Deposition and eviction of histone variants define functional chromatin states in plants

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Short title: Histone variant dynamics in plants

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Abbreviations:
HFD, histone fold domain; **PTMs**, post-translational modifications; **KD**, knock-down; **TEs**, transposable elements; **CC**, central cell; **EC**, egg cell

Abstract:
The organization of DNA with histone proteins into chromatin is fundamental for the regulation of gene expression. Incorporation of different histone variants into the nucleosome together with post-translational modifications of these histone variants allows modulating chromatin accessibility and contributes to the establishment of functional chromatin states either permissive or repressive for transcription. This review highlights emerging mechanisms required to deposit or evict histone variants in a timely and locus-specific manner. This review further discusses how assembly of specific histone variants permits to reinforce transmission of chromatin states during replication, to maintain heterochromatin organization and stability and to reprogram existing epigenetic information.

Histone variants: versatile building blocks of the nucleosome

The organization of the eukaryotic genome into chromatin is critical for gene expression control as it allows compartmentalizing chromosomes into functional domains more or less accessible to the transcription machinery. The basic subunits of chromatin, the nucleosomes, are aligned as 'pearls on the string' along the DNA thread. Each nucleosome structures ~ 146bp of DNA, which wrap in two left-handed super helical turns around an octamer of histone proteins comprising two molecules each of histone H3, H4, H2A and H2B [1]. Linker histones H1 bind to nucleosomal and linker DNA thereby modulating folding of the chromatin fiber facilitating chromatin compaction [2]. Histones are some of the evolutionary most highly conserved proteins [3,4]. As an example, even between such distantly related species as mouse and Arabidopsis, H4 proteins differ by only two amino acids. Histones are abundantly post-translationally modified; both in the histone fold domain (HFD) and the tail region. These covalent post-translational modifications (PTMs) recruit or repel specific effector proteins that influence all DNA-based processes including transcription. Except for histone H4, most eukaryotic genomes encode different types of histone variants that may differ by only a few amino acids or larger protein domains (Figure 1) [3,5]. Multiple combinations of these histone variants can be assembled in a given nucleosome, thereby directly modifying its biochemical properties [6,7] as well as downstream processes by influencing histone PTMs [8,9].

Histone variants - precious cargoes

Depositing histone H3 variants and H4

Genome wide analyses of histone variants revealed specific enrichment patterns along the genome [10–13] and their contribution to the definition of functional chromatin states [14] permissive or repressive for transcription (Figure 2). Given this important role in chromatin control, deposition and eviction of histone variants are tightly coordinated through a network of histone chaperones and chromatin remodeling complexes. Nucleosome assembly starts with the deposition of H3-H4 via specific assembly pathways that employ dedicated H3 variants. To meet the requirement for high histone supply to reconstruct the chromatin fiber after DNA replication during S-phase, the histone chaperone CHROMATIN ASSEMBLY COMPLEX 1 (CAF-1) deposits H3.1-H4 tetramers onto newly replicated DNA [15,16]. Outside S-phase, deposition of H3.3-H4 at regions of high nucleosome turnover or upon chromatin reorganization during DNA-repair and transcription requires HISTONE REGULATOR A (HIRA) [17,18] and the Alpha Thalassemia-mental Retardation X-linked (ATRX) ortholog [19]. The importance of these pathways becomes evident upon their

perturbation that leads to reduced nucleosome occupancy or altered cellular H3.1/H3.3 ratios [16,19–21]. H3.1 and H3.3 differing by only 4-5 amino acids are believed to have emerged repeatedly during evolution [22]. Interestingly, both in plants and animals, two amino acid differences are situated within the second alpha helix of the HFD (Figure 1), determining deposition mode [23]. Given that the distinct assembly pathways predate the appearance of distinct replicative and replacement variants such as in yeast, it is tempting to speculate that these variants have evolved to facilitate histone supply via the evolutionary conserved histone deposition pathways and/or to allow the controlled setting of histone PTMs (discussed below). In contrast to H3.1 and H3.3, the centromeric histone cenH3 evolves rapidly [24,25] together with dedicated assembly proteins. How cenH3 is deposited at centromeric repeats in plants remains still to be determined but NUCLEAR AUTOANTIGENIC SPERM PROTEIN (NASP) has recently been identified to escort cenH3 in addition to H3.1 and H3.3 [26].

