ISSN: 2230-9926

Available online at http://www.journalijdr.com

IJDR

International Journal of DEVELOPMENT RESEARCH

International Journal of Development Research
Vol. 3, Issue, 9, pp.055-061, September, 2013

Full Length Review Article

REVIEW ON GENE IMPRINTING DURING SEED DEVELOPMENT

Lakshmi Prasanna, K. and Bhaskaran, M.

Department of Seed science and technology, TNAU, Coimbatore

ARTICLE INFO

Article History:
Received 15h June, 2013
Received in revised form
10th July, 2013
Accepted 28th August, 2013
Published online 19th September, 2013

Key words:
Gene imprinting,
Seed,
Endosperm

ABSTRACT

Imprinting is a mitotically stable epigenetic modification that results in functional non equivalency of both parental genomes following fertilization. The phenomenon in which a set of genes is expressed according to their parent of origin. Imprinting occurs primarily in the placenta of mammals and in the endosperm of flowering plants. Imprinting is implemented to allocate limited resources to the offspring over which both paternal and maternal parents are competing. Parental conflict hypothesis and Differential dosage hypothesis explains the origin of genome imprinting in endosperm. Inter genomic conflict is evolutionary driving force for the origin of imprinting according to parental conflict hypothesis where as relative dosage of the regulatory factors in the endosperm is driving force in differential dosage hypothesis. DNA methylation, histone modification and chromatin remodelling are the mechanisms of gene imprinting (Kohler and Molisch, 2010). Three types of gene imprinting are noticed i.e., allele specific, gene specific and genome wide imprinting (Garnier *et al*., 2008). More convincing evidence of imprinting operating in the endosperm came from chromosomal translocation studies in maize. Little is known of the molecular and genetic mechanisms responsible for the endosperm acting as a hybridization barrier in plants. Gene dosage and imprinting effects in the endosperm are currently considered the 'gatekeepers' of endosperm development. Conclusive evidence linking these processes with hybrid failure remains patchy. Analysis of the molecular mechanisms regulating endosperm development in hybrids reveal the parts played by maternal determinants and parental imprinting in this complex process.

INTRODUCTION

Genomic imprinting is a genetic phenomenon by which certain genes are expressed in a parent-of-origin-specific manner. It is an inheritance process independent of the classical Mendelian inheritance. In mammals and flowering plants, imprinting occurs in the embryo as well as in embryo nourishing tissues, the placenta and the endosperm, respectively, and it has been suggested that imprinted genes control the nutrient flow from the mother to the offspring. Gene imprinting, the differential expression of maternal and paternal alleles, independently evolved in mammals and in flowering plants. A unique feature of flowering plants is a double-fertilization event in which the sperm fertilize not only the egg, which forms the embryo, but also the central cell, which develops into the endosperm (an embryo-supporting tissue). In angiosperms, double fertilization initiates two organs – embryo and endosperm – and their development is highly coordinated.

***Corresponding author:** Lakshmi Prasanna, K.*
Department of Seed science and technology, TNAU, Coimbatore

Crosstalk between these two organs and fertilization signals appear to ensure synchronized development of each organ residing in the same ovule. However, mutations in a specific class of genes disrupt such developmental synchrony and seeds eventually abort.

The Arabidopsis *FIS* class genes *MEDEA* (*MEA*), *FERTILIZATION-INDEPENDENT SEED2* (*FIS2*), *FERTILIZATION-INDEPENDENT ENDOSPERM* (*FIE*) – encode PcG components and their mutations allow the unfertilized central cell to proliferate autonomously without fertilization forming an endosperm-like structure. The characteristic seed abortion phenotype is observed only when the mutation is maternally inherited. Paternal mutations do not affect seed development. Several imprinted genes have been identified in maize and Arabidopsis. The distinctive mechanisms of gene imprinting in the endosperm, which involve DNA demethylation and histone methylation, begin in the central cell and sperm prior to fertilization. Flowering plants might have coevolved double fertilization and imprinting to prevent parthenogenetic development of the endosperm.

Origins of Endosperm Imprinting

The endosperm is an unusual tissue. It is a product of fertilization and could be considered a separate organism from the embryo. However, it does not transmit any genetic information to the next generation. Its single purpose appears to be altruistic, working and sacrificing itself to ensure the success of its embryo sibling. As mentioned previously, the evolutionary origin of endosperm is a mystery. Understanding its evolutionary origin could provide valuable insights into the mechanism of female gametophyte and seed development. Because imprinting in plants appears to be confined to the endosperm, understanding the evolutionary forces that drive imprinting will ultimately provide insight into endosperm origins. Below are hypotheses for the origin of imprinting and recent experiments that attempt to test their validity.

