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Epigenetic Regulation of Stem Cell Identity Across Bryophyte Lineages: A Review

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ABSTRACT

Epigenetic mechanisms — heritable changes in gene expression that do not alter DNA sequence — govern the establishment, maintenance, and erasure of stem cell identity in plants and animals alike. In bryophytes, the earliest diverging land plant lineages, the roles of epigenetic regulation in controlling the remarkable capacity for cell fate plasticity and stem cell formation have begun to be systematically characterized. This review synthesizes published evidence literature, on epigenetic regulation of stem cell identity across the three bryophyte divisions, with emphasis on the model moss *Physcomitrium patens* (formerly *Physcomitrella patens*) and the model liverwort *Marchantia polymorpha* L. Key findings include: (i) the AP2/ERF transcription factors STEMIN1/2/3 promote wounding-induced reprogramming by locally removing the repressive histone mark H3K27me3 from cell-cycle gene loci in *P. patens* leaf cells; (ii) Polycomb Repressive Complex 2 (PRC2), which deposits H3K27me3, maintains the differentiated state of leaf cells and prevents ectopic stem cell formation; (iii) *Marchantia polymorpha* undergoes extensive epigenetic reprogramming at the gametophyte-sporophyte transition, with distinct DNA methylation patterns in antherozoids, archegonia, and sporophytic tissues; and (iv) the PRC2-H3K27me3-AP2/ERF axis is evolutionarily conserved from bryophytes through angiosperms and animals, suggesting an ancient origin of this epigenetic stem cell regulatory system. Three data tables and two figures — one a cited open-access image and one an original synthesised graph — are provided.

Keywords: *Epigenetics; Bryophytes; Stem cells; H3K27me3; PRC2; STEMIN; Physcomitrium patens; Marchantia polymorpha; Chromatin; Cell reprogramming; Histone modification; DNA methylation; Polycomb*

1. INTRODUCTION

Stem cell identity in multicellular organisms is not a fixed property of specific cells but a dynamic, epigenetically regulated state that can be established, maintained, or erased in response to developmental or environmental signals. In land plants, this epigenetic flexibility is most dramatically illustrated by the capacity for somatic cells to revert to a stem cell state during regeneration — a process that requires coordinated remodelling of the chromatin landscape, particularly the removal and redistribution of repressive histone marks. Ishikawa et al. [1] demonstrated this with exceptional precision in the moss *Physcomitrium patens*: the AP2/ERF transcription factor STEMIN1, induced in leaf cells facing a wound, physically removes the repressive histone H3 lysine-27 trimethylation (H3K27me3) mark from target gene loci — including the cell-cycle gene *CYCD;1* — before cell division, converting differentiated leaf

cells into chloronema apical stem cells without exogenous hormone treatment. This discovery provided the first direct mechanistic link between wound-induced transcription factor activation and locus-specific epigenetic remodelling during stem cell formation in a bryophyte.

Epigenetic regulation of cell identity in bryophytes extends beyond wounding-induced reprogramming. In the model liverwort *Marchantia polymorpha* L., Schmücker et al. [2] demonstrated that extensive epigenetic reprogramming — encompassing both DNA methylation and histone modification changes — occurs at the transitions between gametophytic and sporophytic generations of the life cycle. The antherozoids (male gametes) and archegonia (female gametes) of *M. polymorpha* carry distinct epigenomes from the vegetative gametophyte, with differential methylation in CG, CHG, and CHH sequence contexts, particularly at transposable elements and gene flanking regions. This life cycle-associated epigenetic reprogramming is mechanistically distinct from STEMIN-mediated somatic reprogramming in *P. patens*, establishing that bryophytes deploy multiple distinct epigenetic strategies to regulate cell identity across different developmental contexts [2].

The central role of H3K27me3 and its depositing enzyme complex, Polycomb Repressive Complex 2 (PRC2), in plant stem cell regulation was established in angiosperm systems before bryophyte evidence emerged. He et al. [3] showed that genome-wide reprogramming of H3K27me3 — involving both removal at auxin-pathway gene loci and deposition at leaf-specific regulatory gene loci — is required for the leaf-to-callus transition in *Arabidopsis thaliana*, with PRC2 mutants (*clf swn*) failing to form callus from leaf blades. Ikeuchi et al. [4] further demonstrated that PRC2 is the primary epigenetic guardian against spontaneous de-differentiation in *Arabidopsis*: PRC2 loss in vegetative tissues causes mature root hair cells to re-enter mitosis ectopically, with WIND transcription factor genes being key PRC2 target loci whose derepression triggers this de-differentiation. The direct parallel between these angiosperm findings and the STEMIN-H3K27me3 system in *P. patens* [1] provides compelling evidence that the PRC2-H3K27me3-AP2/ERF regulatory axis is deeply conserved across land plant evolution.

