Tansley insight

Paternal imprinting in *Marchantia polymorpha*

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Received: *22 June 2023*
Accepted: *9 October 2023*

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New Phytologist (2024) **241:** 1000–1006
doi: 10.1111/nph.19377

Key words: bryophytes, epigenetic, imprinting, polycomb, sexual reproduction, vivipary.

Summary

We are becoming aware of a growing number of organisms that do not express genetic information equally from both parents as a result of an epigenetic phenomenon called genomic imprinting. Recently, it was shown that the entire paternal genome is repressed during the diploid phase of the life cycle of the liverwort *Marchantia polymorpha*. The deposition of the repressive epigenetic mark H3K27me3 on the male pronucleus is responsible for the imprinted state, which is reset by the end of meiosis. Here, we put these recent reports in perspective of other forms of imprinting and discuss the potential mechanisms of imprinting in bryophytes and the causes of its evolution.

I. Introduction

In animals and flowering plants, both alleles are usually equally expressed, yet crosses between maize and Arabidopsis lines of different ploidy (Brink & Cooper, 1947; Kermicle & Alleman, 1990; Scott *et al.*, 1998) and pronuclear transfer experiments in mouse eggs (McGrath & Solter, 1984; Surani *et al.*, 1984) showed that maternal and paternal alleles are not equivalent during early development. It was later recognized that the parental origin of an allele impacts its activity at specific stages or in specific tissues (reviewed in Tucci *et al.*, 2019; Batista & Köhler, 2020) and that during gametogenesis, an epigenetic mark is deposited on one parental allele but not the other (Ferguson-Smith, 2011; Barlow & Bartolomei, 2014). After fertilization, the allele carrying the mark is imprinted and its expression silenced, although there are far more complex forms of *trans*regulation between imprinted regions and the coding regions they control (Cleaton *et al.*, 2014; Sanli & Feil, 2015). In mammals, parental genomic imprinting involves DNA methylation at the majority of the *c.* 200 imprinted loci (Tucci *et al.*, 2019) and imprints of a smaller number of paternally expressed loci result from the deposition in the oocyte of two histone posttranslational modifications by the Polycomb Repressive Complexes 1 and 2 (PRC1 and PRC2): ubiquitination of histone H2A (H2Aub) and trimethylation of lysine 27 on histone H3 (H3K27me3), respectively (Inoue *et al.*, 2017; Chen & Zhang, 2020; Mei *et al.*, 2021; Inoue, 2023). Flowering plants also display genomic imprinting, which results from an interplay between DNA methylation and H3K27me3 (Choi *et al.*, 2002; Kinoshita *et al.*, 2004; Gehring *et al.*, 2006; Jullien *et al.*, 2006a,b; Makarevich *et al.*, 2008; Tiwari *et al.*, 2008; Hsieh *et al.*, 2011;

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Wolff *et al.*, 2011; Vu *et al.*, 2013; Moreno-Romero *et al.*, 2016, 2019; Hornslien *et al.*, 2019).

Recently, a new type of genomic imprinting has been described in bryophytes, a monophyletic group of plants that includes mosses, liverworts, and hornworts. The life cycle of bryophytes is dominated by a haploid vegetative and reproductive phase while fertilization initiates the development of short-lived, multicellular diploid sporophytes (Fig. 1). In the model liverwort *Marchantia polymorpha* (hereafter referred to as Marchantia), the entire paternal genome is repressed in the sporophyte by a mechanism termed paternal chromosome repression (PCR) (Montgomery *et al.*, 2022). The imprinting breaks down during meiosis and the paternal alleles are expressed in the next haploid generation (Montgomery & Berger, 2023).

Here, we summarize key aspects of PCR in Marchantia and discuss the potential mechanisms that cause imprinting, and its biological significance in relation to its evolutionary origin.

