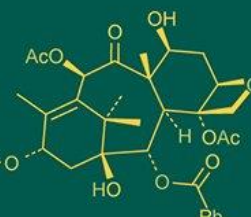
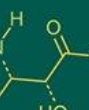
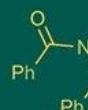


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Genomic imprinting: A new frontier in crop breeding

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Abstract

Future research should focus on functional characterization of imprinted genes and elucidating the epigenetic mechanisms that regulate their expression. Advances in genome and epigenome editing offer potential for targeted manipulation of imprinting to improve seed traits and hybrid performance. Exploring the role of imprinting in overcoming reproductive barriers, such as hybrid incompatibility and triploid block, remains a promising area. Additionally, understanding environmental influences on imprinting could reveal how epigenetic states respond to stress conditions. Identification of conserved and species-specific imprinted genes will further enhance understanding of their evolutionary significance. Integrating multi-omics approaches, including transcriptomics and epigenomics, can provide deeper insights into imprinting regulation. There is also a need to investigate the stability and heritability of imprinting across generations. Such efforts will help in translating fundamental knowledge into practical applications in crop improvement.

Keywords: Genomic Imprinting, DNA methylation, Histon modification, RNA-directed DNA methylation

Introduction

Genomic imprinting is a process that causes monoallelic expression i.e. expression of a gene from one of the two parental chromosomes rather than the typical biallelic expression observed in most genes (Falls *et al.*, 1999) ^[4] (Figure 1). In plants, this process is predominantly observed in the endosperm, a nutritive tissue that plays a central role in supporting embryo development. Over recent decades, genomic imprinting has gained considerable attention due to its significance in seed development, hybridization, and plant breeding strategies. The ability of imprinting to regulate gene expression without altering DNA sequences makes it an important component of epigenetic regulation, with far-reaching implications in agriculture and crop improvement.

Helen Crouse coined the term “imprinting” in 1960. Genomic imprinting is a process where certain genes are epigenetically marked during gamete formation, resulting in differential expression based on parental origin. It has independently evolved in flowering plants and mammals. Genomic imprinting is a non-Mendelian phenomenon that predominantly occurs in the endosperm of plants. According to Mendelian ratios, it does not matter whether the gene came from sperm or from the egg. During this process, one sperm cell fertilizes the egg cell to form the embryo, while the second sperm cell fuses with the central cell to form the triploid endosperm. This endosperm typically contains two maternal genomes and one paternal genome, and proper balance between these genomes is essential for normal seed development (Dickinson and Scholten, 2013) ^[2].

The concept of genomic imprinting revolves around the idea of monoallelic expression controlled by epigenetic marks that are established during gamete formation. These marks are maintained after fertilization and result in selective activation or silencing of specific alleles. In plants, two main categories of imprinted genes are recognized based on their expression pattern. Some genes are expressed only from the maternal allele, while the paternal allele remains silenced, whereas others are expressed from the paternal allele with the maternal allele being inactive. These expression patterns are not random but are tightly regulated by epigenetic mechanisms that ensure proper developmental outcomes (Dickinson and Scholten, 2013) ^[2].

Theories of origin

The evolutionary origin of genomic imprinting has been explained through several theories, among which the kinship theory, also known as the parental conflict hypothesis, is the most widely accepted (Figure 2). This theory proposes that imprinting evolved due to conflicting interests between maternal and paternal genomes over the allocation of resources to offspring. The maternal genome tends to distribute resources equally among all progeny to maximize overall reproductive success, while the paternal genome favors increased allocation of resources to its own offspring, even at the expense of others. This conflict leads to selective expression of genes that either promote or restrict growth, depending on their parental origin. Another explanation for the evolution of imprinting is the defense hypothesis, which suggests that imprinting may have originated as a mechanism to silence transposable elements by DNA methylation and protect genome integrity (Figure 3). The association of many imprinted genes with transposable element-rich regions supports this hypothesis (Ohnishi *et al*, 2014) [9].

