-1-related protein kinase-1) and TOR (target of rapamycin) are two such main players, acting towards energy

conservation by controlling growth and metabolic reprogramming in response to stress (TOMÉ *et al.* 2014; reviewed in LASTDRAGER *et al.* 2014). Integration of plant growth and stress also takes place on the transcriptional level, for example by the ROS-inducible transcription factor WRKY15 (VANDERAUWERA *et al.* 2012), as mentioned in section 5.2. And as a prime example, CDKE1 (Cyclin-dependent kinase E1), which can regulate AOX expression, is described as a cellular switch between normal growth and stress response (NG *et al.* 2013). Another indication that both plant development and stress response require specialized and possibly interconnected mitochondria involvement came from transcriptomic studies on meiotic cells and heat-exposed plants - highly elevated levels of polyadenylated mitochondrial transcripts were detected in both sample types (ADAMO *et al.* 2008; CHEN *et al.* 2010; DUKOWIC-SCHULZE *et al.* 2014a; b) which are rather uncommon because they are usually promptly degraded.

6.2 Cross-talk via hormones and ROS

Plant hormones play an important role in both stress response and development, frequently interacting with ROS signaling (reviewed in (GAPPER and DOLAN 2006; KWAK *et al.* 2006; reviewed in FUJITA *et al.* 2006; SWANSON and GILROY 2010)). This includes intentional ROS production as well as the avoidance of ROS by inducing AOX expression. ROS are produced as signals in growth and stress response by dedicated NADPH oxidases, RBOHs (respiratory burst oxidase homologues) (reviewed in (MITTLER *et al.* 2004; SUZUKI *et al.* 2011)). On the other hand, multiple plant hormones have been reported to induce AOX expression, including salicylic acid (SA), abscisic acid (ABA), methyl salicylate, methyl jasmonate, and ethylene (LENNON *et al.* 1997; FUNG *et al.* 2004; EDERLI *et al.* 2006; GIRAUD *et al.* 2009; LIU *et al.* 2010; WANG *et al.* 2010b). An interesting example implicated in specialized mitochondrial AOX expression is SA, which plays a role in biotic stress response, triggering the expression of pathogenesis-related proteins. Similar promoter elements as in these SA-controlled pathogenesis-related proteins are present in AOX in voodoo lily (RHOADS and MCINTOSH 1993), where AOX is expressed in reproductive tissue for thermogenesis ((RHOADS and MCINTOSH 1992) and reviewed in (ZHU *et al.* 2011)). In addition, SA induces AOX leading to a cytosolic Ca^{2+} increase (KAWANO *et al.* 1998) which might be important for

reproductive growth since e.g. (i) Ca^{2+} -ATPase involvement in inflorescence architecture and vegetative development has been reported in Arabidopsis and tomato (GEORGE *et al.* 2008; WANG *et al.* 2011), and (ii) calcium distribution has been proposed as the trigger for PCD in litchi flower gender determination (WANG *et al.* 2006).

Taken together, mitochondria act as a signaling hub and metabolic effector between normal growth and stress responses, informed by and in turn producing and implementing a plethora of signals (Figure 3). Summing up, contrasting an outdated simplistic view on mitochondria, it is not just energy that is needed, but also energy dissipation; and ROS are not just triggering PCD but also sophisticated downstream pathways in development.

7 Perspectives

In humans, there are strong evolutionary connections between mitotypes and climatic adaptation as well as correlations between heteroplasmy and disease. Evidence now exists in several plant species for differences in the abundance of different chromosomes or subgenomic fragments, but the breadth of genetic variation (mitotypes) and extent of heteroplasmy in natural populations versus cultivated populations and their functional significance is widely unknown. For instance, in cucumber, the large 1.6Mb “master circle” chromosome contains the entire gene content. Two smaller ~84kb and 45kb rings do not carry known coding sequences, but are much more abundant, may replicate on their own, and may serve regulatory roles (ALVERSON *et al.* 2011). Now that abundant mitochondrial sequences are now available, future work should push towards understanding the functional differences.

A great technical challenge that must be overcome is the ability to resolve native mtDNA structures from limited input tissue. Plant organellar genome sequencing is primarily performed on leaf tissue in order to isolate an abundant amount of purified organelles. A caveat of this method is that the genomic structures present in different somatic tissue types may be very different from the structure of the genome that is maintained in the

meristem and inherited across generations. For instance in Arabidopsis, as mentioned earlier, mitochondria fuse and form large tentaculate structures in the meristem during cell division, which facilitates mixing of their heterogeneous genomes (SEGÚ-SIMARRO *et al.* 2008; SEGÚ-SIMARRO and STAEHELIN 2009). Moreover, the gross physical structure of the genomes may change depending on conditions. For instance, in budding yeast, circular structures predominate during bud formation and in the daughter cells, while linear concatamers predominate in mother cells (reviewed in (SOLIERI 2010)). Therefore, improved organellar isolation and sequencing approaches are needed to elucidate mitochondrial genomes from low amounts of input DNA.

Figure Legends

Figure 1. Generation of cybrids and alloplasmic lines. Protoplast fusion is used to generate symmetric (A) and asymmetric (B) cybrids. Circles represent protoplast plant cells (lacking cell wall). The outer and inner circle colors represent the genetic identities of the cytoplasmic and nuclear genomes, respectively. The top row illustrates the native pairings of the nuclear and cytoplasmic genomes. The bottom row represents the result of the cybridization. For symmetric cybrids, two distinct genotypes are fused to generate a cybrid that results in mixing of both the nuclear and cytoplasmic contents (A). To generate asymmetric cybrids, the distinct genotypes are first treated with either radiation or chemicals to damage or inhibit the transfer of either the nuclear or cytoplasmic content (middle row, B), then fused to generate a cybrid with the nuclear content from one genotype mismatched with the cytoplasmic content of the second genotype (bottom row, B). Alloplasmic lines are generated through a repeated backcrossing scheme (C). Two genotypes are crossed to generate an F1 individual that has mixed nuclear genome content, but has inherited the cytoplasm from the maternal parent (assuming maternal inheritance of the cytoplasmic genomes). Recurrent backcrossing to the second genotype “purifies” the nuclear genome content with each cross, until the progeny have nearly 100% of the genetic content from one genotype mismatched with the cytoplasm from the other. Crossing several maternal donors to the same nuclear donor can generate a collection of alloplasmic lines with distinct nuclear-cytoplasmic mismatches (D).

Figure 2. Transmission of organelles in animals versus plants. Animal cells are represented as circles (A) and plant cells are represented as rounded rectangles (B). In both cases, the outer circle color depicts the cytoplasm and the inner circle represents the nucleus. Crosses are shown between a female (left; red/orange color code) and male (right; gray scale) and the resulting progeny at the bottom. In most animals, including humans, mitochondria (yellow stars) are maternally derived and the nuclear genome content is a mixture of male and female alleles (A). Plants contain both mitochondria and plastids (green ovals), which can be transmitted in a number of ways (B). Most plants inherit both organelles from their mother (left); however, there are cases of strict paternal inheritance (middle; e.g. redwood trees), and bi-parental inheritance (right; e.g. cucumber).

Figure 3. Coordination of plant development and stress response via mitochondria. A simplistic overview on the main factors and key signals integrating plant development and stress response via mitochondria. ATP symbolizes the energy production and PCD the cell death role of mitochondria. Key signals involved in perception as well as transducing and reacting to environmental and cellular conditions are calcium ions (Ca^{2+}), plant hormones, carbon status and ROS.

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References

ADAMO A., PINNEY J. W., KUNOVA A., WESTHEAD D. R., MEYER P., 2008 Heat stress enhances the accumulation of polyadenylated mitochondrial transcripts in *Arabidopsis thaliana*. PLoS One 3: e2889.

1145 AKEN O. VAN, GIRAUD E., CLIFTON R., WHELAN J., 2009 Alternative oxidase: A target
1146 and regulator of stress responses. *Physiol. Plant.* **137**: 354–361.

1147 AKSYONOVA E., SINYAVSKAYA M., DANILENKO N., PERSHINA L., NAKAMURA C.,
1148 DAVYDENKO O., 2005 Heteroplasmy and paternally oriented shift of the organellar
1149 DNA composition in barley-wheat hybrids during backcrosses with wheat parents.
1150 *Genome* **48**: 761–9.

1151 ALLEN J. O., 2005 Effect of teosinte cytoplasmic genomes on maize phenotype. *Genetics*
1152 **169**: 863–80.

1153 ALMEIDA A. M., JARMUSZKIEWICZ W., KHOMSI H., ARRUDA P., VERCESI A. E., SLUSE F.
1154 E., 1999 Cyanide-resistant, ATP-synthesis-sustained, and Uncoupling-Protein-
1155 sustained respiration during postharvest ripening of tomato fruit. *Plant Physiol.* **119**:
1156 1323–1330.

1157 ALVERSON A. J., RICE D. W., DICKINSON S., BARRY K., PALMER J. D., 2011 Origins and
1158 recombination of the bacterial-sized multichromosomal mitochondrial genome of
1159 cucumber. *Plant Cell* **23**: 2499–513.

1160 AMIRSADEGHI S., ROBSON C. a., VANLERBERGHE G. C., 2007 The role of the
1161 mitochondrion in plant responses to biotic stress. *Physiol. Plant.* **129**: 253–266.

1162 AN G., YI J., MOON S., LEE Y.-S., ZHU L., LIANG W., ZHANG D.-B., JUNG K.-H., 2015
1163 Defective Tapetum Cell Death 1 (DTC1) Regulates ROS Levels by Binding to
1164 Metallothionein during Tapetum Degeneration. *Plant Physiol.*: pp.15.01561–.

1165 ARCHIBALD J. M., 2015 Endosymbiosis and Eukaryotic Cell Evolution. *Curr. Biol.* **25**:
1166 R911–R921.

1167 ARIMURA S., YAMAMOTO J., AIDA G. P., NAKAZONO M., TSUTSUMI N., 2004 Frequent
1168 fusion and fission of plant mitochondria with unequal nucleoid distribution. *Proc.*
1169 *Natl. Acad. Sci. U. S. A.* **101**: 7805–8.

1170 ARRIETA-MONTIEL M. P., SHEDGE V., DAVILA J., CHRISTENSEN A. C., MACKENZIE S. A.,
1171 2009 Diversity of the Arabidopsis mitochondrial genome occurs via nuclear-
1172 controlled recombination activity. *Genetics* **183**: 1261–8.

1173 ATKINSON N. J., URWIN P. E., 2012 The interaction of plant biotic and abiotic stresses:
1174 from genes to the field. *J. Exp. Bot.* **63**: 3523–43.

1175 ATTIA H., ARNAUD N., KARRAY N., LACHAÂL M., 2008 Long-term effects of mild salt

1176 stress on growth, ion accumulation and superoxide dismutase expression of
 1177 *Arabidopsis* rosette leaves. *Physiol. Plant.* **132**: 293–305.

1178 BAILEY-SERRES J., LEROY P., JONES S. S., WAHLEITHNER J. a., WOLSTENHOLME D. R.,
 1179 1987 Size distributions of circular molecules in plant mitochondrial DNAs. *Curr.*
 1180 *Genet.* **12**: 49–53.

1181 BAL A. K., DE D. N., 1961 Developmental changes in the submicroscopic morphology of
 1182 the cytoplasmic components during microsporogenesis in *Tradescantia*. *Dev. Biol.*
 1183 **3**: 241–254.

1184 BASU U., GOOD A. G., TAYLOR G. J., 2001 Transgenic *Brassica napus* plants
 1185 overexpressing aluminium-induced mitochondrial manganese superoxide dismutase
 1186 cDNA are resistant to aluminium. *Plant, Cell Environ.* **24**: 1278–1269.

1187 BEGZY K., MARIANO E. D., MATTIELLO L., NUNES A. V., MAZZAFERA P., MAIA I. G.,
 1188 MENOSSI M., 2011 An *Arabidopsis* mitochondrial uncoupling protein confers
 1189 tolerance to drought and salt stress in transgenic tobacco plants. *PLoS One* **6**.

1190 BELL P. R., MÜHLETHALER K., 1964 The degeneration and reappearance of mitochondria
 1191 in the egg cells of a plant. *J. Cell Biol.* **20**: 235–248.

1192 BENDICH A. J., 1996 Structural analysis of mitochondrial DNA molecules from fungi and
 1193 plants using moving pictures and pulsed-field gel electrophoresis. *J. Mol. Biol.* **255**:
 1194 564–88.

1195 BENTOLILA S., STEFANOV S., 2012 A Reevaluation of Rice Mitochondrial Evolution
 1196 Based on the Complete Sequence of Male-Fertile and Male-Sterile Mitochondrial
 1197 Genomes. *Plant Physiol.* **158**: 996–1017.

1198 BEREITER-HAHN J., VÖTH M., 1994 Dynamics of mitochondria in living cells: shape
 1199 changes, dislocations, fusion, and fission of mitochondria. *Microsc. Res. Tech.* **27**:
 1200 198–219.

1201 BERETERBIDE A., HERNOULD M., FARBOS I., GLIMELIUS K., MOURAS A., 2002
 1202 Restoration of stamen development and production of functional pollen in an
 1203 alloplasmic CMS tobacco line by ectopic expression of the *Arabidopsis thaliana*
 1204 SUPERMAN gene. *Plant J.* **29**: 607–615.