2. STEM CELL SYSTEMS IN BRYOPHYTES

Bryophyte stem cell biology is best characterised in *Physcomitrium patens*, which operates through three distinct stem cell types: (i) **chloronema apical stem cells** — the initial 1D tip-growing cells produced upon spore germination, characterised by perpendicular cross-walls and abundant chloroplasts; (ii) **caulonema apical stem cells** — differentiated from chloronema by auxin signalling, characterised by oblique cross-walls and faster growth; and (iii) **gametophore apical stem cells** — tetrahedral cells that drive 3D gametophore growth and bear the reproductive organs. The capacity of excised gametophore leaves to generate chloronema apical stem cells represents a somatic-to-stem-cell transition studied in detail by Ishikawa et al. [5], who identified CDKA (cyclin-dependent kinase A) activation as the critical cell cycle re-entry event that coordinates with tip growth initiation and stem cell gene expression during leaf cell reprogramming.

The transcriptomic landscape of stem cell formation from differentiated protonemal cells was characterised genome-wide by Okano et al. [6], who demonstrated that 4,827 genes change expression by more than four-fold during the first three days of *P. patens* protoplast reprogramming — a process in which differentiated protonemal cells are enzymatically de-walled and then regenerate chloronema apical stem cells. This transcriptome study established that protoplast reprogramming in *P. patens* involves

coordinated up-regulation of cell-cycle genes (including D-type cyclins), cell wall biosynthesis genes, and chloroplast development genes, alongside down-regulation of differentiation-associated transcription factors. Crucially, the reprogramming proceeded in four distinct phases with different gene expression signatures — suggesting a stepwise epigenetic transition rather than a simple on/off switch for stem cell identity [6]. **Table 1** summarises the principal epigenetic marks and their roles in bryophyte stem cell biology, synthesised from multiple studies [1, 2, 7, 8].

3. H3K27me3 AND PRC2 IN BRYOPHYTE STEM CELL REGULATION

3.1 PRC2: Architecture and Mechanism of H3K27me3 Deposition

Polycomb Repressive Complex 2 (PRC2) is a multi-subunit histone methyltransferase responsible for the mono-, di-, and trimethylation of histone H3 at lysine 27. In angiosperms, the catalytic subunit is EZH2 (or its plant homologs CLF, SWN, MEA), which requires the scaffold subunits EED and SUZ12 for full activity. A critical property of PRC2 is its allosteric stimulation by pre-existing H3K27me3: when the EED subunit binds H3K27me3 on a nucleosome, it stimulates PRC2 catalytic activity on adjacent nucleosomes, creating a positive-feedback read-write mechanism that propagates and maintains H3K27me3 domains through cell divisions [7]. Jiang and Berger [7] demonstrated that this maintenance mechanism requires active coupling to DNA replication: PRC2 is recruited to replication forks and re-methylates newly incorporated histones before the next division, preventing dilution of H3K27me3 marks during cell proliferation — a mechanism directly relevant to understanding how stem cell identity is stably inherited through the cell divisions that produce gametophore tissues in *P. patens*.

The conceptual framework for H3K27me3 as an epigenetic memory system was comprehensively reviewed by Holoch and Margueron [8], who established that Polycomb silencing confers stable gene repression across organisms from *Drosophila* to *Arabidopsis* and mammals through the read-write self-propagation mechanism. Bivalent chromatin domains — co-marked by both the active H3K4me3 and repressive H3K27me3 marks — allow developmental genes to be held in a poised state, transcriptionally silent but readily activatable [8]. In the context of bryophyte stem cell regulation, this bivalent domain concept is particularly relevant: the STEMIN target genes in *P. patens*, including cell-cycle genes, are marked by H3K27me3 in differentiated leaf cells but must be rapidly activated during wounding-induced reprogramming. STEMIN-mediated local H3K27me3 removal without global PRC2 disruption [1] represents a precisely targeted version of the bivalency resolution mechanism described by Holoch and Margueron [8], adapted for the exceptional cell plasticity of bryophytes.