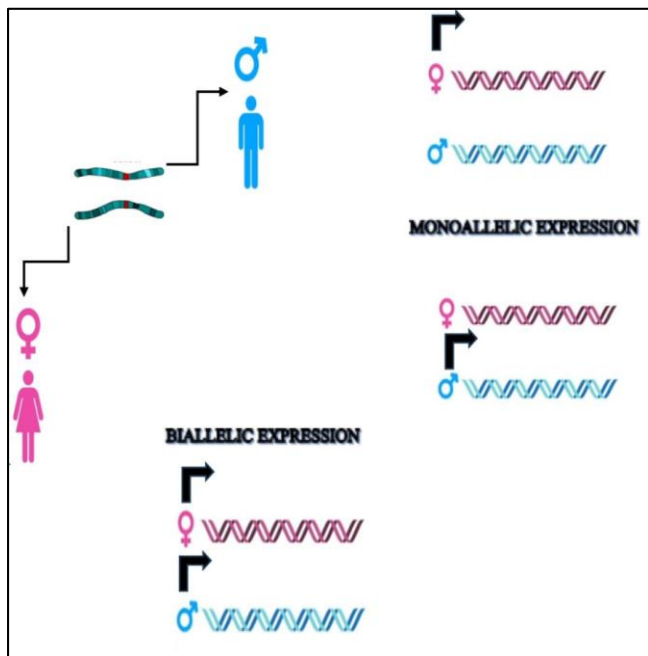


Fig 1: Biallelic and monoallelic gene expression

Types

Genomic imprinting can be classified into several types based on parental origin of expression, degree of allelic bias, and timing of establishment of epigenetic marks. Maternal imprinting refers to the silencing of the maternal allele, resulting in exclusive expression of the paternal allele, with the silencing mark typically established in the female gamete and maintained after fertilization. In contrast, paternal imprinting involves silencing of the paternal allele, allowing expression only from the maternal allele, with the imprinting mark being established during male gametogenesis. Based on the extent of allelic expression, binary imprinting represents the strictest form, where one parental allele is completely active while the other is entirely silenced, resulting in true monoallelic expression. Preferential imprinting, however, reflects a partial bias in which both alleles are expressed but one parental allele shows significantly higher expression than the other. In terms of developmental timing, primary imprinting refers to

epigenetic marks that are established during gamete formation prior to fertilization and dictate allele-specific expression in the offspring, whereas secondary imprinting arises after fertilization during early developmental stages and is influenced by primary imprinting signals and interactions among epigenetic regulatory pathways. Together, these types illustrate the complexity and dynamic regulation of genomic imprinting in controlling parent-of-origin-specific gene expression (Stupar *et al*, 2007) [12].

Mechanism

The molecular mechanisms underlying genomic imprinting are complex and involve multiple layers of epigenetic regulation. DNA methylation is one of the primary mechanisms controlling imprinting. It involves the addition of a methyl group to cytosine residues in DNA, leading to gene silencing. Differential methylation between maternal and paternal alleles plays a crucial role in establishing imprinting patterns. Enzymes such as METHYLTRANSFERASE 1 (*MET1*) are responsible for maintaining methylation, while DOMAINS REARRANGED METHYL TRANSFERASE 2 (*DRM2*) mediates *de novo* methylation. On the other hand, DEMETER (*DME*) functions as a DNA demethylase, removing methyl groups and activating gene expression. The balance between methylation and demethylation is essential for proper imprinting (Ohnishi *et al*, 2014) [9].

Another important mechanism involved in imprinting is RNA-directed DNA methylation, a pathway unique to plants. RNA-directed DNA methylation (RdDM) is a plant-specific pathway that reinforces and establishes DNA methylation at targeted genomic regions. In this mechanism, RNA Polymerase IV initiates the production of single-stranded RNA, which is then converted into double-stranded RNA by RNA-dependent RNA polymerase (RDR2). This double-stranded RNA is processed by DICER-LIKE 3 (*DCL3*) into small interfering RNAs (siRNAs), which are subsequently loaded onto ARGONAUTE proteins such as AGO4. These complexes interact with RNA Polymerase V transcripts at target loci, guiding *DRM2* to deposit methylation marks. This pathway is crucial for silencing transposable elements and regulating imprinted genes (Jiang *et al.*, 2012) [5].