1205 BERTHOMÉ R., THOMASSET M., MAENE M., BOURGEOIS N., FROGER N., BUDAR F., 2008
 1206 *pur4* mutations are lethal to the male, but not the female, gametophyte and affect

1207 sporophyte development in *Arabidopsis*. Plant Physiol. **147**: 650–660.

1208 BIRD J., PORTER E. K., DICKINSON H. G., 1983 Events in the cytoplasm during male
1209 meiosis in *Lilium*. J Cell Sci **59**: 27–42.

1210 BLEIN S., BARDEL C., DANJEAN V., MCGUFFOG L., HEALEY S., BARROWDALE D., LEE
1211 A., DENNIS J., KUCHENBAECKER K. B., SOUCY P., TERRY M. B., CHUNG W. K.,
1212 GOLDGAR D. E., BUYS S. S., JANAVICIUS R., TIHOMIROVA L., TUNG N., DORFLING
1213 C. M., RENSBURG E. J. VAN, NEUHAUSEN S. L., DING Y. C., GERDES A.-M.,
1214 EJLERTSEN B., NIELSEN F. C., HANSEN T. V., OSORIO A., BENITEZ J., ANDRÉS-
1215 CONEJERO R., SEGOTA E., WEITZEL J. N., THELANDER M., PETERLONGO P., RADICE
1216 P., PENSOTTI V., DOLCETTI R., BONANNI B., PEISSEL B., ZAFFARONI D., SCUVERA
1217 G., MANOUKIAN S., VARESCO L., CAPONE G. L., PAPI L., OTTINI L., YANNOUKAKOS
1218 D., KONSTANTOPOULOU I., GARBER J., HAMANN U., DONALDSON A., BRADY A.,
1219 BREWER C., FOO C., EVANS D. G., FROST D., ECCLES D., DOUGLAS F., COOK J.,
1220 ADLARD J., BARWELL J., WALKER L., IZATT L., SIDE L. E., KENNEDY M. J.,
1221 TISCHKOWITZ M., ROGERS M. T., PORTEOUS M. E., MORRISON P. J., PLATTE R.,
1222 EELES R., DAVIDSON R., HODGSON S., COLE T., GODWIN A. K., ISAACS C., CLAES
1223 K., LEENEER K. DE, MEINDL A., GEHRIG A., WAPPENSCHMIDT B., SUTTER C.,
1224 ENGEL C., NIEDERACHER D., STEINEMANN D., PLENDL H., KAST K., RHIEM K.,
1225 DITSCH N., ARNOLD N., VARON-MATEEVA R., SCHMUTZLER R. K., PREISLER-
1226 ADAMS S., MARKOV N. B., WANG-GOHRKE S., PAUW A. DE, LEFOL C., LASSET C.,
1227 LEROUX D., ROULEAU E., DAMIOLA F., DREYFUS H., BARJHOUX L., GOLMARD L.,
1228 UHRHAMMER N., BONADONA V., SORNIN V., BIGNON Y.-J., CARTER J., LE L. VAN,
1229 PIEDMONTE M., DiSILVESTRO P. A., LA HOYA M. DE, CALDES T., NEVANLINNA H.,
1230 AITTO MÄKI K., JAGER A., OUWELAND A. M. VAN DEN, KETS C. M., AALFS C. M.,
1231 LEEUWEN F. E. VAN, HOGERVORST F. B., MEIJERS-HEIJBOER H. E., OOSTERWIJK J.
1232 C., ROOZENDAAL K. E. VAN, ROOKUS M. A., DEVILEE P., LUIJT R. B. VAN DER,
1233 OLAH E., DIEZ O., TEULÉ A., LAZARO C., BLANCO I., VALLE J. DEL, JAKUBOWSKA
1234 A., SUKIENNICKI G., GRONWALD J., LUBINSKI J., DURDA K., JAWORSKA-BIENIEK K.,
1235 AGNARSSON B. A., MAUGARD C., AMADORI A., MONTAGNA M., TEIXEIRA M. R.,
1236 SPURDLE A. B., FOULKES W., OLSWOLD C., LINDOR N., PANKRATZ V. S., SZABO C.
1237 I., LINCOLN A., JACOBS L., CORINES M., ROBSON M., VIJAI J., BERGER A., FINK-

1238 RETTER A., SINGER C. F., RAPPAPORT C., KAULICH D. G., PFEILER G., TEA M.-K.,
 1239 GREENE M. H., MAI P. L., RENNERT G., IMYANITOV E. N., MULLIGAN A. M.,
 1240 GLENDON G., ANDRULIS I. L., TCHATCHOU S., TOLAND A. E., PEDERSEN I. S.,
 1241 THOMASSEN M., KRUSE T. A., JENSEN U. B., CALIGO M. A., FRIEDMAN E., ZIDAN
 1242 J., LAITMAN Y., LINDBLOM A., MELIN B., ARVER B., LOMAN N., ROSENQUIST R.,
 1243 OLOPADE O. I., NUSSBAUM R. L., RAMUS S. J., NATHANSON K. L., DOMCHEK S. M.,
 1244 REBBECK T. R., ARUN B. K., MITCHELL G., KARLAN B. Y., LESTER J., ORSULIC S.,
 1245 STOPPA-LYONNET D., THOMAS G., SIMARD J., COUCH F. J., OFFIT K., EASTON D. F.,
 1246 CHENEVIX-TRENCH G., ANTONIOU A. C., MAZOYER S., PHELAN C. M., SINILNIKOVA
 1247 O. M., COX D. G., 2015 An original phylogenetic approach identified mitochondrial
 1248 haplogroup T1a1 as inversely associated with breast cancer risk in BRCA2 mutation
 1249 carriers. *Breast Cancer Res.* **17**: 61.

1250 BOUTHOUR D., KALAI T., CHAFFEI H. C., GOUIA H., CORPAS F. J., 2015 Differential
 1251 response of NADP-dehydrogenases and carbon metabolism in leaves and roots of
 1252 two durum wheat (*Triticum durum* Desf.) cultivars (Karim and Azizi) with different
 1253 sensitivities to salt stress. *J. Plant Physiol.* **179**: 56–63.

1254 BRANDALISE M., MAIA I. G., BORECKÝ J., VERCESI A. E., ARRUDA P., 2003
 1255 Overexpression of Plant Uncoupling Mitochondrial Protein in Transgenic Tobacco
 1256 Increases Tolerance to Oxidative Stress. *J. Bioenerg. Biomembr.* **35**: 203–209.

1257 BRAS M., QUEENAN B., SUSIN S. A., 2005 Programmed cell death via mitochondria:
 1258 Different modes of dying. *Biochem.* **70**: 231–239.

1259 BROWN R. C., LEMMON B. E., 2001 The cytoskeleton and spatial control of cytokinesis in
 1260 the plant life cycle. *Protoplasma* **215**: 35–49.

1261 BROWNFIELD L., YI J., JIANG H., MININA E. A., TWELL D., KÖHLER C., 2015 Organelles
 1262 maintain spindle position in plant meiosis. *Nat. Commun.* **6**: 6492.

1263 BUDAR F., PELLETIER G., 2001 Male sterility in plants: occurrence, determinism,
 1264 significance and use. *Comptes Rendus l'Académie des Sci. - Ser. III - Sci. la Vie*
 1265 **324**: 543–550.

1266 CABRERA-OREFICE A., GUERRERO-CASTILLO S., LUÉVANO-MARTÍNEZ L. A., PEÑA A.,
 1267 URIBE-CARVAJAL S., 2010 Mitochondria from the salt-tolerant yeast *Debaryomyces*
 1268 *hansenii* (halophilic organelles?). *J. Bioenerg. Biomembr.* **42**: 11–19.

1269 CANTER J. A., KALLIANPUR A. R., PARL F. F., MILLIKAN R. C., 2005 Mitochondrial
1270 DNA G10398A polymorphism and invasive breast cancer in African-American
1271 women. *Cancer Res.* **65**: 8028–33.

1272 CAO J., SCHNEEBERGER K., OSSOWSKI S., GÜNTHER T., BENDER S., FITZ J., KOENIG D.,
1273 LANZ C., STEGLE O., LIPPERT C., WANG X., OTT F., MÜLLER J., ALONSO-BLANCO
1274 C., BORGWARDT K., SCHMID K. J., WEIGEL D., 2011 Whole-genome sequencing of
1275 multiple *Arabidopsis thaliana* populations. *Nat. Genet.* **43**: 956–63.

1276 CASTRO I. P. DE, MARTINS L. M., TUFI R., 2010 Mitochondrial quality control and
1277 neurological disease: an emerging connection. *Expert Rev. Mol. Med.* **12**: e12.

1278 CHAN D. C., 2006 Mitochondria: dynamic organelles in disease, aging, and development.
1279 *Cell* **125**: 1241–52.

1280 CHANG S., YANG T., DU T., HUANG Y., CHEN J., YAN J., HE J., GUAN R., 2011
1281 Mitochondrial genome sequencing helps show the evolutionary mechanism of
1282 mitochondrial genome formation in *Brassica*. *BMC Genomics* **12**: 497.

1283 CHEN C.-B., XU Y.-Y., MA H., CHONG K., 2005 Cell Biological Characterization of Male
1284 Meiosis and Pollen Development in Rice. *J. Integr. Plant Biol.* **47**: 734–744.

1285 CHEN C., FARMER A. D., LANGLEY R. J., MUDGE J., CROW J. A., MAY G. D., HUNTLEY
1286 J., SMITH A. G., RETZEL E. F., 2010 Meiosis-specific gene discovery in plants:
1287 RNA-Seq applied to isolated *Arabidopsis* male meiocytes. *BMC Plant Biol.* **10**: 280.

1288 CHEN J., GUAN R., CHANG S., DU T., ZHANG H., XING H., 2011 Substoichiometrically
1289 different mitotypes coexist in mitochondrial genomes of *Brassica napus* L. *PLoS*
1290 *One* **6**: e17662.

1291 CHEN L., LIU Y.-G., 2014 Male sterility and fertility restoration in crops. *Annu. Rev.*
1292 *Plant Biol.* **65**: 579–606.

1293 CHIVASA S., 1998 Cyanide Restores N GeneMediated Resistance to Tobacco Mosaic
1294 Virus in Transgenic Tobacco Expressing Salicylic Acid Hydroxylase. *PLANT*
1295 *CELL ONLINE* **10**: 1489–1498.

1296 CHRISTENSEN C. A., GORSICH S. W., BROWN R. H., JONES L. G., BROWN J., SHAW J. M.,
1297 DREWS G. N., 2002 Mitochondrial GFA2 is required for synergid cell death in
1298 *Arabidopsis*. *Plant Cell* **14**: 2215–2232.

1299 CLIFTON S. W., MINX P., FAURON C. M.-R., GIBSON M., ALLEN J. O., SUN H.,

1300 THOMPSON M., BARBAZUK W. B., KANUGANTI S., TAYLOE C., MEYER L., WILSON
1301 R. K., NEWTON K. J., 2004 Sequence and comparative analysis of the maize NB
1302 mitochondrial genome. *Plant Physiol.* **136**: 3486–3503.

1303 CLIFTON R., MILLAR A. H., WHELAN J., 2006 Alternative oxidases in *Arabidopsis*: a
1304 comparative analysis of differential expression in the gene family provides new
1305 insights into function of non-phosphorylating bypasses. *Biochim. Biophys. Acta*
1306 **1757**: 730–741.

1307 COLOMBATTI F., GONZALEZ D. H., WELCHEN E., 2014 Plant mitochondria under
1308 pathogen attack: a sigh of relief or a last breath? *Mitochondrion* **19 Pt B**: 238–44.

1309 CONSIDINE M. J., DALEY D. O., WHELAN J., 2001 The expression of alternative oxidase
1310 and uncoupling protein during fruit ripening in mango. *Plant Physiol.* **126**: 1619–
1311 1629.

1312 CRAMER G. R., URANO K., DELROT S., PEZZOTTI M., SHINOZAKI K., 2011 Effects of
1313 abiotic stress on plants: a systems biology perspective. *BMC Plant Biol.* **11**: 163.

1314 CROSATTI C., QUANSAH L., MARÉ C., GIUSTI L., RONCAGLIA E., ATIENZA S. G.,
1315 CATTIVELLI L., FAIT A., 2013 Cytoplasmic genome substitution in wheat affects the
1316 nuclear-cytoplasmic cross-talk leading to transcript and metabolite alterations. *BMC*
1317 *Genomics* **14**: 868.

1318 CUGUEN J., WATTIER R., SAUMITOU-LAPRADE P., FORCIOLI D., MÖRCHEN M., DIJK H.
1319 VAN, VERNET P., 1994 Gynodioecy and mitochondrial DNA polymorphism in
1320 natural populations of *Beta vulgaris* ssp *maritima*. *Genet. Sel. Evol.* **26**: S87.

1321 CUPP J. D., NIELSEN B. L., 2013 *Arabidopsis thaliana* organellar DNA polymerase IB
1322 mutants exhibit reduced mtDNA levels with a decrease in mitochondrial area
1323 density. *Physiol. Plant.* **149**: 91–103.