II. Imprinting in Marchantia embryos

Paternal chromosome repression is distinct from polycomb-mediated imprinting in flowering plants. In flowering plants, specific maternal loci are targeted for the deposition or maintenance of H3K27me3, which results in their silencing in the endosperm (Batista & Köhler, 2020). By contrast, in PCR, it is the entire paternal genome that is coated and repressed by deposition of H3K27me3 (Fig. 1). Immunofluorescence microscopy highlighted the dense spread of H3K27me3 along the length of each mitotic chromosome, and the formation of large, dense heterochromatic foci within interphase nuclei. The heterochromatinization of the paternal genome in Marchantia is reminiscent of paternal genome elimination in insects (Crouse, 1960; Bain *et al.*, 2021; de la Filia *et al.*, 2021; Hodson *et al.*, 2023). While the coverage of an entire chromosome by a repressive histone modification can also be seen in imprinted X chromosome inactivation (XCI) in mammals (Żylicz & Heard, 2020), PCR does not affect the entirety o the male sex chromosome, which is rather occupied by H3K9 methylation (constitutive heterochromatin; Montgomery *et al.*, 2022). Genetically, PCR depends on two maternally encoded, embryo-specific catalytic subunits of PRC2, E(z)2 and E(z)3. Knockout of these two genes in females results in a loss of PCR in embryos when crossed to wild-type males, and ultimately in embryo lethality, suggesting that PCR is essential for embryo development (Montgomery *et al.*, 2022).

As H3K27me3 is associated with transcriptional repression, PCR results in gene expression primarily from the maternal genome (Fig. 1). Therefore, despite being genetically diploid, Marchantia embryos are functionally haploid and under a predominant maternal control, although exceptions exist (Dierschke *et al.*, 2021). One advantage of diploid organisms is thought to be the presence of two alleles of every gene, which allows for the survival of an individual in the event of a recessive deleterious mutation of one allele. However, this advantage is less relevant in Marchantia because it already spends most of its life cycle in a haploid state. Therefore, any deleterious mutations would already be exposed to selection before sexual reproduction.

5-methylcytosine DNA methylation often acts as the imprinting mark in placental mammals and flowering plants. DNA methylation is also paternally biased in Marchantia embryos; however, it is also paternally biased in *ez(2)/ez(3)* mutant embryos wherein PCR is disrupted (Montgomery & Berger, 2023). Additionally, DNA methylation remains paternally biased after meiosis and through spore maturation; thus, DNA methylation is likely decoupled from the repression of the paternal genome, but this remains to be tested directly.