- Parental conflict hypothesis
- Differential dosage hypothesis

Parental conflict hypothesis

In angiosperms, the developing seeds all have the same maternal origin but potentially have different pollen donors. The parental conflict theory for the evolution of imprinting is based on the idea that the inherited maternal and paternal genomes have a different interest in the allocation of resources. For example, the maternal plant contributes genetic information to all seeds and would evolve to distribute resources to all progeny equally, whereas the paternal genome would evolve to maximize resource allocation by taking away resources from seeds resulting from less-fit pollen parents. This theoretical framework, along with the parent-of-origin effects observed in both mammals and plants, predicts that an inter genomic conflict between the maternal and paternal genomes for the allocation of resources is the evolutionary driving force for the origin of imprinting. Alleles in the maternal genome that would increase resource acquisition would be silenced, while the paternal genome would express them. Alleles in the paternal genome that would inhibit nutrient acquisition would be silenced while the maternal genomes would express them (Haig and Westoby, 1991).

The parental conflict hypothesis is supported by the results from interploidy crosses: crossing a diploid (2×) with a tetraploid (4×). In A. thaliana, crossing a 2× with a 4× pollen donor (creating a 2m:2p endosperm) results in viable seeds that are slightly larger than normal (Scott *et al*., 1998). This result appears to be consistent with the parental conflict hypothesis suggesting that the extra paternal genome would cause an overabundance of paternal imprinted genes, acquiring more resources than normal. Seed abortion in A. thaliana occurs if a 6× pollen donor is used, suggesting that an increase in dosage of the paternal genome also disrupts normal endosperm development (Scott *et al*., 1998). The reciprocal cross, a maternal 4× crossed with a paternal 2× (a 4m:1p endosperm) also produces viable but smaller than wild-type seeds, again consistent with the parental conflict theory predicting that the extra maternal copies would further inhibit nutrient acquisition (Scott *et al*., 1998). Crossing wild-type and DNA methylation mutant plants creates a similar phenotype, further supporting the link between phenotypes of interploidy crosses and the number and origin of imprinted genes (Adams *et al*., 2000).

Differential Dosage Hypothesis

The differential dosage hypothesis predicts that imprinting evolved to control the relative dosage of the regulatory factors in the endosperm (Dilkes and Comai, 2004). According to the parental conflict theory, when double fertilization arose, an inter genomic conflict between the maternal and paternal alleles for the allocation of resources was created. By contrast, according to the differential dosage hypothesis, double fertilization created an imbalance, and imprinting mechanisms were used to adjust the dosage of regulators participating in multi protein complexes. This might exert positive selection on elements, such as promoters, that influence the dosage of regulators. The differential dosage hypothesis is supported by the loss of PHE1 imprinting in interspecific crosses (Josefsson *et al*., 2006). PHE1 is normally paternally expressed and maternally silenced in the A. thaliana endosperm (Kohler *et al*., 2005). In both intra- and interploidy crosses involving A. thaliana and A. arenosa as a pollen parent, imprinting of PHE1 was lost with biallelic expression of paternal and maternal alleles in the endosperm. Imprinting of PHE1 is due to the repressive effects of a maternal Polycomb group complex. It was interpreted that the loss of PHE1 imprinting was due to an overabundance of Polycomb group complex target sites in the A. arenosa paternal genome as compared to the normally inherited A. thaliana paternal genome. Thus, the overabundance of target sites in the A. arenosa paternal genome could overwhelm the dosage of maternal Polycomb group complexes, allowing the maternal PHE1 allele to escape complete silencing. Consistent with that hypothesis, 4× A thaliana, containing a higher dosage of PcG complex, crossed with 2× A. arenosa was able to rescue seed abortion and maintain A. thaliana maternal PHE1 repression (Josefsson *et al*., 2006).