1324 DAI H., LO Y.-S., JANE W.-N., LEE L.-W., CHIANG K.-S., 1998 Population heterogeneity
1325 of higher-plant mitochondria in structure and function. *Eur. J. Cell Biol.* **75**: 198–
1326 209.

1327 DAVILA J. I., ARRIETA-MONTIEL M. P., WAMBOLDT Y., CAO J., HAGMANN J., SHEDGE
1328 V., XU Y.-Z., WEIGEL D., MACKENZIE S. A., 2011 Double-strand break repair
1329 processes drive evolution of the mitochondrial genome in *Arabidopsis*. *BMC Biol.*
1330 **9**: 64.

1331 DEKKERS B. J. W., PEARCE S., BOLDEREN-VELDKAMP R. P. van, MARSHALL A., WIDERA
 1332 P., GILBERT J., DROST H.-G., BASSEL G. W., MÜLLER K., KING J. R., WOOD A. T.
 1333 A., GROSSE I., QUINT M., KRASNOGOR N., LEUBNER-METZGER G., HOLDSWORTH
 1334 M. J., BENTSINK L., 2013 Transcriptional dynamics of two seed compartments with
 1335 opposing roles in Arabidopsis seed germination. *Plant Physiol.* **163**: 205–215.
 1336 DELPH L. F., TOUZET P., BAILEY M. F., 2007 Merging theory and mechanism in studies
 1337 of gynodioecy. *Trends Ecol. Evol.* **22**: 17–24.
 1338 DEWEY R. E., TIMOTHY D. H., LEVINGS C. S., 1991 Chimeric mitochondrial genes
 1339 expressed in the C male-sterile cytoplasm of maize. *Curr. Genet.* **20**: 475–82.
 1340 DICKINSON D. B., 1965 Germination of lily pollen: {Respiration} and tube growth.
 1341 *Science* (80-.). **150**: 1818–1819.
 1342 DICKINSON D. B., 1966 Inhibition of pollen respiration by oligomycin. **210**: 1362–1363.
 1343 DICKINSON H. G., POTTER U., 1978 Cytoplasmic changes accompanying the female
 1344 meiosis in *Lilium longiflorum* Thunb . *J Cell Sci* **29**: 147–169.
 1345 DICKINSON H. G., LI F. L., 1988 Organelle behavior during higher plant gametogenesis.
 1346 In: *The Division and Segregation of Organelles*, Cambridge University Press.
 1347 DUCOS E., TOUZET P., BOUTRY M., 2001 The male sterile G cytoplasm of wild beet
 1348 displays modified mitochondrial respiratory complexes. *Plant J.* **26**: 171–80.
 1349 DUKOWIC-SCHULZE S., HARRIS A., LI J., SUNDARARAJAN A., MUDGE J., RETZEL E.,
 1350 PAWLOWSKI W., CHEN C., 2014a Comparative transcriptomics of early meiosis in
 1351 Arabidopsis and maize. *J. Genet. Genomics* **41**: 139–152.
 1352 DUKOWIC-SCHULZE S., SUNDARARAJAN A., MUDGE J., RAMARAJ T., FARMER A. D.,
 1353 WANG M., SUN Q., PILLARDY J., KIANIAN S., RETZEL E. F., PAWLOWSKI W. P.,
 1354 CHEN C., 2014b The transcriptome landscape of early maize meiosis. *BMC Plant*
 1355 *Biol.* **14**: 118.
 1356 EDERLI L., MORETTINI R., BORGOGNI A., WASTERNAK C., MIERSCH O., REALE L.,
 1357 FERRANTI F., TOSTI N., PASQUALINI S., 2006 Interaction between nitric oxide and
 1358 ethylene in the induction of alternative oxidase in ozone-treated tobacco plants.
 1359 *Plant Physiol.* **142**: 595–608.
 1360 EHRENSHAFT M., BRAMBL R., 1990 Respiration and mitochondrial biogenesis in
 1361 germinating embryos of maize. *Plant Physiol.* **93**: 295–304.

1362 ELANSARY H. O., MÜLLER K., OLSON M. S., STORCHOVÁ H., 2010 Transcription profiles
 1363 of mitochondrial genes correlate with mitochondrial DNA haplotypes in a natural
 1364 population of *Silene vulgaris*. *BMC Plant Biol.* **10**: 11.

1365 ELLIOTT H. R., SAMUELS D. C., EDEN J. A., RELTON C. L., CHINNERY P. F., 2008
 1366 Pathogenic mitochondrial DNA mutations are common in the general population.
 1367 *Am. J. Hum. Genet.* **83**: 254–60.

1368 EPSTEIN C. B., WADDLE J. A., HALE IV W., DAVÉ V., THORNTON J., MACATEE T. L.,
 1369 GARNER H. R., BUTOW R. A., 2001 Genome-wide responses to mitochondrial
 1370 dysfunction. *Mol. Biol. Cell* **12**: 297–308.

1371 ESWARAN N., PARAMESWARAN S., SATHRAM B., ANANTHARAMAN B., KUMAR G R. K.,
 1372 TANGIRALA S. J., 2010 Yeast functional screen to identify genetic determinants
 1373 capable of conferring abiotic stress tolerance in *Jatropha curcas*. *BMC Biotechnol.*
 1374 **10**: 23.

1375 FLAGELLA Z., TRONO D., POMPA M., FONZO N. DI, PASTORE D., 2006 Seawater stress
 1376 applied at germination affects mitochondrial function in durum wheat (*Triticum*
 1377 *durum*) early seedlings. *Funct. Plant Biol.* **33**: 357–366.

1378 FOYER C. H., NOCTOR G., 2005 Redox homeostasis and antioxidant signaling: a
 1379 metabolic interface between stress perception and physiological responses. *Plant*
 1380 *Cell* **17**: 1866–1875.

1381 FRIEDRICH P., 1985 Dynamic compartmentation in soluble multienzyme systems. In:
 1382 *Organized multienzyme systems*, Academic Press, New York, pp. 141–176.

1383 FUJIE M., KUROIWA H., KAWANO S., KUROIWA T., 1993 Studies on the behavior of
 1384 organelles and their nucleoids in the root apical meristem of *Arabidopsis thaliana* L.
 1385 *Col. Planta* **189**: 443–452.

1386 FUJIE M., KUROIWA H., KAWANO S., MUTOH S., KUROIWA T., 1994 Behavior of
 1387 organelles and their nucleoids in the shoot apical meristem during leaf development
 1388 in *Arabidopsis thaliana* L. *Planta* **194**: 395–405.

1389 FUJITA M., FUJITA Y., NOUTOSHI Y., TAKAHASHI F., NARUSAKA Y., YAMAGUCHI-
 1390 SHINOZAKI K., SHINOZAKI K., 2006 Crosstalk between abiotic and biotic stress
 1391 responses: a current view from the points of convergence in the stress signaling
 1392 networks. *Curr. Opin. Plant Biol.* **9**: 436–42.

- 1393 FUNG R. W. M., WANG C. Y., SMITH D. L., GROSS K. C., TIAN M., 2004 MeSA and
1394 MeJA increase steady-state transcript levels of alternative oxidase and resistance
1395 against chilling injury in sweet peppers (*Capsicum annuum* L.). Plant Sci. **166**: 711–
1396 719.
- 1397 GALLUZZI L., KEPP O., TROJEL-HANSEN C., KROEMER G., 2012 Mitochondrial control of
1398 cellular life, stress, and death. Circ. Res. **111**: 1198–207.
- 1399 GAPPER C., DOLAN L., 2006 Control of plant development by reactive oxygen species.
1400 Plant Physiol. **141**: 341–345.
- 1401 GECHEV T. S., BREUSEGEM F. VAN, STONE J. M., DENEV I., LALOI C., 2006 Reactive
1402 oxygen species as signals that modulate plant stress responses and programmed cell
1403 death. BioEssays **28**: 1091–1101.
- 1404 GEDDY R., MAHÉ L., BROWN G. G., 2005 Cell-specific regulation of a *Brassica napus*
1405 CMS-associated gene by a nuclear restorer with related effects on a floral homeotic
1406 gene promoter. Plant J. **41**: 333–345.
- 1407 GELHAYE E., ROUHIER N., GÉRARD J., JOLIVET Y., GUALBERTO J., NAVROT N., OHLSSON
1408 P.-I., WINGSLE G., HIRASAWA M., KNAFF D. B., WANG H., DIZENGREMEL P.,
1409 MEYER Y., JACQUOT J.-P., 2004 A specific form of thioredoxin h occurs in plant
1410 mitochondria and regulates the alternative oxidase. Proc Natl Acad Sci U S A **101**:
1411 14545–14550.
- 1412 GEORGE L., ROMANOWSKY S. M., HARPER J. F., SHARROCK R. A., 2008 The ACA10
1413 Ca²⁺-ATPase regulates adult vegetative development and inflorescence architecture
1414 in Arabidopsis. Plant Physiol. **146**: 716–728.
- 1415 GERHOLD J. M., AUN A., SEDMAN T., JÖERS P., SEDMAN J., 2010 Strand Invasion
1416 Structures in the Inverted Repeat of *Candida albicans* Mitochondrial DNA Reveal a
1417 Role for Homologous Recombination in Replication. Mol. Cell **39**: 851–861.
- 1418 GERSTEL D. U., BURNS J. A., BURK L. G., 1978 Cytoplasmic male sterility in *Nicotiana*,
1419 restoration of fertility, and the nucleolus. Genetics **89**: 157–169.
- 1420 GESTEL K. VAN, KÖHLER R. H., VERBELEN J.-P., 2002 Plant mitochondria move on {F}-
1421 actin, but their positioning in the cortical cytoplasm depends on both {F}-actin and
1422 microtubules. J. Exp. Bot. **53**: 659–667.
- 1423 GESTEL K. VAN, VERBELEN J.-P., 2002 Giant mitochondria are a response to low oxygen

1424 pressure in cells of tobacco (*Nicotiana tabacum* L.). *J. Exp. Bot.* **53**: 1215–8.

1425 GIRAUD E., AKEN O. Van, HO L. H. M., WHELAN J., 2009 The transcription factor ABI4
1426 is a regulator of mitochondrial retrograde expression of ALTERNATIVE OXIDASE
1427 1a. *Plant Physiol.* **150**: 1286–1296.

1428 GLEASON C., HUANG S., THATCHER L. F., FOLEY R. C., ANDERSON C. R., CARROLL A.
1429 J., MILLAR a H., SINGH K. B., 2011 Mitochondrial complex II has a key role in
1430 mitochondrial-derived reactive oxygen species influence on plant stress gene
1431 regulation and defense. *Proc. Natl. Acad. Sci. U. S. A.* **108**: 10768–73.

1432 GOLLDACK D., LI C., MOHAN H., PROBST N., 2014 Tolerance to drought and salt stress in
1433 plants: Unraveling the signaling networks. *Front. Plant Sci.* **5**: 151.

1434 GÓMEZ-DURÁN A., PACHEU-GRAU D., LÓPEZ-GALLARDO E., DíEZ-SÁNCHEZ C.,
1435 MONTAYA J., LÓPEZ-PÉREZ M. J., RUIZ-PESINI E., 2010 Unmasking the causes of
1436 multifactorial disorders: OXPHOS differences between mitochondrial haplogroups.
1437 *Hum. Mol. Genet.* **19**: 3343–53.

1438 GREEN B. R., 2011 Chloroplast genomes of photosynthetic eukaryotes. *Plant J.* **66**: 34–
1439 44.

1440 GREINER S., SOBANSKI J., BOCK R., 2015 Why are most organelle genomes transmitted
1441 maternally? *Bioessays* **37**: 80–94.

1442 GU J., MILES D., NEWTON K. J., 1993 Analysis of Leaf Sectors in the NCS6
1443 Mitochondrial Mutant of Maize. *Plant Cell* **5**: 963–971.

1444 GUARDADO-ESTRADA M., MEDINA-MARTÍNEZ I., JUÁREZ-TORRES E., ROMAN-BASSAURE
1445 E., MACÍAS L., ALFARO A., ALCÁNTARA-VÁZQUEZ A., ALONSO P., GOMEZ G.,
1446 CRUZ-TALONIA F., SERNA L., MUÑOZ-CORTEZ S., BORGES-IBÁÑEZ M., ESPINOSA
1447 A., KOFMAN S., BERUMEN J., 2012 The Amerindian mtDNA haplogroup B2
1448 enhances the risk of HPV for cervical cancer: de-regulation of mitochondrial genes
1449 may be involved. *J. Hum. Genet.* **57**: 269–76.

1450 GUERRERO-CASTILLO S., ARAIZA-OLIVERA D., CABRERA-OREFICE A., ESPINASA-
1451 JARAMILLO J., GUTIÉRREZ-AGUILAR M., LUÉVANO-MARTÍNEZ L. A., ZEPEDA-
1452 BASTIDA A., URIBE-CARVAJAL S., 2011 Physiological uncoupling of mitochondrial
1453 oxidative phosphorylation. Studies in different yeast species. *J. Bioenerg.*
1454 *Biomembr.* **43**: 323–31.