Histone modifications add another layer of regulation to genomic imprinting. Histone proteins, around which DNA is wrapped, can undergo various chemical modifications such as methylation and acetylation. These modifications influence chromatin structure and gene accessibility. For instance, histone methylation at specific residues, such as H3K27me3, is associated with gene repression and is mediated by Polycomb Repressive Complex 2. This complex plays a crucial role in maintaining silenced states of imprinted genes. In contrast, histone acetylation is generally associated with gene activation, as it leads to a more relaxed chromatin structure that facilitates transcription.

These epigenetic mechanisms do not operate in isolation but interact with each other to establish stable and heritable imprinting patterns. The interplay between DNA methylation, histone modification, and RNA-mediated pathways ensures precise regulation of gene expression during seed development. Disruption in any of these mechanisms can lead to abnormal imprinting and developmental defects (Jiang *et al.*, 2012) [5].

Applications in Plant Breeding

The ability to manipulate imprinting provides new opportunities for enhancing yield and adaptability. The following are some key applications of genomic imprinting in plant breeding.

One of the most significant applications of genomic imprinting in plant breeding lies in the regulation of flowering time and adaptation through epigenetic control of key genes such as *FWA*. The *FWA* (FLOWERING WAGENINGEN) gene in *Arabidopsis thaliana* is a classic example of an imprinted gene that is normally silenced in vegetative tissues through DNA methylation but expressed when epigenetic repression is lost. When *FWA* is silenced, plants exhibit normal flowering time, whereas its ectopic expression leads to delayed flowering due to interference with the flowering pathway. This delay occurs because *FWA* indirectly represses flowering signals by affecting the expression of downstream floral integrators. In a breeding context, manipulation of imprinting at loci like *FWA* can be used to fine-tune flowering time, which is critical for environmental adaptation, crop scheduling, and yield optimization (Figure 4. I). For example, early flowering can enable stress escape mechanisms, allowing crops to complete their life cycle before the onset of drought or heat stress, while delayed flowering may be beneficial for biomass accumulation in certain crops. Thus, imprinting-based regulation of flowering genes provides a flexible tool for developing climate-resilient crop varieties (Erdmann *et al.*, 2020)^[3].

Genomic imprinting plays a crucial role in seed development through the regulation of genes controlling endosperm formation, particularly *AGL62* (AGAMOUS-LIKE 62), which functions as a key regulator of endosperm cellularization. *AGL62* is typically expressed in a parent-of-origin-specific manner and is tightly controlled by the *FIS-PRC2* (Fertilization Independent Seed-Polycomb Repressive Complex 2), an epigenetic silencing complex that includes components such as *MEA* and *FIS2*. This complex represses target genes via histone H3K27 trimethylation, ensuring proper timing of endosperm cellularization. When *AGL62* expression is properly regulated, normal endosperm development occurs; however, its overexpression or failure of repression leads to delayed or failed cellularization, resulting in defective seeds. In plant breeding, understanding the imprinting control of *AGL62* enables manipulation of endosperm development,

which directly affects seed viability and grain filling. Fine-tuning this pathway can help improve seed quality and ensure stable seed set under varying environmental conditions (Figure 4. G) (Kradolfer *et al.*, 2013)^[7].

The maternally expressed gene *OsFIE2* (*Oryza sativa* Fertilization Independent Endosperm 2) is a key component of the *FIS-PRC2* complex in rice and plays a central role in repressing autonomous endosperm development. Under normal conditions, *OsFIE2* maintains the requirement for fertilization by silencing genes that would otherwise trigger premature endosperm formation. However, mutation or downregulation of *OsFIE2* disrupts this repression, leading to autonomous endosperm development without fertilization, a phenomenon closely associated with apomixis. This ability to bypass fertilization has significant implications in plant breeding, as it allows the production of clonal seeds that preserve hybrid genotypes across generations. By manipulating imprinting at the *OsFIE2* locus, it may be possible to induce apomictic seed formation in crops, thereby fixing heterosis and eliminating the need for repeated hybrid seed production. This represents a powerful strategy for maintaining desirable traits in high-yielding varieties (Figure 4. A) (Wu *et al.*, 2023)^[13].