Endosperm Development that Bypasses Both Imprinting and Double Fertilization

Double fertilization, which occurs in the vast number of angiosperms, emphasizes the importance of the paternal contributed genome in the endosperm. However, a recent study using a combination of specific mutations revealed that this requirement can be bypassed (Nowack *et al*., 2007). Pollen carrying a mutation in the CDKA;1 gene, a Cdc2/Cdc28 homolog, produces only one sperm nucleus that predominately fertilizes the egg leaving the diploid central cell unfertilized (Iwakawa *et al*., 2006; Nowack *et al*., 2006). The fertilized eggs from a cdka; 1 pollen abort. The unfertilized central cell goes through a few rounds of division before seed abortion, suggesting that the paternal genome is required to complete endosperm development, and that a signal is sent from the fertilized egg to the central cell, triggering its proliferation. Surprisingly, disruptions in the maternal Polycomb group complex (mea, fis2, and fie mutations) can rescue seed abortion due to cdka;1 pollen, albeit the seeds are smaller than wild-type (Nowack *et al*., 2007). This suggests that a developing homodiploid central cell will form a functional endosperm tissue in the absence of maternal Polycomb-mediated imprinting. Development of a homodiploid endosperm with a loss of imprinting supports the hypothesis that the triploid endosperm may have originated from a diploid origin. Thus, the evolutionary origin of endosperm may have been the sexualization of the female gametophyte, rather than the acquisition of an embryo-

nourishing function by a supernumerary embryo. With the loss of Polycomb-mediated imprinting, the diploid central cell apparently has necessary molecular factors that regulate gene expression appropriately, resulting in a functional endosperm that supports embryo development. These results support the idea that one function of imprinting may be to prevent parthenogenic endosperm development. Seeds can develop when both double fertilization and components of imprinting are abolished or when both are present. This highly suggests that the two processes are intimately linked and possibly coevolved.

Mechanisms of Gene Imprinting

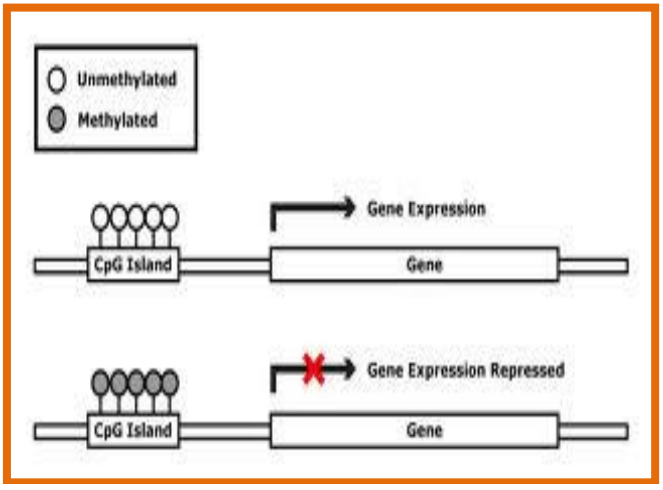
Chromatin level of genome activity is controlled at various levels of DNA and histone modifications. Covalent modifications of histones, DNA methylation, incorporation of histone variants, and other factors, such as chromatin-remodelling enzymes or small RNAs, all contribute to defining distinct chromatin states that modulate access to DNA (Berger, 2007; Kouzarides, 2007; Roudier *et al*., 2011).

The different epigenetic mechanisms include:

- Modification at the DNA level (Cytosine methylation)
- Modifications at protein level - the histone code (Histone acetylation; Histone methylation; Histone phosphorylation; Histone ubiquitination; Different types of histones)
- Chromatin remodeling - chromatin remodeling proteins.

DNA methylation

- Methylation patterns of the cytosine residues in the CpG islands serve as one of the important source code in regulating gene expression in epigenetic mechanism.
- CpG island is a stretch of DNA sequence with high frequency of CpG occurrence and C + G content of more than 50% and most commonly observed near promoter regions (Bird *et al*., 1995).
- Hyper methylation of DNA in CpG islands is associated with the maintenance of gene suppression, while hypomethylation in these regions is associated with gene expression.