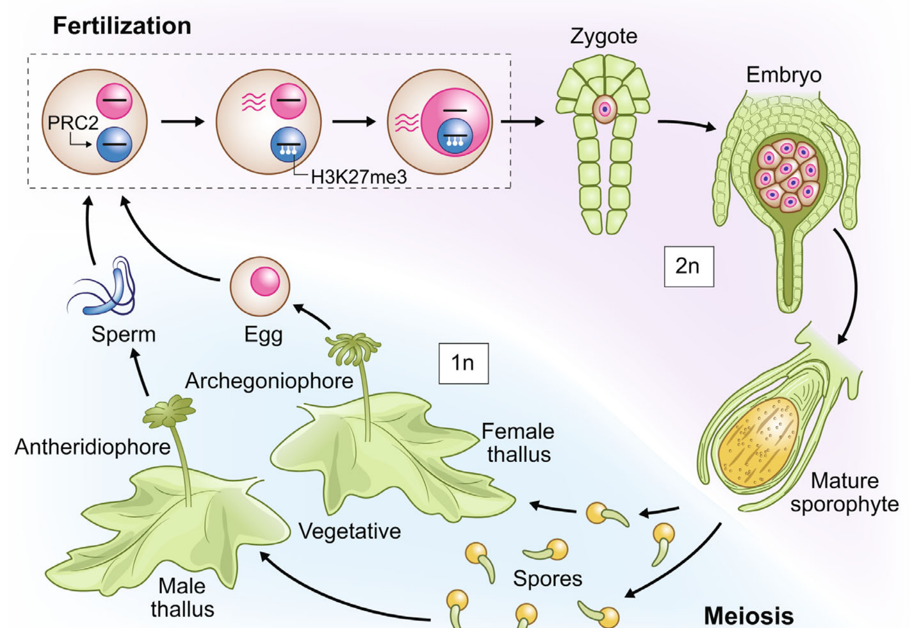

Fig. 1 Genomic imprinting in the life cycle of Marchantia. Multicellular tissues are indicated in green and bold lines, whereas single cells are shown in light lines. Sperm and egg fuse at fertilization to form a zygote and initiate the diploid phase of the life cycle. PRC2 targets the paternal pronucleus (blue circle) to deposit H3K27me3 (white lollipops). Transcription takes place from the maternal genome (pink lines) and continues throughout the diploid life cycle phase after the fusion of pronuclei. About 3 wk after fertilization, meiosis produces haploid spores, initiates the haploid life cycle phase, and is coincident to the resetting of imprinting. Haploid spores grow into the dominant haploid vegetative plants, which later initiate sexual reproduction to make eggs and sperm.

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III. Erasure of imprinting

More than 95% of cell of the sporophyte eventually undergo meiosis, which produces haploid spores (Shimamura, 2015). Spores do not divide and are transcriptionally active until they reach a mature dry state and are released in the environment. If the repression of paternal alleles in the Marchantia diploid sporophyte were to be maintained after meiosis, each spore would inherit a random assortment of 50% of its genes in a repressed state. There are few paralogs in the Marchantia genome; hence, the persistence of paternal imprints in the spore would most likely be lethal, or at the very least deleterious. Thus, PCR was predicted to be erased before gene expression in the haploid generation. By profiling parental biases in H3K27me3 and establishing the timing of meiosis, we have recently reported that PCR is erased by the completion of meiosis (Montgomery & Berger, 2023). Likewise, it appears that the initiation of erasure is coincident with the onset of meiosis, placing meiosis as a key event in the reprogramming of imprinting in Marchantia (Fig. 1). The loss of imprinting via the equalization of H3K27me3 parental bias may occur in several different ways. First, H3K27me3 may be deposited over the entirety of the maternal genome; however, this is unlikely due to its inefficiency, as it would require completely shutting down both genomes for a period of time. Second, H3K27me3 may be erased from the paternal genome by specific H3K27 demethylases. Third, nucleosomes bearing modified H3 may be shuffled between paternal and maternal genomes by a general event that would deposit unmarked nucleosomes causing a massive dilution of nucleosomes bearing H3K27me3. Quantitative measurements of H3K27me3 and tracking of histone dynamics would greatly aid in parsing out the relative contributions of each potential mode of action. Thus, while we know when imprinting is erased, the mechanism of erasure and the signal to initiate erasure remain unknown. Yet, the reprogramming of imprinting by meiosis not only accounts for the necessary removal of repression from half of the parental genomes inherited by spores, but also has deeper implications on the role of meiosis. Viewing meiosis as the step that removes epigenetic modifications accumulated through mitosis was proposed as a central biological significance other than recombination by Robin Holliday in 1984. The recent demonstration of the reconfiguration of chromatin during meiosis in mammals (Alavattam *et al.*, 2019; Vallot & Tachibana, 2020) and transcriptional remodeling in plant meiocytes (Nelms & Walbot, 2019) provides additional support to this view. With meiosis temporally and spatially separated from gametogenesis and fertilization, Marchantia provides an ideal model to understand the association of meiosis with epigenetic reprogramming that is necessary to prevent the inheritance of epigenetic marks.

IV. Establishment of imprinting

In Marchantia, imprinting is established during fertilization, yet the molecular mechanisms remain elusive. During spermatogenesis, nucleosomes are replaced by protamines, basic proteins that aid in the compaction of the paternal genome in sperm (Reynolds & Wolfe, 1978; D'Ippolito *et al.*, 2019). As in most animals, male and female pronuclei remain separated until 4 d after fertilization (Hisanaga *et al.*, 2021) and protamines are removed and histones are deposited to restore nucleosome-based chromatin (Montgomery *et al.*, 2022). This is followed by the deposition of H3K27me3 marks on the paternal pronucleus. After fusion of the male and female pronuclei and subsequent division of the zygotic nucleus, the paternal genome remains coated with H3K27me3 while the maternal genome is comparatively devoid of this mark, resulting in PCR (Fig. 1).