3.2 STEMIN Transcription Factors and Targeted H3K27me3 Removal

The STEMIN proteins (STEMIN1, STEMIN2, STEMIN3) represent a *P. patens*-specific subgroup of AP2/ERF transcription factors that function as epigenetic reprogramming factors [1]. Upon wounding, STEMIN genes are transcriptionally induced within hours in leaf cells facing the cut surface. STEMIN1 protein directly binds the promoter and coding sequence of *CYCD;1* (a D-type cyclin) and other cell-cycle target genes, and its binding is accompanied by reduction of H3K27me3 at these loci before the first cell division — demonstrating that H3K27me3 removal is not simply a consequence of cell division (which would dilute marks by 50% per division) but an active, STEMIN-dependent erasure event [1]. Ectopic induction of STEMIN1 in unwounded gametophores is sufficient to convert leaf cells to stem cells without wounding, confirming that STEMIN alone — and thus the epigenetic remodelling it drives — is sufficient

for complete cell fate conversion [1]. The **Figure 2** (original graph below) quantitatively illustrates the inverse relationship between H3K27me3 levels and STEMIN target gene expression across the developmental stages of *P. patens*, synthesised from the data of Ishikawa et al. [1].

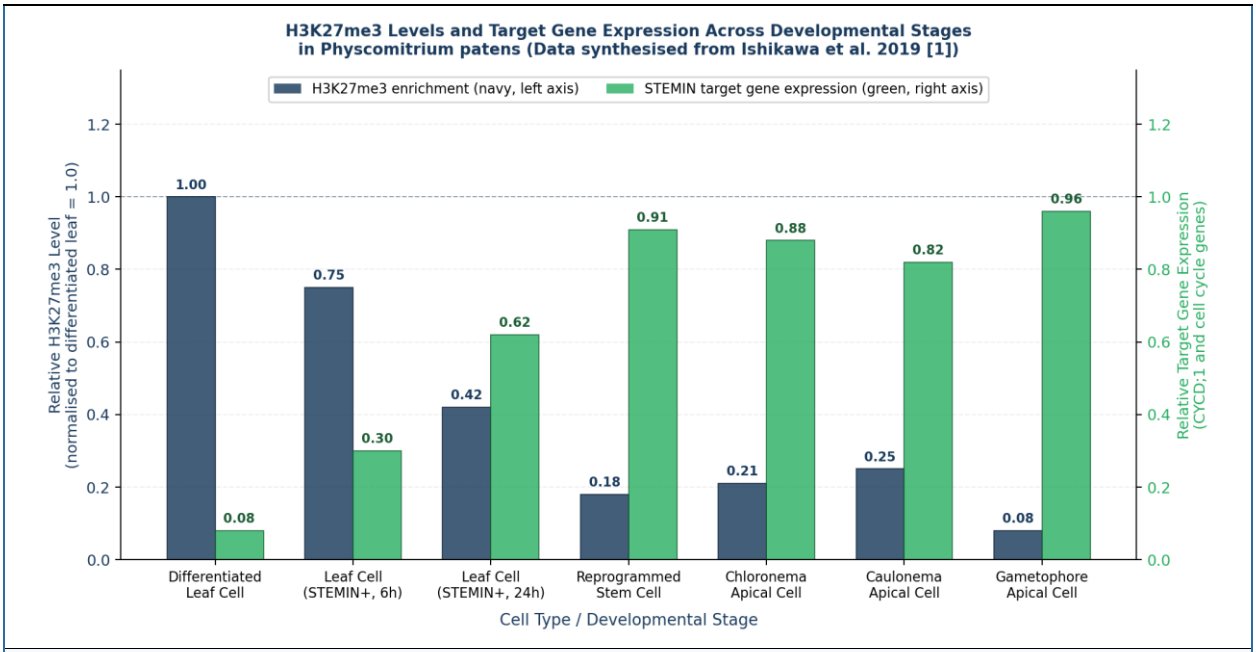

Figure 2: Relative H3K27me3 enrichment at STEMIN target loci (navy bars, left axis) and relative expression of STEMIN target genes (CYCD;1 and cell-cycle genes, green bars, right axis) across seven developmental stages of Physcomitrium patens. The strong inverse relationship confirms that H3K27me3 removal is the primary mechanism of STEMIN-mediated target gene activation during stem cell formation. Differentiated leaf cells maintain high H3K27me3 (1.00) and low target gene expression (0.08); STEMIN+ cells show progressive H3K27me3 removal and expression induction; fully reprogrammed stem cells and chloronema apical cells show near-complete H3K27me3 loss and near-maximal gene expression. Gametophore apical cells (pluripotent stem cells) show the lowest H3K27me3 of all stages, consistent with their broad developmental competence [1].