- DNA methylation is regulated by DNA methyltransferases that transfer methyl groups from S-adenosyl-methionine to 5' position of cytosine residues of CpG island (Biermann and Steger, 2007).
- In plants, DNA methylation occurs at cytosine residues in CG, CHG and CHH different sequence contexts (Law and Jacobsen, 2010).
- Maintenance is carried out by DNA methyltransferase 1 (MET1), variant in methylation (VIM) and decreased DNA methylation 1 (DDM1) (at CG sites), chromomethylase 3 (CMT3) (CHG and CHH) and to some extent de novo CHG methylation is established by domains rearranged methyltransferase 2 (DRM2) in plants (Law and Jacobsen, 2010).
- Twenty-four nucleotide long (24 nt) small interfering RNAs (siRNAs) have shown to mediate De novo DNA methylation through the RNA-directed DNA methylation (RdDM) pathway.
- The DNA glycosylase DEMETER (DME) actively removes DNA methylation (Choi *et al*., 2002; Kinoshita *et al*., 2004) and might contribute to the derepression of genes (Wollmann and Berger, 2012).
- DNA demethylation can also occur passively in the absence of enzymes involved in methylation maintenance process.
- In plant endosperm and mammalian embryo, many differentially methylated regions (DMR) are present in the Imprint Control Regions (ICR) that has critical role in epigenetic regulation of imprinted domains (MacDonald, 2011).
- The methylation pattern of these DMRs are erased in germline, re-established during gametogenesis and maintained throughout the development and lifecycle.
- Further, DNA methylation is coordinated by the position and composition of nucleosomes and associated histone modifications at genome level (Hauser *et al*., 2011).

Histone modification

- The chromatin is made up of nucleosome unit, which is composed of 146 bp and wrapped around an octamer of core histones (H2A, H2B, H3 and H4) and linked by H1.
- The chemical modifications of the amino acid residue present in the N-terminal tail of these histones result in the regulation of the genes.
- The modifications viz., methylation, acetylation, phosphorylation, ubiquitylation and sumoylation at the histone tails constitute histone code (Peterson and Laniel, 2004).
- There are eight common histone modifications that are associated with active or repressed transcriptional state of chromatin (Huan and Springer, 2008).
- Modifications of histones H3 and H4, especially acetylation and methylation of histone lysine residues at N-terminal tails that protrude from the nucleosome are best understood in terms of gene regulation (Hauser *et al*., 2011).
- Histone methylation is the most prominent of the post-translational modification and is monitored by the histone methyl transferases (HMTs).
- HMTs are involved in either addition or deletion of one or two methyl groups from arginine and lysine residues (Singh *et al*., 2011).
- Histone methylation is most commonly associated with the gene silencing, methylation of H3K9 is found in heterochromatin and silenced promoters (Fischle *et al*.,

2003), but it may also associate with gene activation as in histone H3K4 dimethylation in maternal allele of maize *Mez1* and*ZmFie1* (Huan and Springer, 2008).

- Therefore, methylation of Lys9 and Lys27 of histone H3 (H3K9 and H3K27) are linked to heterochromatin and gene silencing, while methylation of Lys4 (H3K4) is linked to transcriptional activity (McDonald, 2011).
- Acetylation is the second most important posttranslational histone modification that has antagonistic role to DNA methylation.
- Increased histone acetylation at lysine residues is mediated by histone acetyl transferases (HATs) signifies active genes and deacetylation through histone deacetylases (HDACs) inhibit gene expression (Singh *et al*., 2011).
- The mouse *Gtl2* DMR of the silent paternal allele is hypoacetylated on H3 and H4, while the active maternal allele carries high levels of acetylation on both histones (Carr *et al*., 2007).
- MYST1, a MYST family protein is a acetyl transferase (HAT), which acetylates H3K16 to impact chromatin architecture (Neal *et al*., 2000) while SIRT1 is a deacetylase (HDAC) that removes acetyl groups from H1, H3 and H4 (Yi and Luo, 2010).
- Phosphorylation of histones at serine and threonine residues and ubiquitylation of lysine residues are associated with either activation or repression of gene depending on the context.
- For example, phosphorylation is usually associated with gene activation but gene silencing is seen when the histone variant H2AX is phosphorylated (Fernandez-Capetillo, 2003) and ubiquitylation of histone H2A is linked to gene silencing (Baarends *et al*., 2003) whereas ubiquitylation of H2B is linked to gene activation (Zhu *et al*., 2005).
- Attachment of small ubiquitin-related modifier proteins, termed as sumoylation is yet another process of posttranslational modification that mediate gene silencing by recruiting HDACs and heterochromatin protein 1 (Shiio and Eisenman, 2003).
- DNA methylation and histone modifications are the two interconnected processes in epigenetic mechanisms that influence each other's recruitment to the silencing complex to reinforce differential epigenetic states (Tariq and Paszkowski, 2004; Cheung and Lau, 2005).