Genomic imprinting of paternally expressed genes (PEGs) plays a significant role in regulating seed development, with *YUCCA10* (*YUC10*) serving as an important example due to its involvement in auxin biosynthesis during endosperm formation. *YUC10* encodes a flavin monooxygenase that catalyzes a key step in the tryptophan-dependent auxin biosynthesis pathway, thereby promoting endosperm growth and proper seed filling. As a PEG, *YUC10* is predominantly expressed from the paternal allele, contributing to the regulation of endosperm proliferation and nutrient accumulation. Mutations or reduced expression of *YUC10* result in impaired auxin production, leading to reduced seed size, increased chalkiness, and defects in endosperm filling, which ultimately affect seed quality and viability. In the context of plant breeding, manipulation of imprinting at the *YUC10* locus offers a potential strategy to enhance seed development by optimizing auxin levels in the endosperm. Controlled expression of such PEGs can improve grain filling efficiency and overall yield, particularly in cereal crops where endosperm development is a key determinant of productivity (Figure 4.B) (Xu *et al.*, 2021)^[14].

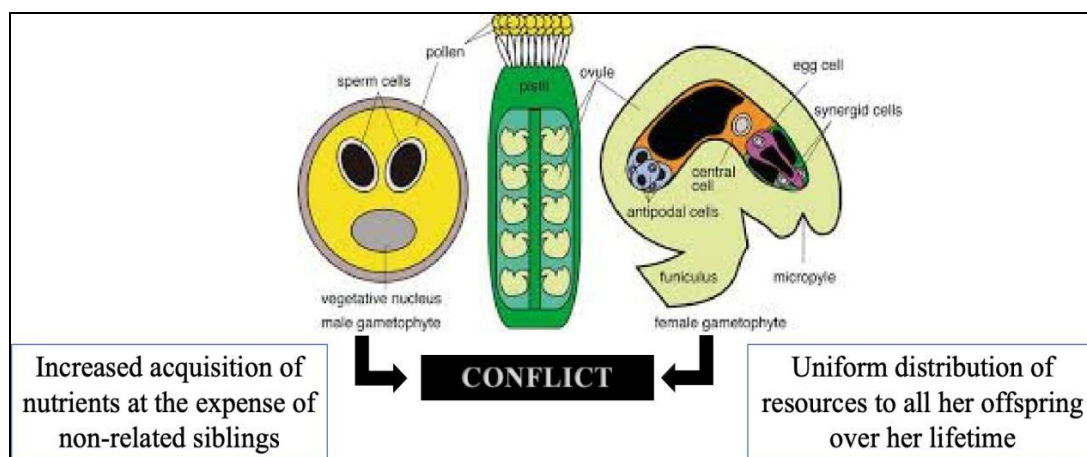


Fig 2: Representation of kinship/parental conflict hypothesis



Fig 3: Representation of defense hypothesis

Genomic imprinting of the maternally expressed gene *MEA* (*MEDEA*) plays a central role in regulating seed development by controlling endosperm proliferation and differentiation. *MEA* encodes a key component of the Polycomb Repressive Complex 2 (*PRC2*), which epigenetically silences target genes through histone H3K27 trimethylation, thereby restricting excessive cell division in the developing endosperm. As a maternally expressed gene, *MEA* ensures proper coordination between embryo and endosperm growth following fertilization. In *mea* mutants, loss of this regulatory control leads to uncontrolled cell proliferation, delayed or failed endosperm cellularization, enlarged embryos, and the formation of collapsed, white, and non-viable seeds. These abnormalities highlight the importance of *MEA*-mediated imprinting in maintaining seed viability and structural integrity. From a plant breeding perspective, understanding the imprinting function of *MEA* provides opportunities to modulate endosperm development and improve seed quality. Fine-tuning *MEA* activity could help optimize seed formation processes, particularly in crops where balanced endosperm development is critical for grain yield and stability (Figure 4.C) (Simonini *et al.*, 2021) ^[11].