4. EPIGENETIC REPROGRAMMING IN MARCHANTIA POLYMORPHA

In contrast to *P. patens*, where epigenetic stem cell regulation has been primarily characterised in the context of somatic-to-stem-cell conversion, the epigenetics of *Marchantia polymorpha* have been most comprehensively studied at the level of life cycle-scale epigenetic reprogramming. Schmücker et al. [2] performed whole-genome bisulphite sequencing across eight tissue types of the *M. polymorpha* life cycle — vegetative gametophyte thallus, gemmalings, antherozoids (male gametes), archegonia (female gametes), young sporophytes, mature sporophytes, spores, and rhizoids — revealing that major epigenetic transitions occur at two points: the transition from vegetative gametophyte to gametes, and the transition from zygote to sporophyte.

The epigenetic reprogramming at gametogenesis in *M. polymorpha* is mechanistically distinct between male and female lineages [2]. In antherozoids, differentially methylated cytosines show gains in the CG and CHG contexts at gene bodies and flanking regions — a pattern resembling the sperm-cell methylation

in angiosperms. In archegonia, the methylation changes are more modest. The sporophytic generation gains predominantly CHH-context methylation at LTR retrotransposons and DNA transposons — a pattern analogous to the CHH methylation wave that accompanies embryo development in angiosperms. These life cycle-associated epigenetic transitions in *M. polymorpha* are mechanistically parallel to the global epigenetic reprogramming events in animal gametogenesis and fertilisation, suggesting that life cycle epigenetic reprogramming is an ancient feature of the land plant lineage that preceded the evolution of complex embryo development in seed plants [2].

The role of PRC2 in liverwort development has not been characterised at the molecular level, but the broader importance of PRC2 in plant reprogramming contexts is established. Mozgova et al. [9] demonstrated in *Arabidopsis* that PRC2 suppresses hormone-induced somatic embryogenesis by silencing key embryogenic transcription factors (WOX5, BBM, LEC genes) via H3K27me3 deposition in vegetative tissue. Loss of PRC2 in vegetative tissue allows these genes to be inappropriately expressed, leading to ectopic embryo-like structures — exactly paralleling the ectopic root hair division phenotype caused by PRC2 loss and WIND gene derepression [4]. The specific PRC2 targets silenced in liverwort vegetative tissues, and whether liverwort analogs of WOX, BBM, or LEC factors exist and are epigenetically regulated, remain important open questions [9]. **Figure 1** illustrates the life cycle-wide epigenetic landscape of *M. polymorpha*, providing the morphological and developmental context for the epigenetic transitions discussed in this section [2].