Chromatin remodeling

- Chromatin structure is associated with the active/repressed state of a gene which is directly influenced by the DNA methylation, histone modifications and chromatin remodeling proteins.
- Open state of the chromatin makes DNA accessible to transcriptional machinery and gene expression while gene expression is repressed when the chromatin attains more compact state of heterohromatin (Fransz and Jong, 2002).
- Histone modifications may impact secondary chromatin structures through nucleosome–DNA or nucleosome–nucleosome interactions and by neutralizing charge in the histone N-terminal tails (Gilbert *et al*., 2007).
- The acetylation of histones corresponds with 'open' chromatin and enhanced transcriptional activity (Strahl and Allis, 2000) and acetylated histone tails increase the affinity of chromatin for bromo-domain proteins (e.g. HATs) and promote transcriptional activation (Turner, 2000).
- Chromatin remodeling is mediated by the alterations in location and structure of nucleosomes by ATP-dependent chromatin remodeling proteins (Narlikar *et al*., 2002; Singh *et al*., 2011) (e.g. the SWITCH2 [SWI2]/ SUCROSE NON-FERMENTING2 [SNF2] complex) and histone-modifying complexes (e.g. the histone deacetylase complex [HDAC]) (Fransz and Jong, 2002).
- Further, the repressive complex is maintained by the heterochromatin-associated protein HP1 that is thought to form a repressive complex by binding to methylated H3K9 via its chromodomain and by interacting with SUV39 (Fransz and Jong, 2002). A plant homolog of HP1, LHP1 (LIKE HETEROCHROMATIN PROTEIN1), has been reported in Arabidopsis (Gaudin *et al*., 2001).

Types of Gene Imprinting

Work with entire genomes or with parts of chromosomes indicates that the parental source of genetic information is important in determining its function. Imprinted genes whose expression varies based on the parental mode of inheritance have been identified in maize and Arabidopsis. All are imprinted in the endosperm, and some have effects on endosperm and seed size, as predicted by the parental conflict theory. Two types of imprinting have been described, allelic imprinting, in which only alleles from a certain background are subject to parent-of origin–specific gene expression, and locus imprinting, in which all known alleles from different backgrounds are under parent-of origin control.

Allelic Imprinting

For many years, the only example of an imprinted angiosperm gene was in alleles of the maize R gene. The R gene conditions anthocyanin accumulation in the aleurone (the outer cell layer of the endosperm) of maize kernels. When an RR female (red) is mated to a rr male (colorless), all of the kernels have a fully coloured aleurone. However, the reciprocal cross gives rise to kernels with mottled aleurone pigmentation, indicative of irregular anthocyanin distribution (Kermicle, 1970). This phenomenon is specific to the endosperm, and no reciprocal differences are observed in embryos or seedlings (Brink *et al*., 1970). Kermicle (1970) demonstrated that the R-mottled phenotype is not a dosage effect (i.e., RR/r endosperm versus rr/R endosperm) but is attributable to the mode of inheritance of the R allele. Kernels are mottled regardless of the number of R alleles inherited paternally and are always solidly colored if an R allele is inherited maternally. However, this phenomenon is observed only with certain R alleles; others (i.e., Rst) respond in a dosage dependent, sex-independent manner.

Alleles of other maize genes, dzr1 anda-zein, also are imprinted in the endosperm. The dzr1 locus post-transcriptionally regulates the accumulation of 10-kDa-zeins in the endosperm (Chaudhuri and Messing, 1994). Zeins are the major storage proteins of cereal endosperm and are not expressed in the embryo (Lopes and Larkins, 1993). dzr1 conditions different levels of zein accumulation in different inbred backgrounds, high in BSSS53 and low in MO17 (Chaudhuri and Messing, 1994). If a BSSS53 female is crossed to a MO17 male, zein RNA and protein accumulation

Table 1

S. No	Allelespecific imprinted genes	Tissue specific expression	Reference
1	R	Endosperm	Kermicle (1970);Ludwig *et al.*(1989)
2	Dzr-1	Endosperm	Chaudhuri & Messing(1994)
3	Zein	Endosperm	Lund *et al.* (1995a)
4	Alpha-tubulin	Endosperm	Lund *et al.* (1995b)
5	Locus-specific imprinted genes	Tissue-specific expression	
6	ZmFie1	Endosperm	Danilevskaya *et al* (2003);Gutierrez-Marcos *et al.* (2006)
7	ZmFie2	Endosperm	Hermon *et al* (2007); Haun and Springer (2008)
8	Nrp1	Endosperm	Danilevskaya *et al.*(2003); Gutierrez-Marcos *et al.* (2006); Hermon *et al*. (2007)
9	Peg1		Guo *et al.*(2003); Haun & Springer (2008)
10	Meg1	Endosperm	Gutierrez-Marcos *et al.* (2003)
11	Mez1	Endosperm	Gutierrez-Marcos *et al.* (2004)
12	Mee1	Embryo& Endosperm	Haun *et al.*(2007); Haun & Springer (2008)
13	VIM5	Endosperm	Jahnke and Scholten (2009)
14.	YUC10	Endosperm	Zhang *et al.* (2011)