1455 GUZMÁN-FULGENCIO M., JIMÉNEZ J. L., GARCÍA-ÁLVAREZ M., BELLÓN J. M.,
 1456 FERNÁNDEZ-RODRIGUEZ A., CAMPOS Y., RODRÍGUEZ C., GONZÁLEZ-GARCIA J.,
 1457 RIERA M., VICIANA P., MUÑOZ-FERNÁNDEZ M. Á., RESINO S., 2013 Mitochondrial
 1458 haplogroups are associated with clinical pattern of AIDS progression in HIV-
 1459 infected patients. *J. Acquir. Immune Defic. Syndr.* **63**: 178–83.
 1460 HAMILTON C. A., GOOD A. G., TAYLOR G. J., 2001 Induction of vacuolar ATPase and
 1461 mitochondrial ATP synthase by aluminum in an aluminum-resistant cultivar of
 1462 wheat. *Plant Physiol.* **125**: 2068–2077.
 1463 HANDA H., 2003 The complete nucleotide sequence and RNA editing content of the
 1464 mitochondrial genome of rapeseed (*Brassica napus* L.): comparative analysis of the
 1465 mitochondrial genomes of rapeseed and *Arabidopsis thaliana*. *Nucleic Acids Res.*
 1466 **31**: 5907–16.
 1467 HANQING F., KUN S., MINGQUAN L., HONGYU L., XIN L., YAN L., YIFENG W., 2010 The
 1468 expression, function and regulation of mitochondrial alternative oxidase under biotic
 1469 stresses. *Mol. Plant Pathol.* **11**: 429–440.
 1470 HANSON M. R., FOLKERTS O., 1992 Structure and function of the higher plant
 1471 mitochondrial genome. *Int Rev Cytol* **141**: 129–172.
 1472 HAVEY M. J., 1997 Predominant Paternal Transmission of the Mitochondrial Genome in
 1473 Cucumber. *J. Hered.* **88**: 232–235.
 1474 HEAZLEWOOD J. L., VERBOOM R. E., TONTI-FILIPPINI J., SMALL I., MILLAR A. H., 2007
 1475 SUBA: The Arabidopsis subcellular database. *Nucleic Acids Res.* **35**.
 1476 HENZE K., MARTIN W., 2001 How do mitochondrial genes get into the nucleus? *Trends*
 1477 *Genet.* **17**: 383–7.
 1478 HOEKSTRA L. A., SIDDIQ M. A., MONTTOOTH K. L., 2013 Pleiotropic effects of a
 1479 mitochondrial-nuclear incompatibility depend upon the accelerating effect of
 1480 temperature in *Drosophila*. *Genetics* **195**: 1129–39.
 1481 HOU N., WU Y. W., LIU C. G., ZHANG C. L., ZHANG Y., 2000 [Studies of salt tolerance
 1482 of alloplasmic wheat]. *Yi Chuan Xue Bao* **27**: 325–30.
 1483 HU J., YI R., ZHANG H., DING Y., 2013 Nucleo-cytoplasmic interactions affect RNA
 1484 editing of *cox2*, *atp6* and *atp9* in alloplasmic male-sterile rice (*Oryza sativa* L.) lines.
 1485 *Mitochondrion* **13**: 87–95.

1486 HUANG J., STRUCK F., MATZINGER D. F., LEVINGS C. S. 3rd, 1994 Flower-enhanced
 1487 expression of a nuclear-encoded mitochondrial respiratory protein is associated with
 1488 changes in mitochondrion number. *Plant Cell* **6**: 439–448.

1489 HUANG C. Y., GRÜNHEIT N., AHMADINEJAD N., TIMMIS J. N., MARTIN W., 2005
 1490 Mutational decay and age of chloroplast and mitochondrial genomes transferred
 1491 recently to angiosperm nuclear chromosomes. *Plant Physiol.* **138**: 1723–33.

1492 HUNER N. P. ., ÖQUIST G., SARHAN F., 1998 Energy balance and acclimation to light and
 1493 cold. *Trends Plant Sci.* **3**: 224–230.

1494 HUNT M. D., NEWTON K. J., 1991 The NCS3 mutation: genetic evidence for the
 1495 expression of ribosomal protein genes in *Zea mays* mitochondria. *EMBO J.* **10**:
 1496 1045–52.

1497 IFTIKAR F. I., MACDONALD J. R., BAKER D. W., RENSHAW G. M. C., HICKEY A. J. R.,
 1498 2014 Could thermal sensitivity of mitochondria determine species distribution in a
 1499 changing climate? *J. Exp. Biol.* **217**: 2348–2357.

1500 ISHIBASHI Y., KODA Y., ZHENG S.-H., YUASA T., IWAYA-INOUE M., 2013 Regulation of
 1501 soybean seed germination through ethylene production in response to reactive
 1502 oxygen species. *Ann. Bot.* **111**: 95–102.

1503 ISHIHARA N., JOFUKU A., EURA Y., MIHARA K., 2003 Regulation of mitochondrial
 1504 morphology by membrane potential, and {DRP}1-dependent division and {FZO}1-
 1505 dependent fusion reaction in mammalian cells. *Biochem. Biophys. Res. Commun.*
 1506 **301**: 891–898.

1507 IWABUCHI M., KYOZUKA J., SHIMAMOTO K., 1993 Processing followed by complete
 1508 editing of an altered mitochondrial atp6 RNA restores fertility of cytoplasmic male
 1509 sterile rice. *EMBO J.* **12**: 1437–46.

1510 JACOBS M. A., PAYNE S. R., BENDICH A. J., 1996 Moving pictures and pulsed-field gel
 1511 electrophoresis show only linear mitochondrial DNA molecules from yeasts with
 1512 linear-mapping and circular-mapping mitochondrial genomes. *Curr. Genet.* **30**: 3–
 1513 11.

1514 JACOBY R. P., MILLAR a. H., TAYLOR N. L., 2010 Wheat mitochondrial proteomes
 1515 provide new links between antioxidant defense and plant salinity tolerance. *J.*
 1516 *Proteome Res.* **9**: 6595–6604.

- 1517 JACOBY R. P., TAYLOR N. L., MILLAR A. H., 2011 The role of mitochondrial respiration
1518 in salinity tolerance. *Trends Plant Sci.* **16**: 614–23.
- 1519 JACOBY R. P., MILLAR A. H., TAYLOR N. L., 2013 Investigating the role of respiration in
1520 plant salinity tolerance by analyzing mitochondrial proteomes from wheat and a
1521 salinity-tolerant Amphiploid (wheat × *Lophopyrum elongatum*). *J. Proteome Res.*
1522 **12**: 4807–29.
- 1523 JANOUSKOVEC J., SOBOTKA R., LAI D.-H., FLEGONTOV P., KONÍK P., KOMENDA J., ALI
1524 S., PRÁŠIL O., PAIN A., OBORNÍK M., LUKES J., KEELING P. J., 2013 Split
1525 photosystem protein, linear-mapping topology, and growth of structural complexity
1526 in the plastid genome of *Chromera velia*. *Mol. Biol. Evol.* **30**: 2447–62.
- 1527 JANSKA H., 1998 Stoichiometric Shifts in the Common Bean Mitochondrial Genome
1528 Leading to Male Sterility and Spontaneous Reversion to Fertility. *PLANT CELL*
1529 *ONLINE* **10**: 1163–1180.
- 1530 JARMUSZKIEWICZ W., 2001 Uncoupling proteins in mitochondria of plants and some
1531 microorganisms. *Acta Biochim. Pol.* **48**: 145–155.
- 1532 JARMUSZKIEWICZ W., CZARNA M., SLUSE-GOFFART C., SLUSE F. E., 2004 The
1533 contribution of uncoupling protein and ATP synthase to state 3 respiration in
1534 *Acanthamoeba castellanii* mitochondria. *Acta Biochim. Pol.* **51**: 533–538.
- 1535 JI F., SHARPLEY M. S., DERBENEVA O., ALVES L. S., QIAN P., WANG Y., CHALKIA D.,
1536 LVOVA M., XU J., YAO W., SIMON M., PLATT J., XU S., ANGELIN A., DAVILA A.,
1537 HUANG T., WANG P. H., CHUANG L.-M., MOORE L. G., QIAN G., WALLACE D. C.,
1538 2012 Mitochondrial DNA variant associated with Leber hereditary optic neuropathy
1539 and high-altitude Tibetans. *Proc. Natl. Acad. Sci.* **109**: 7391–7396.
- 1540 JI J., HUANG W., YIN C., GONG Z., 2013 Mitochondrial cytochrome c oxidase and F1Fo-
1541 ATPase dysfunction in peppers (*Capsicum annuum* L.) with cytoplasmic male
1542 sterility and its association with orf507 and Ψ atp6-2 genes. *Int. J. Mol. Sci.* **14**:
1543 1050–68.
- 1544 JIANG K., BALLINGER T., LI D., ZHANG S., FELDMAN L., 2006 A role for mitochondria in
1545 the establishment and maintenance of the maize root quiescent center. *Plant Physiol*
1546 **140**: 1118–1125.
- 1547 JING B., HENG S., TONG D., WAN Z., FU T., TU J., MA C., YI B., WEN J., SHEN J., 2012

1548 A male sterility-associated cytotoxic protein ORF288 in *Brassica juncea* causes
 1549 aborted pollen development. J. Exp. Bot. **63**: 1285–95.

1550 JOSEPH B., CORWIN J. A., LI B., ATWELL S., KLIEBENSTEIN D. J., 2013 Cytoplasmic
 1551 genetic variation and extensive cytonuclear interactions influence natural variation
 1552 in the metabolome. Elife **2**: e00776.

1553 KANEKO Y., BANG S. W., 2014 Interspecific and intergeneric hybridization and
 1554 chromosomal engineering of Brassicaceae crops. Breed. Sci. **64**: 14–22.

1555 KARPOVA O. V, KUZMIN E. V, ELTHON T. E., NEWTON K. J., 2002 Differential
 1556 expression of alternative oxidase genes in maize mitochondrial mutants. Plant Cell
 1557 **14**: 3271–84.

1558 KAWANO T., SAHASHI N., TAKAHASHI K., UOZUMI N., MUTO S., 1998 Salicylic acid
 1559 induces extracellular superoxide generation followed by an increase in cytosolic
 1560 calcium ion in tobacco suspension culture: the earliest events in salicylic acid signal
 1561 transduction. Plant Cell Physiol. **39**: 721–730.

1562 KELLIHER T., WALBOT V., 2012 Hypoxia triggers meiotic fate acquisition in maize.
 1563 Science **337**: 345–8.

1564 KEMPEN F., HOWARD W., PRING D. R., 1998 Mutations at specific atp6 codons which
 1565 cause human mitochondrial diseases also lead to male sterility in a plant. FEBS Lett.
 1566 **441**: 159–160.

1567 KENNEY M. C., CHWA M., ATILANO S. R., FALATOONZADEH P., RAMIREZ C., MALIK D.,
 1568 TAREK M., CARPIO J. C. DEL, NESBURN A. B., BOYER D. S., KUPPERMANN B. D.,
 1569 VAWTER M. P., JAZWINSKI S. M., MICELI M. V., WALLACE D. C., UDAR N., 2014
 1570 Molecular and bioenergetic differences between cells with African versus European
 1571 inherited mitochondrial DNA haplogroups: Implications for population
 1572 susceptibility to diseases. Biochim. Biophys. Acta - Mol. Basis Dis. **1842**: 208–219.

1573 KEUNEN E., REMANS T., BOHLER S., VANGRONSVELD J., CUYPERS A., 2011 Metal-
 1574 induced oxidative stress and plant mitochondria. Int. J. Mol. Sci. **12**: 6894–6918.

1575 KHOLODOVA V. P., BORMOTOVA T. S., SEMENOV O. G., DMITRIEVA G. a., KUZNETSOV
 1576 V. V., 2007 Physiological mechanisms of adaptation of alloplasmic wheat hybrids to
 1577 soil drought. Russ. J. Plant Physiol. **54**: 480–486.

1578 KIKUSATO M., TOYOMIZU M., 2013 Crucial Role of Membrane Potential in Heat Stress-

1579 Induced Overproduction of Reactive Oxygen Species in Avian Skeletal Muscle
 1580 Mitochondria (M Moran, Ed.). PLoS One **8**.
 1581 KINOSHITA T., SEKI M., 2014 Epigenetic memory for stress response and adaptation in
 1582 plants. Plant Cell Physiol. **55**: 1859–63.
 1583 KRAUSE M., DURNER J., 2004 Harpin inactivates mitochondria in Arabidopsis suspension
 1584 cells. Mol. Plant. Microbe. Interact. **17**: 131–139.
 1585 KRUFT V., EUBEL H., JÄNSCH L., WERHAHN W., BRAUN H. P., 2001 Proteomic approach
 1586 to identify novel mitochondrial proteins in Arabidopsis. Plant Physiol. **127**: 1694–
 1587 710.
 1588 KÜNSTLER A., HAFEZ Y., KIRÁLY L., 2007 Transient suppression of a catalase and an
 1589 alternative oxidase gene during virus-induced local lesion formation (hypersensitive
 1590 response) is independent of the extent of leaf necrotization. Acta Phytopathol.
 1591 Entomol. Hungarica **42**: 185–196.
 1592 KUROIWA T., FUJIE M., KUROIWA H., 1992 Studies on the behavior of mitochondrial
 1593 DNA: Synthesis of mitochondrial DNA occurs actively in a specific regions just
 1594 above the quiescent center in the roo meristem of *Pelargonium zonale*. J Cell Sci
 1595 **101**: 483–493.
 1596 KWAK J. M., NGUYEN V., SCHROEDER J. I., 2006 The role of reactive oxygen species in
 1597 hormonal responses. Plant Physiol. **141**: 323–329.
 1598 LA ROSA SANTAMARIA R. DE, SHAO M.-R., WANG G., NINO-LIU D. O., KUNDARIYA H.,
 1599 WAMBOLDT Y., DWEIKAT I., MACKENZIE S. A., 2014 MSH1-induced non-genetic
 1600 variation provides a source of phenotypic diversity in Sorghum bicolor. PLoS One
 1601 **9**: e108407.
 1602 LACOMME C., ROBY D., 1999 Identification of new early markers of the hypersensitive
 1603 response in *Arabidopsis thaliana*. FEBS Lett. **459**: 149–153.
 1604 LAM E., 2004 Controlled cell death, plant survival and development. Nat. Rev. Mol. Cell
 1605 Biol. **5**: 305–315.
 1606 LASSIG R., GUTERMUTH T., BEY T. D., KONRAD K. R., ROMEIS T., 2014 Pollen tube
 1607 NAD(P)H oxidases act as a speed control to dampen growth rate oscillations during
 1608 polarized cell growth. Plant J. **78**: 94–106.
 1609 LASTDRAGER J., HANSON J., SMEEKENS S., 2014 Sugar signals and the control of plant

1610 growth and development. J. Exp. Bot. **65**: 799–807.