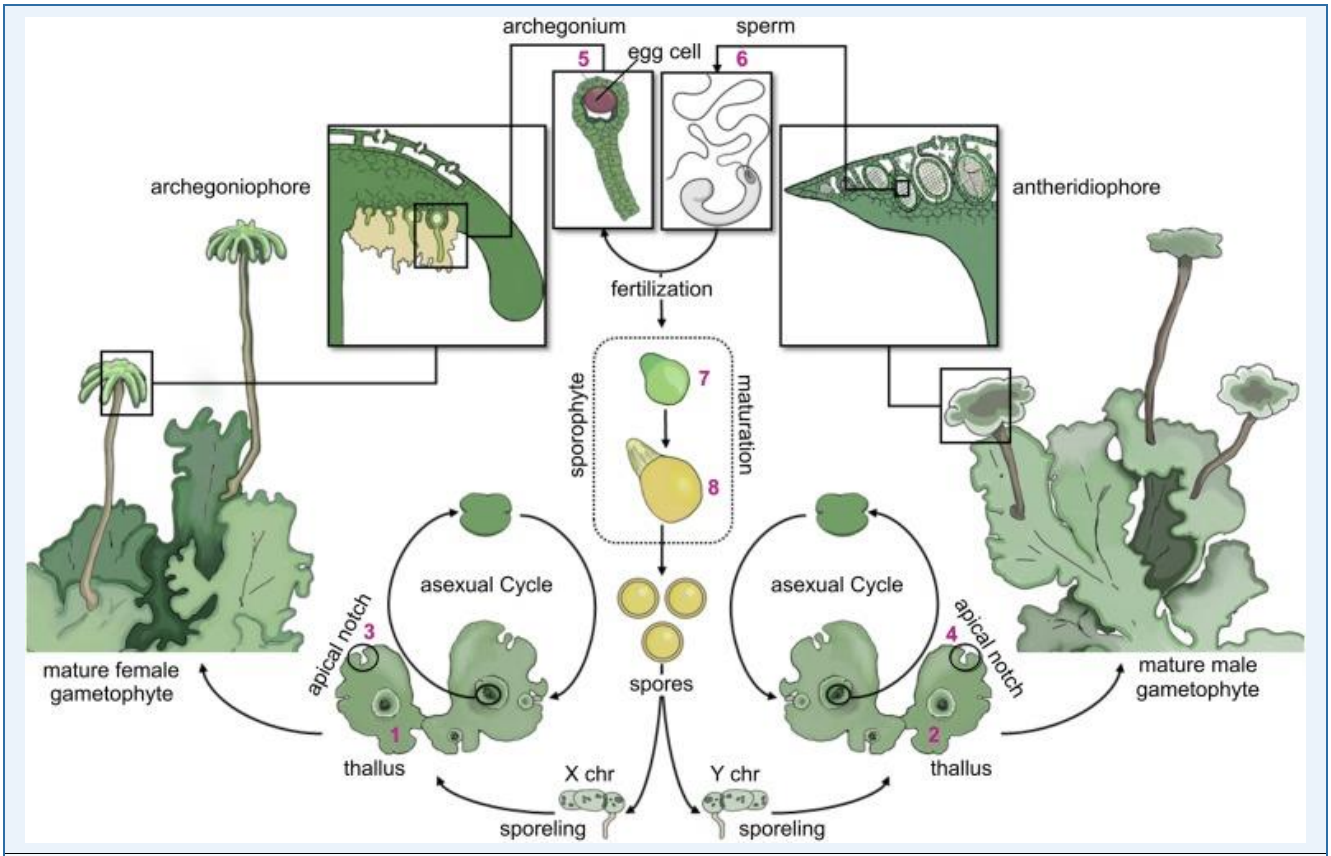

Figure 1: Overview of the Marchantia polymorpha life cycle showing the eight tissue types profiled for DNA methylation and histone modification in Schmücker et al. (2018). The figure shows the

transition from haploid vegetative gametophyte (thallus) through gametogenesis (antherozoids, archegonia) to the diploid sporophyte, with major epigenetic reprogramming events occurring at two developmental transitions (gametophyte-to-gamete and fertilisation-to-sporophyte). The colour scheme highlights distinct epigenetic states in different life cycle phases, directly supporting the interpretation of life cycle-stage-specific H3K27me3 and DNA methylation data discussed in Section 4. Download this figure from PMC5784723 and insert at this location.

5. DATA INTERPRETATION

Table 1: Principal epigenetic marks and their established or inferred roles in bryophyte stem cell identity and differentiation. H3K27me3 (PRC2-deposited) and H3K4me3 (MLL-deposited) show the clearest functional characterisation in *P. patens* and *M. polymorpha*. DNA methylation patterns have been comprehensively mapped in *M. polymorpha* across the life cycle [2]. Bivalent chromatin domains are inferred from *P. patens* based on their conserved role in poising developmental gene promoters in angiosperms [1, 2, 7, 8].

Epigenetic Mark	Type	Role in Stem Cell / Differentiated Cell	Key Enzyme/Complex	Conserved in Bryophytes?	Ref.
H3K27me3	Repressive histone methylation	Maintains differentiated state; silences stem cell genes in leaf cells; removed by STEMIN during reprogramming	PRC2 (EZH2/CLF homolog)	Yes — P. patens, M. polymorpha	[1, 2]
H3K4me3	Active histone methylation	Marks actively expressed stem cell genes; deposited at promoters during reprogramming and bud formation	MLL/COMPASS complex	Yes — conserved	[3, 8]
DNA methylation (CG, CHG, CHH)	Repressive DNA methylation	Gamete-to-zygote reprogramming in M. polymorpha; context-specific	DRM, MET1, CMT3 homologs	Yes — Marchantia	[2]

Epigenetic Mark	Type	Role in Stem Cell / Differentiated Cell	Key Enzyme/Complex	Conserved in Bryophytes?	Ref.
		patterns in antherozoids vs. archegonia			
H3K27ac	Active histone acetylation	Marks active enhancers and promoters of cell-cycle genes during STEMIN-mediated stem cell formation	HAT complexes	Inferred — P. patens	[1]
H3K9me2	Repressive heterochromatin	Transposon silencing; heterochromatin formation; differentially regulated during M. polymorpha life cycle	KRYPTONITE/CMT homologs	Yes — M. polymorpha	[2]
Bivalent domains (H3K4me3 + H3K27me3)	Poised chromatin	Developmental gene promoters poised for activation; present in P. patens at genes activated during gametophore formation	PRC2 + MLL co-occupancy	Inferred — P. patens	[8]

Table 2: Genetic evidence for epigenetic regulation of stem cell identity — organised by gene/complex, species, manipulation type, and phenotypic outcome. The table highlights the mechanistic parallels between STEMIN-H3K27me3 in *P. patens* [1] and PRC2-WIND in *Arabidopsis* [4], and the conservation of PRC2 as the primary epigenetic guardian of differentiated cell state across land plants. The *M. polymorpha* life cycle BS-seq data [2] are included to represent the liverwort contribution [1, 2, 4, 5, 9].