(SOURCE : Bhavani *et al*., 2012)

are high, as in BSSS53. RNA and protein accumulation are low in the reciprocal cross, as in MO17. A simple dosage explanation for this effect was ruled out by using B translocations to introduce two copies of MO17 dzr1 or BSSS53 dzr1 through the male. Regardless of the number of paternal copies of BSSS53 dzr1 present, endosperm that receives maternal MO17 dzr1 has low zein accumulation. The reverse is true of endosperm that maternally inherits BSSS53 dzr1, regardless of the number of paternal copies of MO17 dzr1. However, in crosses to a different background, BSSS53 dzr1, unlike MO17 dzr1, behaved in a dosage-dependent manner (Chaudhuri and Messing, 1994). The authors concluded that the MO17 allele of dzr1 is imprinted such that it has an effect when inherited maternally but not when inherited paternally. Although the high and low accumulation of the10-kD zein is linked to dzr1, it is not known if the MO17 dzr1 locus itself is actually expressed differentially depending on its parent of origin. Imprinting of specifica-zein alleles also has been found in the maize endosperm. Using RNase protection assays, it was demonstrated that members of the SF2 subfamily (a-zeins are divided into four subfamilies based on their sequence homology) are expressed maternally but not paternally. This is specific to the W64A inbred background (Lund *et al*., 1995).

Locus Imprinting

The imprinting of the Arabidopsis MEDEA (MEA) gene has been the subject of intense study. Mutations in MEA were isolated in screens based on two different phenotypes: silique elongation (reproductive development) without fertilization (Chaudhury *et al*., 1997; Kiyosue *et al*., 1999) and seed abortion (Grossniklaus *et al*., 1998). In the absence of fertilization, the diploid central cell nucleus of the mea female gametophyte divides to form a multinucleate central cell, reminiscent of syncytial endosperm, that develops to the point of cellularization (Chaudhury *et al*., 1997; Kiyosue *et al*., 1999). There is no evidence that the proliferating central cell constitutes an endosperm tissue capable of nourishing an embryo (Friedman, 2001). Sporophytic fertilization programs also are activated in the absence of fertilization: the maternal seed coat develops and the silique elongates. The seed-like structures eventually atrophy. This phenotype is only partially penetrant (Kiyosue *et al*., 1999). Thus, one function of MEA is to prevent replication of the central cell nucleus in the absence of fertilization. Additional MEA functions also can be deduced based on the post-fertilization phenotype. Seeds from fertilized mea female gametophytes undergo endosperm over proliferation, embryo arrest, and eventual abortion. mea endosperm nuclei continue to proliferate after the wild-type endosperm has ceased to replicate, resulting in large, balloon-like developing seeds (Kiyosue *et al*., 1999) in which endosperm cellularization is delayed (Grossniklaus *et al*., 1998). Compared with that in the wild type, the mea CZE is specifically enlarged and expanded to more anterior regions (Sørensen *et al*., 2001). Thus, another function of MEA is to restrict endosperm proliferation after fertilization. Also, each morphogenetic stage is lengthened in the embryo, and it arrests at the heart stage (Grossniklaus *et al*., 1998; Kiyosue *et al*., 1999). Eventually, the endosperm collapses around the embryo and the seed aborts. It is unknown whether the embryo and endosperm phenotypes are both direct consequences of the mea mutation or whether one is a primary defect and the other a downstream event. MEA encodes a SET-domain Polycomb group protein that is homologous with Drosophila Enhancer of Zeste [E(z)] (Grossniklaus *et al*., 1998; Luo *et al*., 1999; Kiyosue *et al*., 1999). Polycomb group proteins form complexes that can modify histones and maintain repressed states of gene expression (Orlando, 2003).