The deposition of H3K27me3 is catalyzed by the E(z) subunit of PRC2 (Margueron & Reinberg, 2011), yet diverse mechanisms exist across eukaryotes to mediate the targeting of PRC2 to specific loci or chromosomes. In Drosophila, PRC2 is targeted to loci by the binding of recruitment factors to specific DNA sequences, called polycomb response elements. Given that PCR affects entire autosomes, it is unlikely that such sequence-specific factors exist in Marchantia to mediate PCR. However, based on a model of deposition of H3K27me3 by PRC2 over the FLC locus in Arabidopsis (Costa & Dean, 2019), we envisage that PRC2 may first be targeted to specific regions of chromosomes and that from these initiation sites, a second form of PRC2 is recruited to extend the mark from one initiation site to the next. Which protein could recruit PRC2 to the paternal genome and not the maternal genome remains unclear.

In mammals, the entire X chromosome is coated in repressive chromatin modifications, in a fashion similar to PCR in Marchantia. Imprinted XCI in mammals is initiated by the expression of the long noncoding RNA *Xist* from the imprinted X chromosome (Borsani *et al.*, 1991; Brockdorff *et al.*, 1991; Brown *et al.*, 1991; Chow & Heard, 2009). Thus, it is plausible that noncoding RNAs produced from the Marchantia paternal genome may guide the recruitment of PRC2 (Fig. 2). However, if such noncoding RNAs were to exist in Marchantia, they must reside in at least one locus per chromosome. They must also be silenced on the maternal genome and their expression from the paternal genome would be elicited in the unique period when the paternal genome is devoid of nucleosomes between the loss of protamines and before the deposition of nucleosomes. An investigation of the transcriptional competency and output of the paternal genome at one and two days after fertilization may yield further insights.

As an alternative, the process of protamine removal and nucleosome incorporation may facilitate the recruitment of PRC2 (Fig. 2). In Drosophila, protamines are removed by the ASF protein complex and nucleosomes containing H3.3 are deposited by the HIRA protein complex (Loppin *et al.*, 2005; Pchelintsev *et al.*, 2013; Horard *et al.*, 2018). This model is conserved in animals, but we know very little about this step of fertilization in plants (Loppin & Berger, 2020). In Marchantia, the recruitment of HIRA may be coupled to that of PRC2, thus naturally distinguishing the male pronucleus from the maternal pronucleus, leading to the specific import of PRC2 and deposition of H3K27me3 on the paternal genome (Fig. 2). The targeting of PRC2 may also happen after the completion of nucleosome deposition by proteins residing on the newly assembled nuclear envelope (Fig. 2).

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Canonical genomic imprinting is mediated by DNA methylation, yet loci initially imprinted by DNA methylation may transition to a secondary H3K27me3 imprint (Fig. 2). Intriguingly, sperm DNA hosts higher levels of DNA methylation in sperm than female reproductive tissues (Schmid *et al.*, 2018), which was later revealed to largely be due to 4mC rather than the usual 5mC (Walker *et al.*, 2021). The nature of DNA methylation of the egg cell is unknown, but in the sporophyte, an imbalanced higher level of DNA methylation persists on the paternal genome (Montgomery & Berger, 2023), suggesting an epigenetic asymmetry between maternal and paternal genomes involving DNA methylation at the time of fertilization which could guide PRC2 to the paternal genome.

V. Speculations on the biological significance of imprinting

Montgomery *et al.* (2022) adds bryophytes as another lineage in which imprinting has evolved independently. It is probable that genomic imprinting is present in all liverworts, as maternal effects on spores have been observed (Allen, 1925; Doyle, 1960), though its presence in mosses and hornworts is unknown (Fig. 3). Nonetheless, the innovation of genomic imprinting in liverworts allows for the reconsideration of the conditions under which imprinting may evolve.