Histone exchange via chromatin remodeling to incorporate H2A variants

While H3-H4 deposition is ensured by dedicated chaperones, it is much less defined how the different H2A or H2B variants are incorporated into chromatin. In plants, the histone chaperones NUCLEOSOME ASSEMBLY PROTEINs 1 (NAP1;1-1;4) and NAP1 RELATED PROTEINs (NRP1 and NRP2) [27] are candidates for H2A-H2B transport and deposition, however whether they can distinguish between H2A variants and coordinate a variant-specific assembly is not known. Instead, the differential distribution of H2A variants in the genome may rather rely on specific chromatin remodeling factors. For example, in Arabidopsis as in yeast and metazoans, deposition of the highly conserved H2A.Z histone variants requires the SWI/SNF-RELATED 1 (SWR1) chromatin-remodeling complex [28–30], which exchanges nucleosomal H2A-H2B with H2A.Z-H2B. SWR1 can target specific genes through recruitment by the DNA-binding protein SWC4 that recognizes AT-rich DNA elements in their promoters [31], but other complexes such as the nucleosomal acetyltransferase of H4 (NuA4) complex are also involved in H2A.Z deposition [32]. Given that H2A.Z-containing nucleosomes are evicted from thermo-responsive genes at higher temperatures [33], several studies have investigated how H2A.Z can be removed from chromatin. At PHYTOCHROME-INTERACTING FACTOR 4 (PIF4) target genes, INO80, another ATP-dependent chromatin remodeler, removes H2A.Z, thereby cooperating with transcriptional elongation factors SPT4 and SPT5 (homologs of yeast Suppressor of Ty 4/5) [34]* suggesting that H2A.Z removal occurs during transcription. NRP1 and NRP2 also counteract SWR1-mediated H2A.Z deposition; their loss leading to higher H2A.Z levels at genes, but also within heterochromatin [35] normally H2A.Z-depleted [11]. H2A.Z deposition and removal are hence highly dynamic, relying on multiple deposition and eviction modes by

chromatin remodeling machineries that can be recruited to specific set of genes via specific DNA binding proteins, thereby allowing to dynamically modulating H2A.Z enrichment to fine-tune transcription.

In analogy to H2A.Z, incorporation of the heterochromatin-enriched H2A.W variants [11], also requires chromatin-remodeling activity [36]. H2A.W interacts directly with the chromatin-remodeling factor DECREASE IN DNA METHYLATION (DDM1). DDM1 is required to maintain high levels of H2A.W in particular at transposons and for their silencing [36]*. How exactly DDM1 is recruited to specific sites and whether H2A.W deposition requires passage through S-phase remains to be determined. Deficient H3.1 incorporation during DNA-replication in young seedlings of *fasciata* mutants (lacking a functional CAF-1 complex), also negatively affects H2A.W enrichment [37], indicating that H2A.W deposition could be at least partly coupled to DNA-synthesis dependent nucleosome assembly (Figure 3). This is in agreement with a role for the murine DDM1 homologue Lsh at newly assembled chromatin where it facilitates access to the DNA methylation maintenance machinery [38]. Since H2A.W is not completely absent in *ddm1* mutants, alternative incorporation pathways may exist possibly involving other chromatin remodeling complexes. Once incorporated, one can speculate that H3.1-H2A.W nucleosomes, maybe in combination with specific H2B variants, are favored over other variant combinations allowing H2A.W to be selectively retained.