1611 LAUER M., KNUDSEN C., NEWTON K. J., GABAY-LAUGHNAN S., LAUGHNAN J. R., 1990

1612 A partially deleted mitochondrial cytochrome oxidase gene in the NCS6 abnormal

1613 growth mutant of maize. New Biol. **2**: 179–86.

1614 LÁZARO J. J., JIMÉNEZ A., CAMEJO D., IGLESIAS-BAENA I., MARTÍ M. D. C., LÁZARO-

1615 PAYO A., BARRANCO-MEDINA S., SEVILLA F., 2013 Dissecting the integrative

1616 antioxidant and redox systems in plant mitochondria. Effect of stress and S-

1617 nitrosylation. Front. Plant Sci. **4**: 460.

1618 LEE S.-L. J., WARMKE H. E., 1979 Organelle size and number in fertile and {T}-

1619 cytoplasmic male sterile corn. Am J Bot **66**: 141–148.

1620 LEINO M., TEIXEIRA R., LANDGREN M., GLIMELIUS K., 2003 *Brassica napus* lines with

1621 rearranged *Arabidopsis* mitochondria display CMS and a range of developmental

1622 aberrations. Theor. Appl. Genet. **106**: 1156–1163.

1623 LENNON A. M., NEUENSCHWANDER U. H., RIBAS-CARBO M., GILES L., RYALS J. A.,

1624 SIEDOW J. N., 1997 The effects of salicylic acid and tobacco mosaic virus infection

1625 on the alternative oxidase of tobacco. Plant Physiol. **115**: 783–791.

1626 LEVINGS C. S., 1990 The Texas cytoplasm of maize: cytoplasmic male sterility and

1627 disease susceptibility. Science **250**: 942–7.

1628 LEYMARIE J., VITKAUSKAITĖ G., HOANG H. H., GENDREAU E., CHAZOULE V., MEIMOUN

1629 P., CORBINEAU F., EL-MAAROUF-BOUTEAU H., BAILLY C., 2012 Role of reactive

1630 oxygen species in the regulation of arabidopsis seed dormancy. Plant Cell Physiol.

1631 **53**: 96–106.

1632 LI Z., XING D., 2011 Mechanistic study of mitochondria-dependent programmed cell

1633 death induced by aluminium phytotoxicity using fluorescence techniques. J. Exp.

1634 Bot. **62**: 331–43.

1635 LI C.-R., LIANG D.-D., LI J., DUAN Y.-B., LI H., YANG Y.-C., QIN R.-Y., LI L., WEI P.-

1636 C., YANG J.-B., 2013 Unravelling mitochondrial retrograde regulation in the abiotic

1637 stress induction of rice ALTERNATIVE OXIDASE 1 genes. Plant. Cell Environ.

1638 **36**: 775–88.

1639 LI L., HU L., HAN L.-P., JI H., ZHU Y., WANG X., GE J., XU M., SHEN D., DONG H.,

1640 2014 Expression of turtle riboflavin-binding protein represses mitochondrial

electron transport gene expression and promotes flowering in Arabidopsis. BMC Plant Biol. **14**: 381.

LIMONGELLI G., MASARONE D., D'ALESSANDRO R., ELLIOTT P. M., 2012 Mitochondrial diseases and the heart: an overview of molecular basis, diagnosis, treatment and clinical course. Future Cardiol. **8**: 71–88.

LINKE B., NOTHNAGEL T., BÖRNER T., 2003 Flower development in carrot CMS plants: mitochondria affect the expression of MADS box genes homologous to GLOBOSA and DEFICIENS. Plant J. **34**: 27–37.

LINKE B., BÖRNER T., 2005 Mitochondrial effects on flower and pollen development. Mitochondrion **5**: 389–402.

LIU C. G., WU Y. W., HOU H., ZHANG C., ZHANG Y., 2002 Value and utilization of alloplasmic common wheats with Aegilops crassa cytoplasm. Plant Breed. **121**: 407–410.

LIU C., HOU N., LIU L., LIU G., WU Y., ZHANG C., ZHANG Y., 2005 Genetic basis and characterization of salt tolerance in alloplasmic wheat D2-'Jian 26'. Acta Agron. Sin. **31**: 1007–1013.

LIU Y., HE J., CHEN Z., REN X., HONG X., GONG Z., 2010 ABA overly-sensitive 5 (ABO5), encoding a pentatricopeptide repeat protein required for cis-splicing of mitochondrial nad2 intron 3, is involved in the abscisic acid response in Arabidopsis. Plant J. **63**: 749–765.

LOGAN D. C., LEAVER C. J., 2000 Mitochondria-targeted {GFP} highlights the heterogeneity of mitochondrial shape, size and movement within living plant cells. J. Exp. Bot. **51**: 865–871.

LOGAN D. C., 2006a The mitochondrial compartment. J Exp Bot **57**: 1225–1243.

LOGAN D. C., 2006b Plant mitochondrial dynamics. Biochim Biophys Acta **1763**: 430–441.

LONSDALE D. M., BREARS T., HODGE T. P., MELVILLE S. E., ROTTMANN W. H., 1988 The plant mitochondrial genome: {Homologous} recombination as a mechanism for generating heterogeneity. Philos. Trans. R. Soc. London B Biol. Sci. **319**: 149–163.

LOUGH A. N., ROARK L. M., KATO A., REAM T. S., LAMB J. C., BIRCHLER J. A., NEWTON K. J., 2008 Mitochondrial DNA transfer to the nucleus generates extensive

1672 insertion site variation in maize. *Genetics* **178**: 47–55.

1673 MA H., 2013 A battle between genomes in plant male fertility. *Nat. Genet.* **45**: 472–3.

1674 MACKENZIE S., HE S., LYZNIK A., 1994 The elusive plant mitochondrion as a genetic
1675 system. *Plant Physiol.* **105**: 775–780.

1676 MAŁECKA A., DERBA-MACELUCH M., KACZOROWSKA K., PIECHALAK A.,
1677 TOMASZEWSKA B., 2009 Reactive oxygen species production and antioxidative
1678 defense system in pea root tissues treated with lead ions: mitochondrial and
1679 peroxisomal level. *Acta Physiol. Plant.* **31**: 1065–1075.

1680 MARIENFELD J. R., NEWTON K. J., 1994 The maize NCS2 abnormal growth mutant has a
1681 chimeric nad4-nad7 mitochondrial gene and is associated with reduced complex I
1682 function. *Genetics* **138**: 855–63.

1683 MARTIN M. V., FIOL D. F., SUNDARESAN V., ZABALETA E. J., PAGNUSSAT G. C., 2013
1684 Oiwa, a female gametophytic mutant impaired in a mitochondrial manganese-
1685 superoxide dismutase, reveals crucial roles for reactive oxygen species during
1686 embryo sac development and fertilization in arabidopsis. *Plant Cell* **25**: 1573–1591.

1687 MARTIN W. F., GARG S., ZIMORSKI V., 2015 Endosymbiotic theories for eukaryote
1688 origin. *Philos. Trans. R. Soc. B Biol. Sci.* **370**: 20140330.

1689 MARTÍN-TRILLO M., CUBAS P., 2010 *TCP* genes: a family snapshot ten years later.
1690 *Trends Plant Sci* **15**: 31–39.

1691 MATTENBERGER Y., JAMES D. I., MARTINOU J.-C., 2003 Fusion of mitochondria in
1692 mammalian cells is dependent on the mitochondrial inner membrane potential and
1693 independent of microtubules or actin. *FEBS Lett.* **538**: 53–59.

1694 MAZZUCOTELLI E., MASTRANGELO A. M., CROSATTI C., GUERRA D., STANCA A. M.,
1695 CATTIVELLI L., 2008 Abiotic stress response in plants: When post-transcriptional
1696 and post-translational regulations control transcription. *Plant Sci.* **174**: 420–431.

1697 MCCABE T., DALEX D. O., WHELAN J., 2000 Regulatory, developmental and tissue
1698 aspects of mitochondria biogenesis in plants. *Plant Biol.* **2**: 121–135.

1699 McCONCHIE C. A., HOUGH T., KNOX R. B., 1987 Ultrastructural analysis of the sperm
1700 cells of mature pollen of maize, *Zea mays*. *Protoplasma* **139**: 9–19.

1701 MICKELBART M. V., HASEGAWA P. M., BAILEY-SERRES J., 2015 Genetic mechanisms of
1702 abiotic stress tolerance that translate to crop yield stability. *Nat. Rev. Genet.* **16**:

1703 237–251.

1704 MILLER-MESSMER M., KÜHN K., BICHARA M., RET M. LE, IMBAULT P., GUALBERTO J.,
 1705 2012 RECA-dependent DNA repair results in increased heteroplasmy of the
 1706 Arabidopsis mitochondrial genome. *Plant Physiol.*

1707 MISHMAR D., RUIZ-PESINI E., GOLIK P., MACAULAY V., CLARK A. G., HOSSEINI S.,
 1708 BRANDON M., EASLEY K., CHEN E., BROWN M. D., SUKERNIK R. I., OLCKERS A.,
 1709 WALLACE D. C., 2003 Natural selection shaped regional mtDNA variation in
 1710 humans. *Proc. Natl. Acad. Sci. U. S. A.* **100**: 171–6.

1711 MITTLER R., VANDERAUWERA S., GOLLERY M., BREUSEGEM F. VAN, 2004 Reactive
 1712 oxygen gene network of plants. *Trends Plant Sci.* **9**: 490–498.

1713 MITTOVA V., GUY M., TAL M., VOLOKITA M., 2004 Salinity up-regulates the
 1714 antioxidative system in root mitochondria and peroxisomes of the wild salt-tolerant
 1715 tomato species *Lycopersicon pennellii*. *J. Exp. Bot.* **55**: 1105–13.

1716 MOGENSEN H. L., WAGNER V. T., DUMAS C., 1990 Quantitative, three-dimensional
 1717 ultrastructure of isolated corn (*Zea mays*) sperm cells. *Protoplasma* **153**: 136–140.

1718 MOGHADAM A. A., EBRAHIMIE E., TAGHAVI S. M., NIAZI A., BABGOHARI M. Z., DEIHIMI
 1719 T., DJAVAHARI M., RAMEZANI A., 2013 How the nucleus and mitochondria
 1720 communicate in energy production during stress: nuclear MtATP6, an early-stress
 1721 responsive gene, regulates the mitochondrial F₁F₀-ATP synthase complex. *Mol.*
 1722 *Biotechnol.* **54**: 756–69.

1723 MØLLER I. M., 2001 PLANT MITOCHONDRIA AND OXIDATIVE STRESS : Electron
 1724 Transport, NADPH Turnover, and Metabolism of Reactive Oxygen Species. *Annu.*
 1725 *Rev. Plant Physiol. Plant Mol. Biol.* **52**: 561–591.

1726 MØLLER I. M., SWEETLOVE L. J., 2010 ROS signalling - specificity is required. *Trends*
 1727 *Plant Sci.* **15**: 370–374.

1728 MORENO-LOSHUERTOS R., ACÍN-PÉREZ R., FERNÁNDEZ-SILVA P., MOVILLA N., PÉREZ-
 1729 MARTOS A., RODRIGUEZ DE CORDOBA S., GALLARDO M. E., ENRÍQUEZ J. A., 2006
 1730 Differences in reactive oxygen species production explain the phenotypes associated
 1731 with common mouse mitochondrial DNA variants. *Nat. Genet.* **38**: 1261–1268.

1732 MUJAHID A., SATO K., AKIBA Y., TOYOMIZU M., 2006 Acute heat stress stimulates
 1733 mitochondrial superoxide production in broiler skeletal muscle, possibly via

1734 downregulation of uncoupling protein content. *Poult. Sci.* **85**: 1259–1265.