Genomic imprinting plays a crucial role in seed size heterosis by regulating the parent-of-origin-specific expression of genes that control endosperm growth and nutrient allocation. In developing seeds, maternally and paternally derived genomes exert contrasting effects, where maternally expressed genes (MEGs) generally act to restrict endosperm proliferation, while paternally expressed genes (PEGs) promote cell division and resource accumulation. This antagonistic interaction establishes a balanced growth system, but in hybrids, altered dosage and expression of imprinted genes can lead to enhanced endosperm development, resulting in increased seed size. The differential regulation of imprinted genes influences key developmental processes such as cell proliferation, cellularization timing, and nutrient transfer to the embryo, all of which contribute to heterotic effects. In plant breeding, understanding the imprinting-based control of seed size provides opportunities to exploit heterosis by optimizing parental genome contributions. Manipulating the expression of specific MEGs and PEGs can enhance grain weight and yield, particularly in cereal crops where seed size is a major determinant of productivity (Figure 4. F) (Cao, 2024).

Genomic imprinting plays an important role in regulating seed dormancy and stress escape through the parent-of-origin-specific expression of genes such as *CP1*

(CYSTEINE PROTEASE 1). *CP1* encodes a cysteine protease involved in the mobilization of storage proteins during seed germination, thereby influencing the timing of dormancy release. Imprinting of *CP1* leads to monoallelic expression in early stages, which gradually transitions to biallelic expression as seeds undergo dry after-ripening. This shift in expression pattern is closely associated with dormancy status, where low or restricted *CP1* expression maintains dormancy, while increased expression promotes germination. Environmental cues such as cold exposure and after-ripening duration further modulate *CP1* activity, enabling seeds to synchronize germination with favorable conditions. In different parental crosses, variation in imprinting strength and timing of *CP1* activation can alter dormancy depth and germination behavior. From a plant breeding perspective, manipulation of imprinting at the *CP1* locus offers a strategy to control seed dormancy levels, ensuring optimal germination timing and enhancing stress escape under adverse environmental conditions such as drought or temperature stress (Figure 4. E) (Piskurewicz *et al.*, 2016) ^[10].

Genomic imprinting plays a crucial role in seed development by maintaining the proper balance between maternal and paternal genome contributions, particularly in the endosperm, where the optimal maternal-to-paternal ratio of 2:1—referred to as the endosperm balance number—is essential for normal growth. Disruption of this balance, as observed in interploidy crosses, leads to the triploid block, a reproductive barrier characterized by abnormal expression of imprinted genes, defective endosperm development, and eventual seed collapse (Figure 4. H) (Lafon-Placette *et al.*, 2016) ^[8].