Imprinting thus evolved independently in placental mammals, flowering plants, and bryophytes (Fig. 3), leading many to

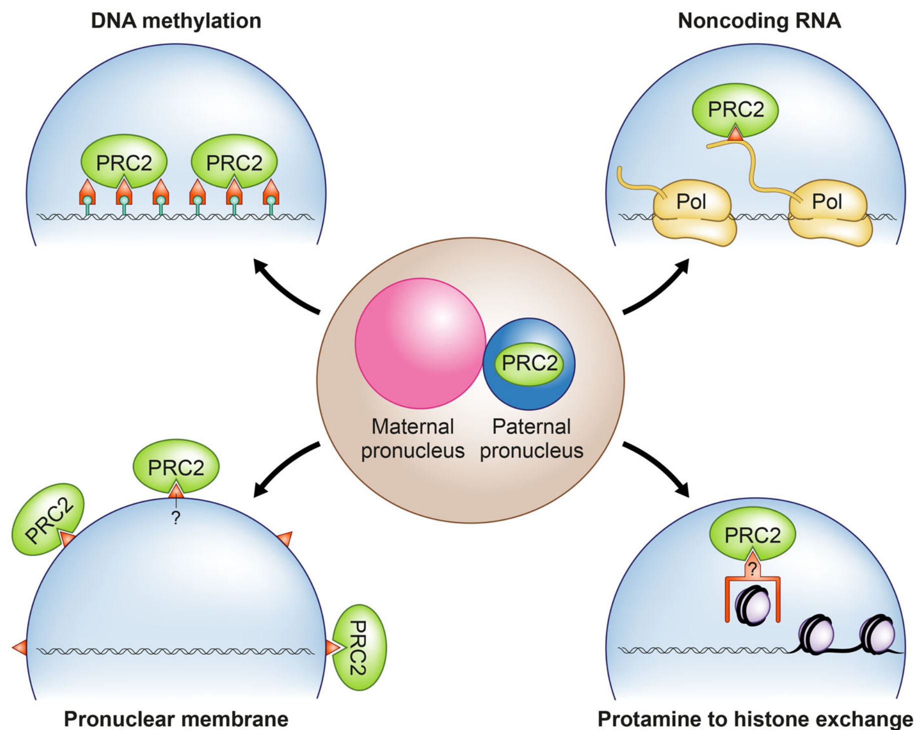

Fig. 2 Hypotheses of polycomb recruitment to paternal chromatin. PRC2 is recruited to the paternal pronucleus (teal circle) before fusion with the maternal pronucleus (pink circle) (middle). Noncoding RNAs produced by RNA polymerase may recruit PRC2 (top right). A protein involved in the deposition of nucleosomes into paternal chromatin after protamine removal may recruit PRC2 (bottom right). Pronuclear membrane proteins from plasmogamy may direct PRC2 to the paternal pronucleus (bottom left). DNA methylation (green lollipops) may recruit a binding factor which in turn recruits PRC2 (top left). Grey lines depict DNA, black lines depict RNA.