1735 MURAI K., TAKUMI S., KOGA H., OGIHARA Y., 2002 Pistillody, homeotic transformation
 1736 of stamens into pistil-like structures, caused by nuclear-cytoplasm interaction in
 1737 wheat. *Plant J.* **29**: 169–181.

1738 MURCHA M. W., KMIEC B., KUBISZEWSKI-JAKUBIAK S., TEIXEIRA P. F., GLASER E.,
 1739 WHELAN J., 2014 Protein import into plant mitochondria: signals, machinery,
 1740 processing, and regulation. *J. Exp. Bot.* **65**: 6301–35.

1741 NAGATA N., 2010 Mechanisms for independent cytoplasmic inheritance of mitochondria
 1742 and plastids in angiosperms. *J. Plant Res.* **123**: 193–9.

1743 NEALE D. B., MARSHALL K. A., SEDEROFF R. R., 1989 Chloroplast and mitochondrial
 1744 DNA are paternally inherited in *Sequoia sempervirens* D. Don Endl. *Proc. Natl.*
 1745 *Acad. Sci. U. S. A.* **86**: 9347–9.

1746 NEWTON K. J., COE E. H., 1986 Mitochondrial DNA changes in abnormal growth
 1747 (nonchromosomal stripe) mutants of maize. *Proc. Natl. Acad. Sci. U. S. A.* **83**:
 1748 7363–6.

1749 NEWTON K. J., KNUDSEN C., GABAY-LAUGHNAN S., LAUGHNAN J. R., 1990 An abnormal
 1750 growth mutant in maize has a defective mitochondrial cytochrome oxidase gene.
 1751 *Plant Cell* **2**: 107–13.

1752 NEWTON K. J., MARIANO J. M., GIBSON C. M., KUZMIN E., GABAY-LAUGHNAN S., 1996
 1753 Involvement of S2 episomal sequences in the generation of NCS4 deletion mutation
 1754 in maize mitochondria. *Dev. Genet.* **19**: 277–86.

1755 NG S., GIRAUD E., DUNCAN O., LAW S. R., WANG Y., XU L., NARSAI R., CARRIE C.,
 1756 WALKER H., DAY D. A., BLANCO N. E., STRAND Å., WHELAN J., IVANOVA A., 2013
 1757 Cyclin-dependent kinase E1 (CDKE1) provides a cellular switch in plants between
 1758 growth and stress responses. *J. Biol. Chem.* **288**: 3449–3459.

1759 NG S., CLERCQ I. DE, AKEN O. VAN, LAW S. R., IVANOVA A., WILLEMS P., GIRAUD E.,
 1760 BREUSEGEM F. VAN, WHELAN J., 2014 Anterograde and retrograde regulation of
 1761 nuclear genes encoding mitochondrial proteins during growth, development, and
 1762 stress. *Mol. Plant* **7**: 1075–93.

1763 NICK P., 2011 Plant cell harakiri - programmed cell death in development. *Protoplasma*
 1764 **248**: 633–634.

- 1765 NIKOVA V., VLADOVA R., 2014 Wild *Nicotiana* species as a source of Cytoplasmic Male
1766 Sterility in *Nicotiana tabacum*. Beiträge zur Tab. / Contrib. to Tob. Res. **20**: 301–
1767 311.
- 1768 NISHIMURA T., WATANUKI S., 2014 Relationship between mitochondrial haplogroup and
1769 seasonal changes of physiological responses to cold. J. Physiol. Anthropol. **33**: 27.
- 1770 NOGUEIRA F. T. S., BORECKÝ J., VERCESI A. E., ARRUDA P., 2005 Genomic structure
1771 and regulation of mitochondrial uncoupling protein genes in mammals and plants.
1772 Biosci. Rep. **25**: 209–26.
- 1773 NOGUEIRA C., ALMEIDA L. S., NESTI C., PEZZINI I., VIDEIRA A., VILARINHO L.,
1774 SANTORELLI F. M., 2014 Syndromes associated with mitochondrial DNA depletion.
1775 Ital. J. Pediatr. **40**: 34.
- 1776 NOYSZEWSKI A. K., GHAVAMI F., ALNEMER L. M., SOLTANI A., GU Y. Q., HUO N.,
1777 MEINHARDT S., KIANIAN P. M. A., KIANIAN S. F., 2014 Accelerated evolution of the
1778 mitochondrial genome in an alloplasmic line of durum wheat. BMC Genomics **15**:
1779 67.
- 1780 OLDENBURG D. J., BENDICH A. J., 2015 The linear plastid chromosomes of maize:
1781 terminal sequences, structures, and implications for DNA replication. Curr. Genet.
- 1782 OLYSLAEGERS G., VERBELEN J.-P., 1998 Improved staining of {F}-actin and co-
1783 localization of mitochondria in plant cells. J. Microsc. **192**: 73–77.
- 1784 ONDA Y., KATO Y., ABE Y., ITO T., MOROHASHI M., ITO Y., ICHIKAWA M.,
1785 MATSUKAWA K., KAKIZAKI Y., KOIWA H., ITO K., 2008 Functional coexpression of
1786 the mitochondrial alternative oxidase and uncoupling protein underlies
1787 thermoregulation in the thermogenic florets of skunk cabbage. Plant Physiol. **146**:
1788 636–645.
- 1789 ORACZ K., EL-MAAROUF-BOUTEAU H., KRANNER I., BOGATEK R., CORBINEAU F.,
1790 BAILLY C., 2009 The mechanisms involved in seed dormancy alleviation by
1791 hydrogen cyanide unravel the role of reactive oxygen species as key factors of
1792 cellular signaling during germination. Plant Physiol. **150**: 494–505.
- 1793 ORDOG S. H., HIGGINS V. J., VANLERBERGHE G. C., 2002 Mitochondrial alternative
1794 oxidase is not a critical component of plant viral resistance but may play a role in the
1795 hypersensitive response. Plant Physiol. **129**: 1858–65.

- 1796 OSHIMA M., KIKUCHI R., IMAMURA J., HANDA H., 2010 Origin of the CMS gene locus in
1797 rapeseed cybrid mitochondria: active and inactive recombination produces the
1798 complex CMS gene region in the mitochondrial genomes of Brassicaceae. *Genes*
1799 *Genet. Syst.* **85**: 311–8.
- 1800 PALIWAL S., FIUMERA A. C., FIUMERA H. L., 2014 Mitochondrial-Nuclear Epistasis
1801 Contributes to Phenotypic Variation and Coadaptation in Natural Isolates of
1802 *Saccharomyces cerevisiae*. *Genetics*.
- 1803 PALMER J. D., ADAMS K. L., CHO Y., PARKINSON C. L., QIU Y.-L., SONG K., 2000
1804 Dynamic evolution of plant mitochondrial genomes: Mobile genes and introns and
1805 highly variable mutation rates. *Proc. Natl. Acad. Sci.* **97**: 6960–6966.
- 1806 PANAYOTOV I., 1980 New cytoplasmic male sterility sources in common wheat: Their
1807 genetical and breeding considerations. *Theor. Appl. Genet.* **56**: 153–60.
- 1808 PAPINI A., MOSTI S., BRIGHIGNA L., 1999 Programmed-cell-death events during tapetum
1809 development of angiosperms. *Protoplasma* **207**: 213–221.
- 1810 PAPINI A., MOSTI S., MILOCANI E., TANI G., FALCO P. DI, BRIGHIGNA L., 2011
1811 Megasporogenesis and programmed cell death in *Tillandsia* (Bromeliaceae).
1812 *Protoplasma* **248**: 651–662.
- 1813 PARENT J.-S., LEPAGE E., BRISSON N., 2011 Divergent roles for the two PolI-like
1814 organelle DNA polymerases of Arabidopsis. *Plant Physiol.* **156**: 254–262.
- 1815 PASTORE D., TRONO D., LAUS M. N., FONZO N. DI, FLAGELLA Z., 2007 Possible plant
1816 mitochondria involvement in cell adaptation to drought stress. A case study: durum
1817 wheat mitochondria. *J. Exp. Bot.* **58**: 195–210.
- 1818 PERL A., AVIV D., GALUN E., 1991 Nuclear-organelle interaction in *Solanum*:
1819 interspecific cybridizations and their correlation with a plastome dendrogram. *Mol.*
1820 *Gen. Genet.* **228**: 193–200.
- 1821 PETROV V., HILLE J., MUELLER-ROEBER B., GECHEV T. S., 2015 ROS-mediated abiotic
1822 stress-induced programmed cell death in plants. *Front. Plant Sci.* **6**: 69.
- 1823 PU X., LV X., TAN T., FU F., QIN G., LIN H., 2015 Roles of mitochondrial energy
1824 dissipation systems in plant development and acclimation to stress. *Ann. Bot.* **116**:
1825 583–600.
- 1826 PYLE A., FOLTYNIE T., TIANGYOU W., LAMBERT C., KEERS S. M., ALLCOCK L. M.,

1827 DAVISON J., LEWIS S. J., PERRY R. H., BARKER R., BURN D. J., CHINNERY P. F.,
1828 2005 Mitochondrial DNA haplogroup cluster UKJT reduces the risk of PD. *Ann.*
1829 *Neurol.* **57**: 564–7.

1830 RAKHMATULLINA D., PONOMAREVA A., GAZIZOVA N., MINIBAYEVA F., 2015
1831 Mitochondrial morphology and dynamics in *Triticum aestivum* roots in response to
1832 rotenone and antimycin A. *Protoplasma*.

1833 RASMUSSEN A. G., SOOLE K. L., ELTHON T. E., 2004 Alternative NAD(P)H
1834 dehydrogenases of plant mitochondria. *Annu. Rev. Plant Biol.* **55**: 23–39.

1835 RAVAGNAN L., ROUMIER T., KROEMER G., 2002 Mitochondria, the killer organelles and
1836 their weapons. *J. Cell. Physiol.* **192**: 131–137.

1837 RHOADS D. M., MCINTOSH L., 1992 Salicylic acid regulation of respiration in higher
1838 plants: alternative oxidase expression. *Plant Cell* **4**: 1131–1139.

1839 RHOADS D. M., MCINTOSH L., 1993 The salicylic acid-inducible alternative oxidase gene
1840 *aox1* and genes encoding pathogenesis-related proteins share regions of sequence
1841 similarity in their promoters. *Plant Mol. Biol.* **21**: 615–624.

1842 RHOADS D. M., UMBACH A. L., SWEET C. R., LENNON A. M., RAUCH G. S., SIEDOW J.
1843 N., 1998 Regulation of the cyanide-resistant alternative oxidase of plant
1844 mitochondria. Identification of the cysteine residue involved in alpha-keto acid
1845 stimulation and intersubunit disulfide bond formation. *J Biol Chem* **273**: 30750–
1846 30756.

1847 RICE D. W., ALVERSON A. J., RICHARDSON A. O., YOUNG G. J., SANCHEZ-PUERTA M.
1848 V., MUNZINGER J., BARRY K., BOORE J. L., ZHANG Y., DEPAMPHILIS C. W., KNOX
1849 E. B., PALMER J. D., 2013 Horizontal transfer of entire genomes via mitochondrial
1850 fusion in the angiosperm *Amborella*. *Science* **342**: 1468–73.

1851 RONFORT J., SAUMITOU-LAPRADE P., CUGUEN J., COUVET D., 1995 Mitochondrial DNA
1852 diversity and male sterility in natural populations of *Daucus carota* ssp *carota*.
1853 *Theor. Appl. Genet.* **91**: 150–159.

1854 SAHA D., PRASAD A. M., SRINIVASAN R., 2007 Pentatricopeptide repeat proteins and
1855 their emerging roles in plants. *Plant Physiol. Biochem.* PPB / Société Fr. Physiol.
1856 végétale **45**: 521–34.

1857 SAIRAM R. K., SRIVASTAVA G. ., 2002 Changes in antioxidant activity in sub-cellular

1858 fractions of tolerant and susceptible wheat genotypes in response to long term salt
1859 stress. *Plant Sci.* **162**: 897–904.

1860 SAISHO D., NAKAZONO M., LEE K. H., TSUTSUMI N., AKITA S., HIRAI A., 2001 The gene
1861 for alternative oxidase-2 (*AOX2*) from *Arabidopsis thaliana* consists of five exons
1862 unlike other *AOX* genes and is transcribed at an early stage during germination.
1863 *Genes Genet Syst* **76**: 89–97.

1864 SANCHEZ-PUERTA M. V., ZUBKO M. K., PALMER J. D., 2015 Homologous recombination
1865 and retention of a single form of most genes shape the highly chimeric
1866 mitochondrial genome of a cybrid plant. *New Phytol.* **206**: 381–396.

1867 SANDHU A. P. S., ABDELNOOR R. V., MACKENZIE S. A., 2007 Transgenic induction of
1868 mitochondrial rearrangements for cytoplasmic male sterility in crop plants. *Proc.*
1869 *Natl. Acad. Sci. U. S. A.* **104**: 1766–70.

1870 SATO M., SATO K., 2013 Maternal inheritance of mitochondrial DNA by diverse
1871 mechanisms to eliminate paternal mitochondrial DNA. *Biochim. Biophys. Acta*
1872 **1833**: 1979–84.

1873 SATOH M., NEMOTO Y., KAWANO S., NAGATA T., HIROKAWA H., KUROIWA T., 1993
1874 Organization of heterogeneous mitochondrial DNA molecules in mitochondrial
1875 nuclei of cultured tobacco cells. *Protoplasma* **175**: 112–120.