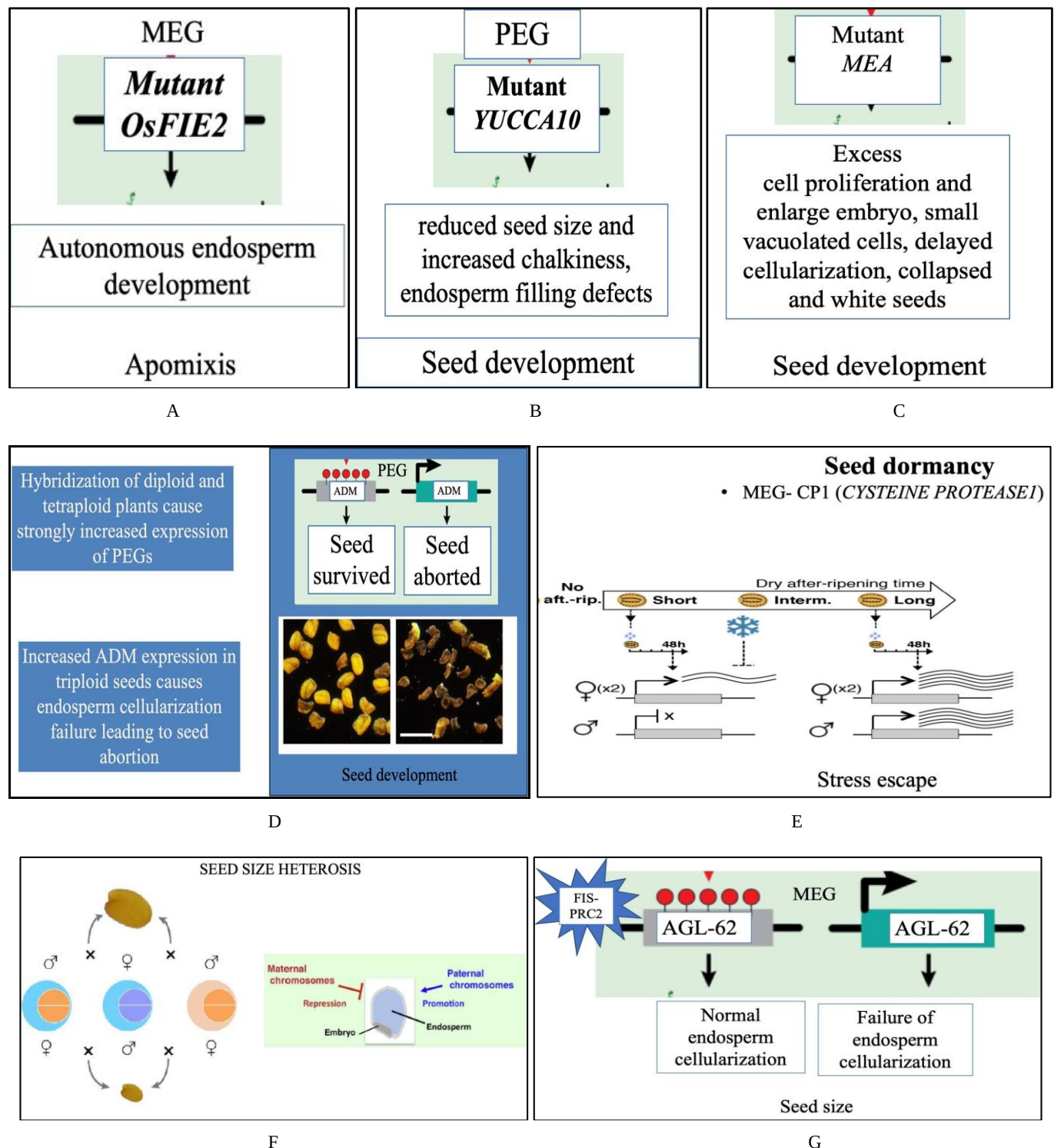
Paternally expressed genes (PEGs) such as *ADM* (*ADMETOS*), which is highly sensitive to parental genome dosage in the endosperm. *ADM* is preferentially expressed from the paternal allele and contributes to the control of endosperm proliferation and cellularization. In normal diploid crosses, balanced expression of *ADM* supports proper endosperm development and viable seed formation. However, in interploidy crosses, such as hybridization between diploid and tetraploid plants, there is an increased paternal genome contribution that leads to overexpression of PEGs, including *ADM*. This elevated *ADM* expression disrupts normal developmental regulation, resulting in failure of endosperm cellularization, abnormal tissue organization, and ultimately seed abortion. These defects are characteristic of dosage imbalance associated with the triploid block. From a plant breeding perspective, understanding the imprinting regulation of *ADM* provides a