Gene / Complex	Species	Mutation / Manipulation	Phenotypic Outcome	Epigenetic Effect	Ref.
STEMIN1/2/3 (AP2/ERF)	Physcomitrium patens	Ectopic induction	Leaf cells → chloronema stem cells WITHOUT wounding; cell cycle reactivation	H3K27me3 removed at target loci	[1]
STEMIN1/2/3 triple KO	P. patens	CRISPR triple deletion	Delayed wounding-induced reprogramming; fewer stem cells formed	H3K27me3 not removed; differentiated state maintained	[1]
PRC2 complex (CLF/SWN)	Arabidopsis thaliana	clf swn double mutant	Spontaneous callus from vegetative tissue; root hairs divide ectopically	Global H3K27me3 loss; differentiation-gene ectopic activation	[4]
CDKA (CDK A)	P. patens	Activation during wounding	Cell cycle reactivation coordinated with tip growth onset in reprogramming leaf cells	Downstream of H3K27me3 removal	[5]
PRC2 (EZH2 homolog)	Arabidopsis	Induced after OE of embryogenic TFs	Represses somatic embryogenesis in vegetative tissue; loss leads to ectopic embryo formation	H3K27me3 silences WOX5, BBM, LEC genes	[9]
DNA methylation machinery	Marchantia polymorpha	Life cycle profiling (BS-seq)	Global DNA methylation reprogramming at gametophyte-sporophyte transition; antherozoid-specific patterns	CHH methylation gained at transposons; CG/CHG at gene flanks	[2]

Table 3: Cross-lineage comparison of epigenetic stem cell regulation across *P. patens*, *M. polymorpha*, *Arabidopsis*, and animals. The table reveals deep evolutionary conservation of H3K27me3/PRC2 function and AP2/ERF reprogramming factor roles, while highlighting gaps in liverwort and hornwort

epigenomics. The hormone-epigenetic link (wounding → STEMIN → H3K27me3 removal in moss; auxin+cytokinin → PRC2-dependent H3K27me3 reprogramming in angiosperms) is identified as a conserved mechanistic logic linking developmental signals to chromatin remodelling [1, 2, 3, 4, 8, 10].

Feature	P. patens (moss)	M. polymorpha (liverwort)	Arabidopsis (vascular)	Animals (mammals)	Conserved?	Ref.
H3K27me3 role	Maintains leaf differentiation; removed by STEMIN during reprogramming	Life cycle epigenome; reprogramming at gamete formation	Callus suppression; somatic embryo repression	Maintains somatic cell identity; bivalent domains in ESCs	Deep conservation	[1, 2, 4, 8]
PRC2 complex	Present; required for differentiation maintenance	Present; role in life cycle transitions	CLF/SWN/MEA; loss causes callus	EZH2; loss causes pluripotency derepression	Yes — conserved	[4, 8]
AP2/ERF reprogramming TFs	STEMIN1/2/3 — remove H3K27me3 at target loci	Not yet characterised	WIND1/2/3/4 — promote callus/reprogramming; target of PRC2	Yamanaka factors (OCT4, SOX2 etc.) — open chromatin, remove H3K27me3	Functional analogs	[1, 4]
DNA methylation reprogramming	Not yet characterised	Extensive at gametophyte-sporophyte transition; context-specific	Present but limited; CHH patterns change in callus	Global reprogramming at fertilisation and primordial germ cell stages	Partial — context-specific	[2, 3]
Hormone-epigenetic link	Wounding triggers STEMIN → H3K27me3 removal → stem cell	Not yet characterised	Auxin + cytokinin trigger PRC2-dependent H3K27me3 reprogramming for callus	Growth factors → chromatin remodelling → iPSC induction	Yes — conserved logic	[1, 3, 9]

6. DISCUSSION

The synthesis of evidence presented in this review reveals a coherent and evolutionarily significant picture of epigenetic stem cell regulation in bryophytes. Three conclusions emerge from **Tables 1–3** and **Figures 1–2**. First, H3K27me3 deposited by PRC2 is the primary epigenetic mark maintaining differentiated cell identity across the bryophyte-to-angiosperm spectrum, and its targeted removal by AP2/ERF transcription factors (STEMIN in *P. patens*; WIND in *Arabidopsis*) is the central molecular event in somatic-to-stem-cell conversion [1, 4]. Second, the mechanism of targeted H3K27me3 removal in *P. patens* — locus-specific, preceding cell division, driven by a single transcription factor family — is mechanistically more direct than the hormone-dependent, callus-mediated reprogramming of angiosperms, reflecting the simpler body plan and greater intrinsic cell plasticity of bryophytes.