The mutant phenotypes suggest that MEA maintains the repression of genes involved in cell proliferation. The type I MADS box gene PHERES1 was identified recently as a direct target of MEA (Kohler *et al*., 2003). The mea mutation exhibits parent-of-origin effects on seed development. Phenotypic consequences arise only when mea is inherited through the female. If a MEA/mea female is crossed to a wild-type male, ~50% of the seeds abort. If a wild-type female is crossed to a MEA/mea male, all of the seeds are normal and viable. Thus, seed viability depends only on the genotype of the maternal MEA allele; the paternal allele is dispensable and cannot zygotically rescue a seed that has inherited a mutant maternal mea allele. Occasionally, mea can be transmitted to the next generation through the female, allowing the generation of mea/mea plants with between 95 and 100% seed abortion. MEA is imprinted in the endosperm. The maternal allele is expressed and the paternal allele is silenced. Kinoshita *et al*. (1999) used ecotype polymorphisms in the MEA coding sequence to distinguish maternal and paternal allele expression by reverse transcription (RT) PCR. Seeds were dissected into embryo and endosperm plus maternal seed coat at 6, 7, and 8 DAP, corresponding to the torpedo, walking stick, and early maturation stages of embryo development. Expression from both maternal and paternal alleles was found at all stages of

embryo development. Only the maternal allele was expressed in the endosperm. MEA is expressed from both alleles in vegetative tissues, including the seedling, rosette leaf, stem, and root (Kinoshita *et al*., 1999).

Different imprinted genes and their origin

Genetic imprinting is found to have major role in many key developmental processes and genome dosage is one of the factors contributing to the imprinting. Genome dosage has reported to have direct implication on the seed size in maize. To date, the scientific community is still debating on its role in evolution and significance in the process of crop improvement. Advanced technologies like genome-wide approaches may contribute in helping the researchers to unravel the potential mechanism of genetic imprinting and its possible benefits to crop improvement.

REFERENCES

Adams, S., Vinkenoog, R., Spielman, M., Dickinson, H.G., and Scott, R.J. (2000). Parent-of-origin effects on seed development in Arabidopsis thaliana require DNA methylation. Development 127, 2493–2502.

Baarends W.M., Wassenaar E., Hoogerbrugge J.W., van Cappellen G., Roest H.P., Vreeburg J., Ooms M., Hoeijmakers J.H., Grootegoed J.A., 2003, Loss of HR6B ubiquitin-conjugating activity results in damaged synaptonemal complex structure and increased crossing-over frequency during the male meiotic prophase, Mol. Cell Biol., 23: 1151-1162

Berger, F., Fitz Gerald, J.N., and Ingouff, M. (2007). Arabidopsis as a model for understanding the basics of endosperm development. In Endosperm: Developmental and Molecular Biology, O.-A. Olsen, ed. (Berlin: Springer-Verlag), pp. 91–110.

Bhavani P., Harinikumar K M., Shashidhar H E and Sajad Majeed Zargar. 2012, Genetic Imprinting in Maize, Maize Genomics and Genetics. 3(3):13-21.

Biermann K., and Steger K., 2007, Epigenetics in male germ cells, J. Androl., 28: 466-480.

Bird A., Tate P., Nan X., Campoy J., Meehan R., Cross S., Tweedie S., Charlton J., and Macleod, D., 1995, Studies of DNA methylation in animals, J. Cell Sci. Suppl., 19: 37-39.

Carr M.S., Yevtodiyenko A., Schmidt C.L., and Schmidt J.V., 2007, Allele-specific histone modifications regulate expression of the Dlk1-Gtl2 imprinted domain, Genomics, 89(2): 280-290

Cheung P., and Lau P., 2005, Epigenetic regulation by histone methylation and histone variants, Molecular Endocrinology, 19(3): 563-573.

Choi Y., Gehring M., Johnson L., Hannon M., Harada J.J., Goldberg RB., Jacobsen S.E., and Fischer R.L., 2002, DEMETER, a DNA glycosylase domain protein, is required for endosperm gene imprinting and seed viability in Arabidopsis, Cell, 110: 33-42.

Dilkes, B.P., and Comai, L. (2004). A differential dosage hypothesis for parental effects in seed development. Plant Cell 16, 3174–3180.

Fernandez-Capetillo O., Mahadevaiah S.K., Celeste A., Romanienko P.J., Camerini-Otero R.D., Bonner W.M., Manova K., Burgoyne P., and Nussenzweig A., 2003, H2AX is required for chromatin remodeling and inactivation of sex chromosomes in male mouse meiosis, Dev. Cell, 4: 497-508.