1876 SCHERTL P., BRAUN H.-P., 2014 Respiratory electron transfer pathways in plant
1877 mitochondria. *Front. Plant Sci.* **5**: 163.

1878 SECHNYAK A. L., VASILIEV A. A., MANDRYCHENKO T. A., GOLUB J. V., 2010 Resistance
1879 of alloplasmic wheats to brown leaf rust. *Analele Univ. din Oradea, Fasc. Biol.:*
1880 292–295.

1881 SEGUÍ-SIMARRO J. M., CORONADO M. J., STAEHELIN L. A., 2008 The mitochondrial
1882 cycle of *Arabidopsis* shoot apical meristem and leaf primordium meristematic cells
1883 is defined by a perinuclear tentaculate/cage-like mitochondrion. *Plant Physiol.* **148**:
1884 1380–1393.

1885 SEGUÍ-SIMARRO J. M., STAEHELIN L. A., 2009 Mitochondrial reticulation in shoot apical
1886 meristem cells of *Arabidopsis* provides a mechanism for homogenization of mtDNA
1887 prior to gamete formation. *Plant Signal. Behav.* **4**: 168–171.

1888 SEYMOUR R. S., GIBERNAU M., ITO K., 2003 Thermogenesis and respiration of

1889 inflorescences of the dead horse arum *Helicodiceros muscivorus*, a pseudo-
1890 thermoregulatory aroid associated with fly pollination. *Funct. Ecol.* **17**: 886–894.

1891 SEYMOUR R. S., GIBERNAU M., 2008 Respiration of thermogenic inflorescences of
1892 *Philodendron melinonii*: natural pattern and responses to experimental
1893 temperatures. *J. Exp. Bot.* **59**: 1353–1362.

1894 SEYMOUR R. S., ITO Y., ONDA Y., ITO K., 2009 Effects of floral thermogenesis on pollen
1895 function in Asian skunk cabbage *Symplocarpus renifolius*. *Biol. Lett.* **5**: 568–70.

1896 SHEAHAN M. B., ROSE R. J., MCCURDY D. W., 2004 Organelle inheritance in plant cell
1897 division: the actin cytoskeleton is required for unbiased inheritance of chloroplasts,
1898 mitochondria and endoplasmic reticulum in dividing protoplasts. *Plant J.* **37**: 379–
1899 390.

1900 SHEAHAN M. B., MCCURDY D. W., ROSE R. J., 2005 Mitochondria as a connected
1901 population: ensuring continuity of the mitochondrial genome during plant cell
1902 dedifferentiation through massive mitochondrial fusion. *Plant J.* **44**: 744–55.

1903 SHEDGE V., DAVILA J., ARRIETA-MONTIEL M. P., MOHAMMED S., MACKENZIE S. a, 2010
1904 Extensive rearrangement of the Arabidopsis mitochondrial genome elicits cellular
1905 conditions for thermotolerance. *Plant Physiol.* **152**: 1960–70.

1906 SHUMWAY L. K., BAUMAN L. F., 1967 Nonchromosomal stripe of maize. *Genetics* **55**:
1907 33–8.

1908 SIEDOW J. N., UMBACH A. L., 2000 The mitochondrial cyanide-resistant oxidase:
1909 structural conservation amid regulatory diversity. *Biochim. Biophys. Acta* **1459**:
1910 432–9.

1911 SIMONS B. H., 1999 Enhanced Expression and Activation of the Alternative Oxidase
1912 during Infection of Arabidopsis with *Pseudomonas syringae* pv tomato. *PLANT*
1913 *Physiol.* **120**: 529–538.

1914 SINIAVSKAIA M. G., DANILENKO N. G., DAVYDENKO O. G., ERMISHINA N. M., BEL'KO
1915 N. B., GORDEĬ I. A., 2004 [Inheritance of organelle DNA in rye (*Secale cereale* L.)
1916 and wheat (x *Triticale* Thch.) hybrids]. *Genetika* **40**: 218–23.

1917 SKINNER D. J., BAKER S. C., MEISTER R. J., BROADHVEST J., SCHNEITZ K., GASSER C.
1918 S., 2001 The *Arabidopsis* *HUELLENLOS* gene, which is essential for normal ovule
1919 development, encodes a mitochondrial ribosomal protein. *Plant Cell* **13**: 2719–2730.

1920 SLOAN D. B., ALVERSON A. J., CHUCKALOVCAK J. P., WU M., MCCAULEY D. E.,
1921 PALMER J. D., TAYLOR D. R., 2012 Rapid evolution of enormous,
1922 multichromosomal genomes in flowering plant mitochondria with exceptionally
1923 high mutation rates. *PLoS Biol.* **10**: e1001241.

1924 SLOAN D. B., 2013 One ring to rule them all? Genome sequencing provides new insights
1925 into the “master circle” model of plant mitochondrial DNA structure. *New Phytol.*
1926 **200**: 978–985.

1927 SMALL I. D., ISAAC P. G., LEAVER C. J., 1987 Stoichiometric differences in DNA
1928 molecules containing the *atpA* gene suggest mechanisms for the generation of
1929 mitochondrial genome diversity in maize. *EMBO J.* **6**: 865–9.

1930 SMART C. J., MONÉGER F., LEAVER C. J., 1994 Cell-specific regulation of gene
1931 expression in mitochondria during anther development in sunflower. *Plant Cell* **6**:
1932 811–825.

1933 SMITH C. A., MELINO V. J., SWEETMAN C., SOOLE K. L., 2009 Manipulation of
1934 alternative oxidase can influence salt tolerance in *Arabidopsis thaliana*. *Physiol.*
1935 *Plant.* **137**: 459–472.

1936 SMITH D. R., KEELING P. J., 2015 Mitochondrial and plastid genome architecture:
1937 Reoccurring themes, but significant differences at the extremes. *Proc. Natl. Acad.*
1938 *Sci.* **112**: 201422049.

1939 SMITH D. R., 2015 The past, present and future of mitochondrial genomics: have we
1940 sequenced enough mtDNAs? *Brief. Funct. Genomics.*

1941 SOLIERI L., 2010 Mitochondrial inheritance in budding yeasts: towards an integrated
1942 understanding. *Trends Microbiol.* **18**: 521–30.

1943 SOLTANI A., GHAVAMI F., MERGOU M., HEGSTAD J., NOYSZEWSKI A., MEINHARDT S.,
1944 KIANIAN S. F., 2014 Analysis of ATP6 sequence diversity in the *Triticum-Aegilops*
1945 species group reveals the crucial role of rearrangement in mitochondrial genome
1946 evolution. *Genome* **57**: 279–88.

1947 SPOONER R., YILMAZ Ö., 2011 The role of reactive-oxygen-species in microbial
1948 persistence and inflammation. *Int. J. Mol. Sci.* **12**: 334–352.

1949 STEINBORN R., LINKE B., NOTHNAGEL T., BÖRNER T., 1995 Inheritance of chloroplast
1950 and mitochondrial DNA in alloplasmic forms of the genus *Daucus*. *Theor. Appl.*

- 1951 Genet. **91**: 632–8.
- 1952 STICKENS D., VERBELEN J.-P., 1996 Spatial structure of mitochondria and ER denotes
1953 changes in cell physiology of cultured tobacco protoplasts. Plant J. **9**: 85–92.
- 1954 SUZUKI N., MILLER G., MORALES J., SHULAEV V., TORRES M. A., MITTLER R., 2011
1955 Respiratory burst oxidases: the engines of ROS signaling. Curr. Opin. Plant Biol. **14**:
1956 691–699.
- 1957 SUZUKI N., KOUSSEVITZKY S., MITTLER R., MILLER G., 2012 ROS and redox signalling
1958 in the response of plants to abiotic stress. Plant. Cell Environ. **35**: 259–70.
- 1959 SWANSON S., GILROY S., 2010 ROS in plant development. Physiol. Plant. **138**: 384–392.
- 1960 SZABADOS L., SAVOURÉ A., 2010 Proline: a multifunctional amino acid. Trends Plant
1961 Sci. **15**: 89–97.
- 1962 TEIXEIRA R. T., FARBOS I., GLIMELIUS K., 2005 Expression levels of meristem identity
1963 and homeotic genes are modified by nuclear-mitochondrial interactions in
1964 alloplasmic male-sterile lines of *Brassica napus*. Plant J. **42**: 731–742.
- 1965 THIRKETTLE-WATTS D., MCCABE T. C., CLIFTON R., MOORE C., FINNEGAN P. M., DAY
1966 D. A., WHELAN J., 2003 Analysis of the alternative oxidase promoters from
1967 soybean. Plant Physiol **133**: 1158–1169.
- 1968 TOMÉ F., NÄGELE T., ADAMO M., GARG A., MARCO-LLORCA C., NUKARINEN E.,
1969 PEDROTTI L., PEVIANI A., SIMEUNOVIC A., TATKIEWICZ A., TOMAR M., GAMM M.,
1970 2014 The low energy signaling network. Plant Physiol. **5**: 353.
- 1971 TSUKAGOSHI H., BUSCH W., BENFEY P. N., 2010 Transcriptional regulation of ROS
1972 controls transition from proliferation to differentiation in the root. Cell **143**: 606–16.
- 1973 TSUNEWAKI K., 1980 Introduction. In: Tsunewaki K (Ed.), *Genetic Diversity of the*
1974 *Cytoplasm in Triticum and Aegilops*, Japan Society for the Promotion of Science,
1975 pp. 1–12.
- 1976 TSUNEWAKI K., WANG G. Z., MATSUOKA Y., 1996 Plasmon analysis of Triticum (wheat)
1977 and Aegilops. 1. Production of alloplasmic common wheats and their fertilities.
1978 Genes Genet. Syst. **71**: 293–311.
- 1979 TSUNEWAKI K., WANG G.-Z., MATSUOKA Y., 2002 Plasmon analysis of Triticum (wheat)
1980 and Aegilops. 2. Characterization and classification of 47 plasmons based on their
1981 effects on common wheat phenotype. Genes Genet. Syst. **77**: 409–27.

- 1982 UMBACH A. L., FIORANI F., SIEDOW J. N., 2005 Characterization of transformed
1983 *Arabidopsis* with altered alternative oxidase levels and analysis of effects on
1984 reactive oxygen species in tissue. *Plant Physiol* **139**: 1806–1820.
- 1985 VANDERAUWERA S., VANDENBROUCKE K., INZÉ A., COTTE B. VAN DE, MÜHLENBOCK P.,
1986 RYCKE R. DE, NAOUAR N., GAEVER T. VAN, MONTAGU M. C. E. VAN, BREUSEGEM
1987 F. VAN, 2012 AtWRKY15 perturbation abolishes the mitochondrial stress response
1988 that steers osmotic stress tolerance in *Arabidopsis*. *Proc. Natl. Acad. Sci. U. S. A.*
1989 **109**: 20113–8.
- 1990 VANLERBERGHE G. C., MCINTOSH L., 1996 Signals Regulating the Expression of the
1991 Nuclear Gene Encoding Alternative Oxidase of Plant Mitochondria. *Plant Physiol.*
1992 **111**: 589–595.
- 1993 VANLERBERGHE G. C., MCINTOSH L., 1997 Alternative oxidase: from gene to function.
1994 *Annu. Rev. Plant Physiol. Plant Mol. Biol.* **48**: 703–734.
- 1995 VANLERBERGHE G. C., 2013 Alternative oxidase: A mitochondrial respiratory pathway to
1996 maintain metabolic and signaling homeostasis during abiotic and biotic stress in
1997 plants. *Int. J. Mol. Sci.* **14**: 6805–6847.
- 1998 VERSLUES P. E., SHARMA S., 2010 Proline metabolism and its implications for plant-
1999 environment interaction. *Arabidopsis Book* **8**: e0140.
- 2000 VIDAL G., RIBAS-CARBO M., GARMIER M., DUBERTRET G., RASMUSSEN A. G., MATHIEU
2001 C., FOYER C. H., PAEPE R. DE, 2007 Lack of Respiratory Chain Complex I Impairs
2002 Alternative Oxidase Engagement and Modulates Redox Signaling during Elicitor-
2003 Induced Cell Death in Tobacco. *PLANT CELL ONLINE* **19**: 640–655.
- 2004 VIRDI K. S., LAURIE J. D., XU Y.-Z., YU J., SHAO M.-R., SANCHEZ R., KUNDARIYA H.,
2005 WANG D., RIETHOVEN J.-J. M., WAMBOLDT Y., ARRIETA-MONTIEL M. P., SHEDGE
2006 V., MACKENZIE S. A., 2015 *Arabidopsis* MSH1 mutation alters the epigenome and
2007 produces heritable changes in plant growth. *Nat. Commun.* **6**: 6386.
- 2008 WALLACE D. C., 2005 A mitochondrial paradigm of metabolic and degenerative diseases,
2009 aging, and cancer: A dawn for evolutionary biology. *Annu Rev Genet* **39**: 359–407.
- 2010 WALLACE D. C., FAN W., 2010 Energetics, epigenetics, mitochondrial genetics.
2011 *Mitochondrion* **10**: 12–31.
- 2012 WALLACE D. C., CHALKIA D., 2013 Mitochondrial DNA genetics and the heteroplasmy