Taken together, assembly and eviction of histone variants requires regulation at multiple levels involving availability, by tight transcriptional control, assembly mode by histone chaperones, exchange by chromatin remodeling factors and potentially selective retention of particular stable nucleosomes with certain histone variant combinations.

Specific roles for histone variants in somatic cells

While histone variants exist in all eukaryotic organisms, it remains a challenge to identify their specialization and complementarity. Least questioned is the importance of cenH3 as its loss causes lethality [24] and aberrant cenH3 proteins are preferentially removed from chromatin in egg cells and zygotes when backcrossed to wild type [25], underlining the critical function of cenH3 in centromere organization and kinetochore establishment. Instead, absolute requirement for H3.1 or H3.3 may depend on organisms and developmental stages. In Drosophila, H3.1 can replace H3.3 in somatic tissues and restore transcriptional defects of *h3.3* mutants [39], while in Arabidopsis, complete loss of H3.3 causes lethality [40]. Despite the specific enrichment of H3.3 in the 3' end of transcriptionally active genes [10], reduction of H3.3 levels in *H3.3 knockdown (KD)* lines only modestly disturbs gene expression without obvious correlation with the initial H3.3 enrichment [39]. Instead, reduction in H3.3 reduces

gene body methylation and increases H2A.Z and linker histone H1 occupancy over gene bodies. Notwithstanding the modest changes in gene expression, diverse developmental defects are observed in *H3.3 KD* plants including late flowering due to a role of H3.3 in regulating expression of *FLOWERING LOCUS C (FLC)* [41]*. Indeed, *FLC* expression and H3.3 deposition is enhanced in the presence of functional FRIGIDA (FRI), a component of the transcription activator complex FRI-C, which directly recruits the H3.3 chaperone HIRA to *FLC* [41]*. The presence of H3.3 further promotes FRI-induced loop formation of the *FLC* gene. H3.3 enrichment at 5' and 3' ends has also been observed at silent genes [42], suggesting a more general role for H3.3 or its deposition machinery in favoring small chromatin loops at gene bodies and therefore in the 3D organization of the genome.

H3.1 is deposited at the replication fork and *H3.1 KD* leads to developmental defects as upon perturbation of the H3.1 assembly machinery [16]. Phenotypes of *H3.1 KD* plants cannot be rescued by expressing H3.3 coding sequence under *H3.1* promoters, confirming that the few amino acids differing between the two variants are of functional importance [16]. Indeed at least two histone methyltransferases, ARABIDOPSIS TRITHORAX-RELATED PROTEIN 5 (ATXR5) and ATXR6, exclusively mono-methylate lysine 27 of the H3.1 variant [43]. Recruited by the DNA clamp PROLIFERATING CELL NUCLEAR ANTIGEN (PCNA), ATXR proteins propagate H3K27me1 at the replication fork (Figure 3). Within genes, they thereby contribute to pre-mark new H3.1 variants during their deposition for further methylation to the trimethylated state by POLYCOMB REPRESSIVE COMPLEX 2 [16] (Figure 3). Therefore, H3.1 deposition at the replication fork is tightly linked to K27 methylation maintenance, similar to the mechanism suggested for H3K9me3 maintenance in mammalian cells [8].

Arabidopsis TONSOKU (TSK), the plant orthologue of mammalian TONSOKU-LIKE (TONSL), also specifically interacts with the H3.1 tail [44]*. TSK is recruited via newly deposited H3.1 at the replication fork, where it is thought to initiate similar to TONSL the repair in case of double strand breaks at replication forks via homologous recombination. TSK binding to the H3.1 tail is reduced by methylation of several lysines in particular mono-methylation of K27 set by ATXR5/6 [44]*. This suggests that TSK is progressively released from newly incorporated H3.1 once the H3.1 tail becomes methylated during chromatin maturation restricting TSK-mediated homologous recombination to the replication fork (Figure 3).