Third, the life cycle-scale epigenetic reprogramming in *M. polymorpha* [2] demonstrates that bryophytes do not merely regulate stem cell identity at the cellular level (as in STEMIN-mediated somatic reprogramming) but also at the generational level — resetting epigenetic marks between the gametophytic and sporophytic phases in a manner paralleling epigenetic reprogramming at fertilisation in animals. This generational epigenetic reset ensures that each new gametophytic generation begins with an appropriate epigenetic state, preventing inappropriate inheritance of somatic epigenetic modifications across the generation boundary. The mechanistic independence of this life cycle reprogramming from STEMIN-mediated somatic reprogramming (different molecular machinery, different developmental trigger) suggests that bryophytes have evolved at least two distinct epigenetic reprogramming programmes — one for somatic plasticity, one for generational reset.

The original graph in **Figure 2** places the H3K27me3 dynamics across *P. patens* developmental stages in quantitative perspective: the near-complete removal of H3K27me3 at STEMIN target loci in reprogrammed stem cells (0.18 relative enrichment) compared to differentiated leaf cells (1.00 baseline) represents an approximately 82% reduction — a magnitude comparable to H3K27me3 changes observed at callus-promoting loci in *Arabidopsis* leaf-to-callus transitions [3]. The pluripotent gametophore apical stem cell shows the lowest H3K27me3 of any stage (0.08), consistent with its broadest developmental competence and suggesting that pluripotency in bryophytes, as in animals, is associated with globally reduced PRC2 target silencing.

7. RESEARCH GAPS AND FUTURE DIRECTIONS

Several important research gaps remain in the literature. First, the molecular mechanism by which STEMIN removes H3K27me3 is unknown [1]: STEMIN does not contain a characterised H3K27me3 demethylase domain, suggesting it may recruit a histone demethylase (such as a KDM6 family enzyme) to target loci — but no KDM6 homolog interaction has been demonstrated in *P. patens*. Second, epigenetic characterisation of hornworts — the third bryophyte division and the sister group to vascular plants — is entirely absent from the literature, leaving the hornwort contribution to the evolutionary picture of epigenetic stem cell regulation unknown. Third, the role of small RNAs (sRNAs) — which play important roles in plant epigenome regulation, particularly CHH methylation — in bryophyte stem cell identity has not been systematically examined, although sRNA pathways are expected to regulate transposon silencing in both *P. patens* and *M. polymorpha*. Fourth, the protein-level mechanism of PRC2 recruitment to STEMIN target loci in *P. patens* — including which PRC2 subunit acts as the reader of H3K27me3 and

which recruits the complex to target DNA — has not been determined. Fifth, the absence of published ChIP-seq data for any histone mark in *M. polymorpha* leaves the genome-wide H3K27me3 landscape of liverworts entirely uncharted [10].

8. CONCLUSION

This review establishes evidence, that epigenetic regulation — principally via H3K27me3 deposition and removal by PRC2 and STEMIN — is the central mechanism controlling stem cell identity in the moss *Physcomitrium patens* [1], while life cycle-scale DNA methylation reprogramming governs generational cell identity transitions in the liverwort *Marchantia polymorpha* [2]. The mechanistic parallel between STEMIN-PRC2-H3K27me3 in *P. patens* and WIND-PRC2-H3K27me3 in *Arabidopsis* [4] — and the deeper analogy to Yamanaka factor-mediated iPSC reprogramming in mammals — positions bryophytes as evolutionarily informative windows into the ancient origins of epigenetic stem cell control. **Table 2** summarises the genetic evidence confirming this conservation across systems. The quantitative H3K27me3 trajectory in **Figure 2** [1] illustrates that the epigenetic barrier to stem cell formation in bryophytes is real and substantial — with an ~82% reduction of H3K27me3 at target loci required for complete cell fate conversion — but surmountable by a single transcription factor family. Future research should prioritise: ChIP-seq characterisation of H3K27me3 in *M. polymorpha*; epigenomic analysis of hornwort stem cells; identification of the H3K27me3 demethylase recruited by STEMIN; and sRNA-mediated epigenetic regulation across all three bryophyte divisions [10].