2013 conundrum in evolution and disease. Cold Spring Harb. Perspect. Biol. **5**: a021220.
 2014 WALT J. M. VAN DER, DEMENTIEVA Y. A., MARTIN E. R., SCOTT W. K., NICODEMUS K.
 2015 K., KRONER C. C., WELSH-BOHMER K. A., SAUNDERS A. M., ROSES A. D., SMALL
 2016 G. W., SCHMECHEL D. E., MURALI DORAISWAMY P., GILBERT J. R., HAINES J. L.,
 2017 VANCE J. M., PERICAK-VANCE M. A., 2004 Analysis of European mitochondrial
 2018 haplogroups with Alzheimer disease risk. Neurosci. Lett. **365**: 28–32.
 2019 WANG H. M., KETELA T., KELLER W. A., GLEDDIE S. C., BROWN G. G., 1995 Genetic
 2020 correlation of the orf224/atp6 gene region with Polima CMS in Brassica somatic
 2021 hybrids. Plant Mol. Biol. **27**: 801–7.
 2022 WANG G. Z., MATSUOKA Y., TSUNEWAKI K., 2000 Evolutionary features of chondriome
 2023 divergence in Triticum (wheat) and Aegilops shown by RFLP analysis of
 2024 mitochondrial DNAs. Theor. Appl. Genet. **100**: 221–231.
 2025 WANG Y., YING Y., CHEN J., WANG X., 2004 Transgenic Arabidopsis overexpressing
 2026 Mn-SOD enhanced salt-tolerance. Plant Sci. **167**: 671–677.
 2027 WANG X.-P., SU L.-X., SU J.-W., 2006 Distribution changes of calcium and programmed
 2028 cell death in the pistil of litchi (Litchi chinensis Sonn.) flower during its
 2029 development. J. Plant Physiol. Mol. Biol. **32**: 607–616.
 2030 WANG Y. C., QU G. Z., LI H. Y., WU Y. J., WANG C., LIU G. F., YANG C. P., 2010a
 2031 Enhanced salt tolerance of transgenic poplar plants expressing a manganese
 2032 superoxide dismutase from Tamarix androssowii. Mol. Biol. Rep. **37**: 1119–1124.
 2033 WANG H., LIANG X., HUANG J., ZHANG D., LU H., LIU Z., BI Y., 2010b Involvement of
 2034 ethylene and hydrogen peroxide in induction of alternative respiratory pathway in
 2035 salt-treated arabidopsis calluses. Plant Cell Physiol. **51**: 1754–1765.
 2036 WANG Y., ITAYA A., ZHONG X., WU Y., ZHANG J., KNAAP E. VAN DER, OLMSTEAD R.,
 2037 QI Y., DING B., 2011 Function and evolution of a MicroRNA that regulates a Ca²⁺-
 2038 ATPase and triggers the formation of phased small interfering RNAs in tomato
 2039 reproductive growth. Plant Cell **23**: 3185–3203.
 2040 WARMKE H. E., LEE S. L., 1978 Pollen abortion in T cytoplasmic male-sterile corn (*Zea*
 2041 *mays*): A suggested mechanism. Science (80-.). **200**: 561–563.
 2042 WASHINGTON W. J., MAAN S. S., 1974 Disease Reaction of Wheat with Alien
 2043 Cytoplasms1. Crop Sci. **14**: 903.

2044 WENG S.-W., LIN T.-K., WANG P.-W., CHEN S.-D., CHUANG Y.-C., LIOU C.-W., 2013
 2045 Single nucleotide polymorphisms in the mitochondrial control region are associated
 2046 with metabolic phenotypes and oxidative stress. *Gene* **531**: 370–376.

2047 WILSON K. E., IVANOV A. G., ÖQUIST G., GRODZINSKI B., SARHAN F., HUNER N. P. A.,
 2048 2006 Energy balance, organellar redox status, and acclimation to environmental
 2049 stress. *Can. J. Bot.* **84**: 1355–1370.

2050 WITTKOPP S., STAIMER N., TJOA T., GILLEN D., DAHER N., SHAFER M., SCHAUER J. J.,
 2051 SIOUTAS C., DELFINO R. J., 2013 Mitochondrial Genetic Background Modifies the
 2052 Relationship between Traffic-Related Air Pollution Exposure and Systemic
 2053 Biomarkers of Inflammation. *PLoS One* **8**: e64444.

2054 WOLOSZYNSKA M., 2010 Heteroplasmy and stoichiometric complexity of plant
 2055 mitochondrial genomes--though this be madness, yet there's method in't. *J. Exp.*
 2056 *Bot.* **61**: 657–71.

2057 WOODSON J. D., CHORY J., 2008 Coordination of gene expression between organellar
 2058 and nuclear genomes. *Nat. Rev. Genet.* **9**: 383–395.

2059 WU Y., ZHANG C., LIU C., SHUXIN R., YAN Z., 1998 Breeding technology of alloplasmic
 2060 wheat. *Sci. China. C. Life Sci.* **41**: 449–58.

2061 WU Z., CUTHBERT J. M., TAYLOR D. R., SLOAN D. B., 2015a The massive mitochondrial
 2062 genome of the angiosperm *Silene noctiflora* is evolving by gain or loss of entire
 2063 chromosomes. *Proc. Natl. Acad. Sci. U. S. A.* **112**: 10185–91.

2064 WU B., BULJIC A., HAO W., 2015b Extensive Horizontal Transfer and Homologous
 2065 Recombination Generate Highly Chimeric Mitochondrial Genomes in Yeast. *Mol.*
 2066 *Biol. Evol.* **32**: 2559–2570.

2067 XIE H.-T., WAN Z.-Y., LI S., ZHANG Y., 2014 Spatiotemporal Production of Reactive
 2068 Oxygen Species by NADPH Oxidase Is Critical for Tapetal Programmed Cell Death
 2069 and Pollen Development in Arabidopsis. *Plant Cell* **26**: 2007–2023.

2070 XU J., 2005 The inheritance of organelle genes and genomes: patterns and mechanisms.
 2071 *Genome / Natl. Res. Counc. Canada = Génome / Cons. Natl. Rech. Canada* **48**: 951–
 2072 8.

2073 XU X. Y., LIU J. H., DENG X. X., 2006 Isolation of cytoplasts from Satsuma mandarin
 2074 (*Citrus unshiu* Marc.) and production of alloplasmic hybrid calluses via cytoplast-

2075 protoplast fusion. *Plant Cell Rep.* **25**: 533–9.

2076 XU Y.-Z., ARRIETA-MONTIEL M. P., VIRDI K. S., PAULA W. B. M. DE, WIDHALM J. R.,
 2077 BASSET G. J., DAVILA J. I., ELTHON T. E., ELOWSKY C. G., SATO S. J., CLEMENTE
 2078 T. E., MACKENZIE S. A., 2011 MutS HOMOLOG1 is a nucleoid protein that alters
 2079 mitochondrial and plastid properties and plant response to high light. *Plant Cell* **23**:
 2080 3428–41.

2081 XU Y.-Z., SANTAMARIA R. de la R., VIRDI K. S., ARRIETA-MONTIEL M. P., RAZVI F., LI
 2082 S., REN G., YU B., ALEXANDER D., GUO L., FENG X., DWEIKAT I. M., CLEMENTE T.
 2083 E., MACKENZIE S. A., 2012a The chloroplast triggers developmental reprogramming
 2084 when mutS HOMOLOG1 is suppressed in plants. *Plant Physiol.* **159**: 710–20.

2085 XU F., YUAN S., ZHANG D.-W., LV X., LIN H.-H., 2012b The role of alternative oxidase
 2086 in tomato fruit ripening and its regulatory interaction with ethylene. *J. Exp. Bot.* **63**:
 2087 5705–5716.

2088 Y WANG, M WISNIEWSKI R. M., 2007 Ectopic expression of Mn-SOD in *Lycopersicon*
 2089 *esculentum* leads to enhanced tolerance to salt and oxidative stress. *J. Appl. Hortic.*
 2090 **9(1)**: 3–8.

2091 YAFFE M. P., 1999 The machinery of mitochondrial inheritance and behavior. *Science*
 2092 (80-.). **283**: 1493–1497.

2093 YANG X., KUNDARIYA H., XU Y.-Z., SANDHU A., YU J., HUTTON S. F., ZHANG M.,
 2094 MACKENZIE S. A., 2015 MutS HOMOLOG1-derived epigenetic breeding potential
 2095 in tomato. *Plant Physiol.* **168**: 222–32.

2096 YE K., LU J., MA F., KEINAN A., GU Z., 2014 Extensive pathogenicity of mitochondrial
 2097 heteroplasmy in healthy human individuals. *Proc. Natl. Acad. Sci.* **111**: 10654–
 2098 10659.

2099 YEMM E. W., 1965 The respiration of plants and their organs. In: Steward FC (Ed.), *Plant*
 2100 *Physiology, A Treatise, Vol IV-A*, Academic Press, Inc., New York, pp. 231–310.

2101 YOSHINAGA K., ARIMURA S.-I., NIWA Y., TSUTSUMI N., UCHIMIYA H., KAWAI-YAMADA
 2102 M., 2005 Mitochondrial behaviour in the early stages of ROS stress leading to cell
 2103 death in *Arabidopsis thaliana*. *Ann. Bot.* **96**: 337–342.

2104 ZABALETA, HEISER, GROHMANN, BRENNICKE, 1998 Promoters of nuclear-encoded
 2105 respiratory chain Complex I genes from *Arabidopsis thaliana* contain a region

2106 essential for anther/pollen-specific expression. *Plant J.* **15**: 49–59.

2107 ZHANG Z., GREEN B. R., CAVALIER-SMITH T., 1999 Single gene circles in dinoflagellate
2108 chloroplast genomes. **400**: 155–159.

2109 ZHANG X., TAKANO T., LIU S., 2006 Identification of a mitochondrial ATP synthase
2110 small subunit gene (RMtATP6) expressed in response to salts and osmotic stresses
2111 in rice (*Oryza sativa* L.). In: *Journal of Experimental Botany*, pp. 193–200.

2112 ZHANG X., LIU S., TAKANO T., 2008 Overexpression of a mitochondrial ATP synthase
2113 small subunit gene (AtMtATP6) confers tolerance to several abiotic stresses in
2114 *Saccharomyces cerevisiae* and *Arabidopsis thaliana*. *Biotechnol. Lett.* **30**: 1289–
2115 1294.

2116 ZHANG L., OH Y., LI H., BALDWIN I. T., GALIS I., 2012 Alternative Oxidase in
2117 Resistance to Biotic Stresses: *Nicotiana attenuata* AOX Contributes to Resistance to
2118 a Pathogen and a Piercing-Sucking Insect But Not *Manduca sexta* Larvae. *Plant*
2119 *Physiol.* **160**: 1453–1467.

2120 ZHANG B., AKEN O. Van, THATCHER L., CLERCQ I. De, DUNCAN O., LAW S. R.,
2121 MURCHA M. W., MERWE M. VAN DER, SEIFI H. S., CARRIE C., CAZZONELLI C.,
2122 RADOMILJAC J., HÖFTE M., SINGH K. B., BREUSEGEM F. Van, WHELAN J., 2014 The
2123 mitochondrial outer membrane AAA ATPase AtOM66 affects cell death and
2124 pathogen resistance in *Arabidopsis thaliana*. *Plant J.*: 709–727.

2125 ZHAO N., XU X., WAMBOLDT Y., MACKENZIE S. A., YANG X., HU Z., YANG J., ZHANG
2126 M., 2015 MutS HOMOLOG1 silencing mediates ORF220 substoichiometric shifting
2127 and causes male sterility in *Brassica juncea*. *J. Exp. Bot.*

2128 ZHU Y., LU J., WANG J., CHEN F., LENG F., LI H., 2011 Regulation of thermogenesis in
2129 plants: the interaction of alternative oxidase and plant uncoupling mitochondrial
2130 protein. *J Integr Plant Biol* **53**: 7–13.

2131 ZOTTINI M., BARIZZA E., BASTIANELLI F., CARIMI F., SCHIAVO F. LO, 2006 Growth and
2132 senescence of *Medicago truncatula* cultured cells are associated with
2133 characteristic mitochondrial morphology. *New Phytol* **172**: 239–247.

2134 ZUBKO M. K., ZUBKO E. I., RUBAN A. V., ADLER K., MOCK H.-P., MISERA S., GLEBA Y.
2135 Y. U., GRIMM B., 2001 Extensive developmental and metabolic alterations in
2136 cybrids *Nicotiana tabacum* (+*Hyoscyamus niger*) are caused by complex nucleo-

2137 cytoplasmic incompatibility. *Plant J.* **25**: 627–639.

2138 ZUBKO M. K., ZUBKO E. I., ADLER K., GRIMM B., GLEBA Y. Y., 2003 New CMS-

2139 associated phenotypes in cybrids *Nicotiana tabacum* L. (+*Hyoscyamus niger* L.).

2140 *Ann. Bot.* **92**: 281–288.

2141 ZUBKO M. K., 2004 Mitochondrial tuning fork in nuclear homeotic functions. *Trends*

2142 *Plant Sci.* **9**: 61–64.

2143

Figure 1

Figure 2

A

B

Figure 3