Fischle W., Wang Y., Jacobs S.A., Kim Y., Allis C.D., and Khorasanizadeh S., 2003, Molecular basis for the discrimination of repressive methyllysine marks in histone H3 by polycomb and HP1 chromodomains, Genes Dev., 17: 1870-1881.

Haig, D., and Westoby, M. (1989). Parent-specific gene expression and the triploid endosperm. Am. Nat.134, 147–155.

Haun W.J., and Springer N.M., 2008, Maternal and paternal alleles exhibit differential histone methylation and acetylation at maize imprinted genes, Plant J., 56: 903-912.

Iwakawa, H., Shinmyo, A., and Sekine, M. (2006). Arabidopsis CDKA; 1, acdc2 homologue, controls proliferation of generative cells in male gametogenesis. Plant J. 45, 819–831.

Josefsson, C., Dilkes, B., and Comai, L. (2006). Parent-dependent loss of gene silencing during interspecies hybridization. Curr. Biol. 16, 1322–1328.

Kinoshita T., Miura A., Choi Y., Kinoshita Y., Cao X., Jacobsen S.E., Fischer R.L., and Kakutani T., 2004, One-way control of FWA imprinting in Arabidopsis endosperm by DNA methylation, Science, 303: 521-523.

Kohler, C., Hennig, L., Bouveret, R., Gheyselinck, J., Grossniklaus, U., and Gruissem, W. (2003a). Arabidopsis MSI1 is a component of the MEA/FIE Polycomb group complex and required for seed development. EMBO J. 22, 4804–4814.

Kouzarides T., 2007, Chromatin modifications and their function, Cell, 128: 693-705.

Law J.A., and Jacobsen S.E., 2010, Establishing, maintaining and modifying DNA methylation patterns in plants and animals, Nat. Rev. Genet., 11: 204-220.

MacDonald W.A., 2011, Epigenetic mechanisms of genomic imprinting: common themes in the regulation of imprinted regions in mammals, plants, and insects, Genetics Research International. 38, 63–67.

Neal K.C., Pannuti A., Smith E.R., and Lucchesi J.C., 2000, A new human member of the MYST family of histone acetyl transferases with high sequence similarity to drosophila MOF, Biochim. Biophys. Acta, 1490: 170-174.

Nowack, M.K., Grini, P.E., Jakoby, M.J., Lafos, M., Koncz, C., and Schnittger, A. (2006). A positive signal from the fertilization of the egg cell sets off endosperm proliferation in angiosperm embryogenesis. Nat. Genet. 38, 63–67.

Nowack, M.K., Shirzadi, R., Dissmeyer, N., Dolf, A., Endl, E., Grini, P.E., and Schnittger, A. (2007). Bypassing genomic imprinting allows seed development. Nature 447, 312–315.

Roudier F., Ahmed I., Berard C., Sarazin A., Mary-Huard T., Cortijo S., Bouyer D., Caillieux E., Duvernois-Berthet E., Al-Shikhley L., Giraut L., Despres B., Drevensek S., Barneche F., Derozier S., Brunaud V., Aubourg S., Schnittger A., Bowler C., Martin-Magniette M., Robin S., Caboche M., and Colot V., 2011, Integrative epigenomic mapping defines four main chromatin states in Arabidopsis, EMBO J., 2011: 1-11.

Scott RJ, Spielman M, Bailey J, Dickinson HG (1998). Parent-oforigin effects on seed development in Arabidopsis thaliana. Development125: 3329–3341.

Shiio Y., and Eisenman R.N., 2003, Histone sumoylation is associated with transcriptional repression, Proc. Natl. Acad. Sci., 100: 13225-13230.

Singh R., Avery K., and Agarwal A., 2011, Epigenetics, spermatogenesis and male infertility, Mutation Research, 727: 62-71.

Tariq M., and Paszkowski J., 2004, DNA and histone methylation in plants, Trends in Genetics, 20(6): 244-251.

Wollmann H., and Berger F., 2012, Epigenetic reprogramming during plant reproduction and seed development, Current Opinion in Plant Biology, 15: 63-69.

Yi J., and Luo J., 2010, SIRT1 and p53, effect on cancer, senescence and beyond, Biochim. Biophys. Acta, 1804: 1684-1689.

Zhu B., Zheng Y., Pham A.D., Mandal S.S., Erdjument-Bromage H., Tempst P., Reinberg D., 2005, Mono-ubiquitination of human histone H2B: the factors involved and their roles in HOX gene regulation, Molecular Cell, 20: 601-611.

****