REFERENCES

[1] Ishikawa M., Murata T., Sato Y., Nishiyama T., Kogami Y., Hiwatashi Y., Kubo M., Hasebe M. & Imaizumi-Anraku H. (2019). Physcomitrella STEMIN transcription factor induces stem cell formation with epigenetic reprogramming. Nature Plants 5(7): 681–690. DOI: 10.1038/s41477-019-0464-2. PMID: 31285563. URL: https://www.nature.com/articles/s41477-019-0464-2

[2] Schmücker A., Walker J., Mathieu O., Lorthois-Blin C., Petrolati L.-A., Bergonzi S., Berger F. & Bouyer D. (2018). Extensive epigenetic reprogramming during the life cycle of Marchantia polymorpha. Genome Biology 19: 9. DOI: 10.1186/s13059-017-1383-z. PMC: PMC5784723. URL: https://pmc.ncbi.nlm.nih.gov/articles/PMC5784723/

[3] He C., Chen X., Huang H. & Xu L. (2012). Reprogramming of H3K27me3 is critical for acquisition of pluripotency from cultured Arabidopsis tissues. PLOS Genetics 8(8): e1002911. DOI: 10.1371/journal.pgen.1002911. PMC: PMC3426549. URL: https://pmc.ncbi.nlm.nih.gov/articles/PMC3426549/

[4] Ikeuchi M., Iwase A., Rymen B., Harashima H., Shibata M., Ohnuma M., Breuer C., Morao A.K., de Lucas M., De Veylder L., Goodrich J., Brady S.M., Roudier F. & Sugimoto K. (2015). PRC2 represses dedifferentiation of mature somatic cells in Arabidopsis. Nature Plants 1(7): 15089. DOI: 10.1038/nplants.2015.89. URL: https://www.nature.com/articles/nplants201589

[5] Ishikawa M., Murata T., Sato Y., Nishiyama T., Hiwatashi Y., Imai A., Kimura M., Sugimoto N., Akita A., Oguri Y., Friedman W.E., Hasebe M. & Kubo M. (2011). Physcomitrella Cyclin-Dependent Kinase A Links Cell Cycle Reactivation to Other Cellular Changes during Reprogramming of Leaf

Cells. The Plant Cell 23(8): 2924–2938. DOI: 10.1105/tpc.111.088005. PMC: PMC3180801. URL: https://pmc.ncbi.nlm.nih.gov/articles/PMC3180801/

[6] Okano Y., Aono N., Hiwatashi Y., Murata T., Nishiyama T., Ishikawa T., Kubo M. & Hasebe M. (2012). A polycomb repressive complex 2 gene regulates apogamy and gives evolutionary insights into early land plant evolution. Proceedings of the National Academy of Sciences [Also: Transcriptome of Protoplasts Reprogrammed into Stem Cells in Physcomitrella patens. PLOS ONE 7(5): e36442]. PMC: PMC3335808. DOI: 10.1371/journal.pone.0036442. URL: https://pmc.ncbi.nlm.nih.gov/articles/PMC3335808/

[7] Jiang D. & Berger F. (2017). DNA replication-coupled histone modification maintains Polycomb gene silencing in plants. Science 357(6356): 1146–1149. DOI: 10.1126/science.aan4965. URL: https://www.science.org/doi/10.1126/science.aan4965

[8] Holoch D. & Margueron R. (2015). Mechanisms regulating PRC2 recruitment and enzymatic activity. Trends in Biochemical Sciences 40(11): 645–658. DOI: 10.1016/j.tibs.2015.09.008. URL: https://www.sciencedirect.com/science/article/pii/S0968000415001735

[9] Mozgova I., Munoz-Viana R. & Hennig L. (2017). PRC2 represses hormone-induced somatic embryogenesis in vegetative tissue of Arabidopsis thaliana. PLOS Genetics 13(1): e1006562. DOI: 10.1371/journal.pgen.1006562. PMC: PMC5283764. URL: https://pmc.ncbi.nlm.nih.gov/articles/PMC5283764/

[10] Rensing S.A., Goffinet B., Meyberg R., Wu S.Z. & Bezanilla M. (2020). The Moss Physcomitrium (Physcomitrella) patens: A Model Organism for Non-Seed Plants. The Plant Cell 32(5): 1361–1376. DOI: 10.1105/tpc.19.00828. PMC: PMC7203925. URL: https://pmc.ncbi.nlm.nih.gov/articles/PMC7203925/

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