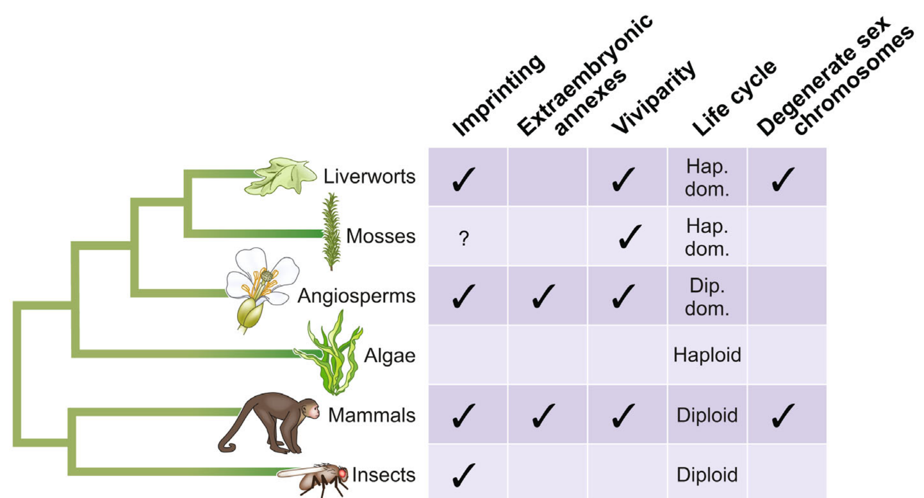

Fig. 3 Distribution of imprinting and associated traits across evolution. Imprinting evolved independently in bryophytes, flowering plants, mammals, and insects; however, it is unknown whether imprinting exists in mosses. Extraembryonic annexes were thought to be highly coincident with the evolution of imprinting, but imprinting in Marchantia highlights a more likely link with viviparity, the development of embryos on or in mothers. Land plants have an alternation between multicellular haploid and diploid life cycle phases, with the haploid stage longer lived in liverworts and mosses, and the diploid stage longer lived in flowering plants. Sex chromosomes, when present, are not degenerate in mosses and flowering plants, yet are degenerate in liverworts and mammals.

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wonder what conditions prompted the innovation of genomic imprinting (Rodrigues & Zilberman, 2015; Patten *et al.*, 2016; Sazhenova & Lebedev, 2021). The biological significance and evolution of genomic imprinting in bryophytes may be considered based on several nonmutually exclusive models: maternal dependence of the embryo, impact of sex chromosomes, adaptation to gene dosage, and developmentally programmed epigenetic source-sink transcriptional regulation (Haig & Wilczek, 2006; Shaw *et al.*, 2011; Haig, 2013; Carey *et al.*, 2021; Montgomery & Berger, 2021). The development of embryos on or in mothers, termed viviparity, may favor the innovation of genomic imprinting to modulate the communication or flow of resources between mother and embryo (Haig & Westoby, 1989; Wolf & Hager, 2006). Especially, a specialized tissue called the foot develops in the sporophyte at the junction with the maternal tissue. Even if PCR is not limited to this tissue, there might be differential regulation of PCR in the few cells types of the sporophyte. Paternal chromosome repression results in the diploid embryos becoming functionally haploid, potentially reverting gene dosage levels to those optimized in the dominant haploid stage or from the haploid algal ancestors of land plants. Likewise, PCR may serve to sequester PRC2 by acting as a sink, thus allowing sporophyte-specific genes on the maternal genome repressed by PRC2 to become expressed. Genomic imprinting has also been implicated in forming postzygotic reproductive barriers (Wolff *et al.*, 2015). Interspecific hybrids in the Marchantia genus have been found in nature (Linde *et al.*, 2020), and PCR may affect this process in two ways. First, hybrids may be possible if there are deleterious interactions between alleles at a single locus (Chae *et al.*, 2014), as the silenced paternal alleles would not interfere with the function of the maternal alleles. Second, hybrids would be unable to form when the paternal genome is unable to be silenced due to a lack of recognition of the factor that distinguishes the parental genomes, as mutants with active paternal genomes in the embryo are not viable.

Overall, although at first glance different forms of genomic imprinting may appear similar, they likely arose under different selective pressures. Attempting to unify a similar cause of imprinting in different organisms might blind us to the diversity of its roots. We thus expect the discovery of diverse facets of imprinting in many more species (Rodrigues & Zilberman, 2015; Montgomery & Berger, 2021; Sazhenova & Lebedev, 2021). They utilize the same repressive epigenetic machinery in novel ways and further studies of imprinting will be a source of new mechanisms and conceptual insights.

Acknowledgements

This work was funded by FWF grants P26887, P28320, P32054, P33380 to FB and EMBO Postdoctoral Fellowship ALTF-0067 to SAM.

Competing interests

None declared.

Author contributions

SAM and FB wrote the manuscript.

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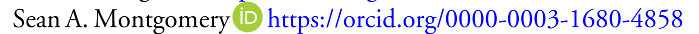
Sean A. Montgomery  https://orcid.org/0000-0003-1680-4858

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