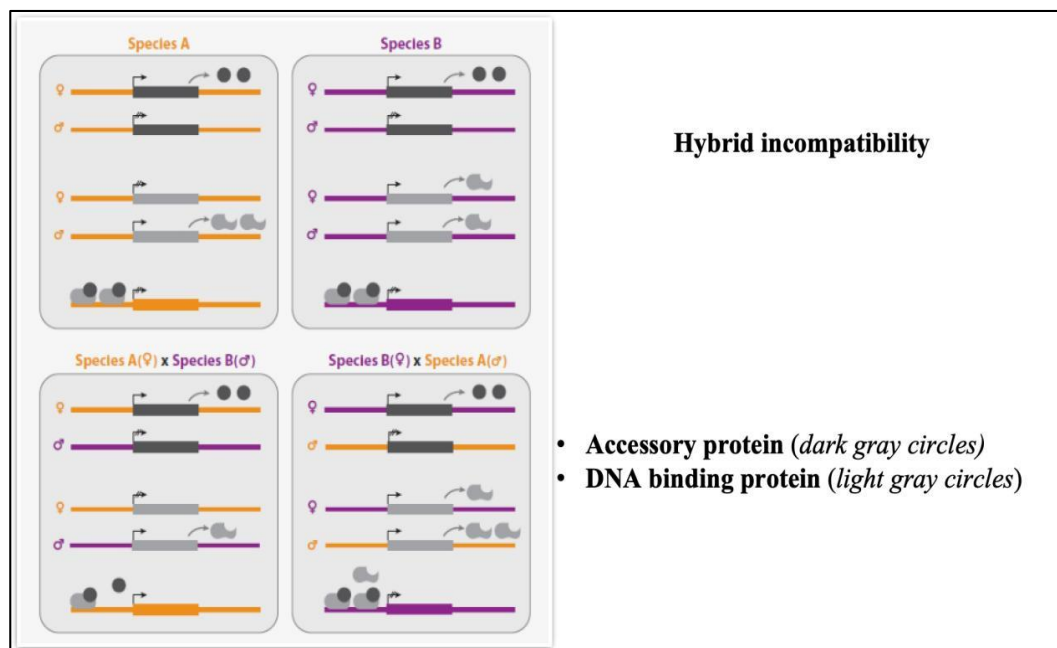
valuable approach to manipulate seed development outcomes. Modulating *ADM* expression or restoring parental genome balance can help overcome hybridization barriers and improve seed viability in interploidy crosses, thereby expanding opportunities for crop improvement (Figure 4. D) (Kradolfer *et al.*, 2013)^[7].

Future prospects

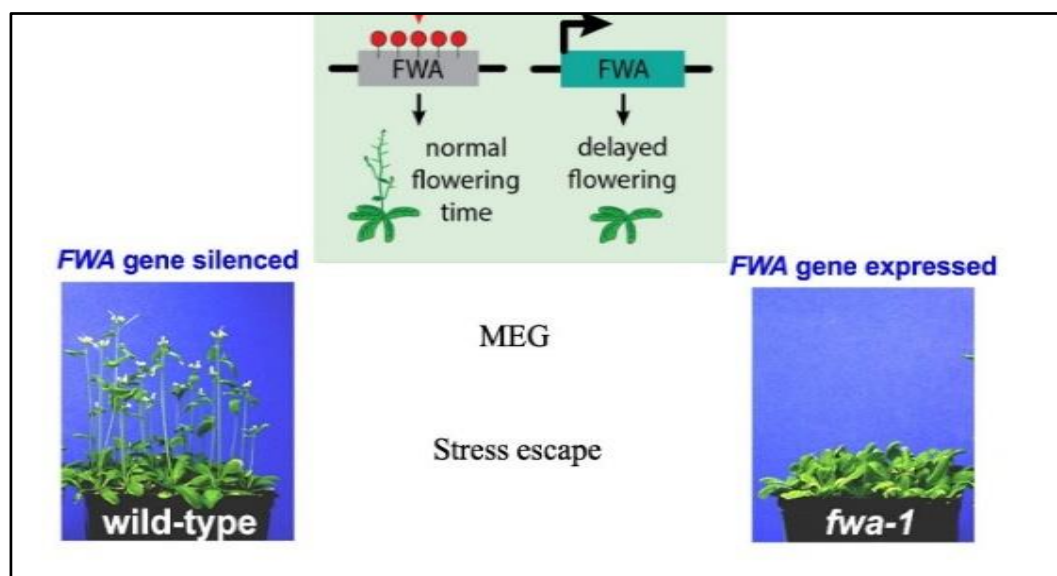
Future research should focus on functional characterization of imprinted genes and elucidating the epigenetic mechanisms that regulate their expression. Advances in genome and epigenome editing offer potential for targeted manipulation of imprinting to improve seed traits and hybrid performance. Exploring the role of imprinting in

overcoming reproductive barriers, such as hybrid incompatibility and triploid block, remains a promising area. Additionally, understanding environmental influences on imprinting could reveal how epigenetic states respond to stress conditions. Identification of conserved and species-specific imprinted genes will further enhance understanding of their evolutionary significance. Integrating multi-omics approaches, including transcriptomics and epigenomics, can provide deeper insights into imprinting regulation. There is also a need to investigate the stability and heritability of imprinting across generations. Such efforts will help in translating fundamental knowledge into practical applications in crop improvement.





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Fig 4: Applications of genomic imprinting in plant breeding

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