Given the role for H2A.Z in transcriptional regulation, *h2a.z* mutants with strongly reduced *H2A.Z* transcript levels [13] or mutants in the H2A.Z deposition machinery [28,29,33] show pleiotropic developmental defects including altered stature, reduced fertility and early flowering. In contrast to *h2a.z* mutants, complete loss-of-function *h2a.w* mutant plants are indistinguishable from wild type plants [45] and unexpectedly, most transposable elements (TEs) remain silenced, suggesting the existence of multiple pathways that conjointly keep

TEs under control. This recalls the phenotype of *h1.1 h1.2 h1.3* (*3h1*) triple mutants, in which, despite strong H1 presence in wild type heterochromatin, and reduced nucleosomal occupancy in *3h1* mutants, the vast majority of TEs are not reactivated [46]*. Nevertheless, both *3h1* [46]* and *h2a.w* [11] reduce the condensation of repetitive sequences into chromocenters, a phenotype further enhanced in plants lacking both H2A.W and H1 [45]. Closer inspection further revealed that H2A.W prevents overaccumulation of H1 and that both H2A.W and H1 cooperatively control accessibility within heterochromatin [45].

In contrast to H2A.W, which is restricted to heterochromatin, H1 is also present in euchromatin, where it controls nucleosome density and restricts nucleosome mobility [46]*. H1 is further involved in H3K27me3 homeostasis [46,47], as H1 favors H3K27me3 enrichment at Polycomb-regulated genes and prevents over-accumulation of H3K27me3 at interstitial telomeric repeats [47].

Specific roles for histone variants in H3K27me3 reprogramming during reproduction

H3K27me3 plays a central role in gene expression control during development [48–50] and a common function of several histone variants seems to be the regulation of this repressive mark not only by ensuring maintenance at the replication fork [16,43] or preventing redistribution along the genome [46,47] but also by facilitating rapid H3K27me3 reprogramming [51,52]. An example for the latter is incorporation of the atypical histone variant H3.15 that lacks lysine 27 (Figure 1) and is induced upon wounding in roots. H3.15 deposition leads to H3K27me3 reduction at certain promoters facilitating expression of genes activated upon wound-induced callus formation [51].

H3K27me3 also needs to be reset during generations to prevent formation of new transmittable epialleles [50] and part of this resetting occurs in the male gametophyte. Pollen sperm cell nuclei show low H3K27me3 levels [52]* associated with increased accessibility likely to facilitate expression of genes specific for sperm cells or required during early embryonic development [49]. Sperm cells express a specific variant, H3.10 (Figure 2) that contributes to H3K27me3 resetting as sequence variation downstream of lysine 27 in H3.10 (Figure 1) makes it a poor substrate for H3K27me3 methyltransferases [52]*. H3.10 does not act alone, but is one element in the multilayer mechanism of H3K27me3 reprogramming that involves also regulation of histone methyltransferase and demethylase expression. Interestingly, another variant is also enriched in pollen sperm nuclei, namely H2B.8 [12,53,54]. Whether these two sperm-specific variants assemble in the same nucleosomes and how they impact nucleosome stability remains to be determined.

Perspectives

Several new studies have complemented our view on the function of histone variants in plants. While some variants have a more constitutive role in gene expression, ensuring histone supply and turnover, others are specifically expressed in only a few cell types or tissues. However, the function of atypical H3 variants, other than H3.10, remains yet to be elucidated, including H3.6, H3.11 and H3.14, which differ by several amino acids in their N-terminal tail with the potential to affect deposition of histone modifications. *H3.14* is specifically expressed in the vegetative nucleus of the pollen but also in the female central cell [55] and both *H3.6* and *H3.14* are upregulated upon depletion of the DNA synthesis-dependent histone deposition pathway [37]. This could be a compensatory mechanism to ensure nucleosome occupancy or their assembly at specific loci may directly affect gene expression. Even though most of the atypical H3 variants are expressed in specific cells, it will be interesting to investigate whether they are incorporated genome-wide like H3.10 or whether they could be deposited at specific sets of genes, as shown for the deposition of H3.3 at the *FLC* locus by recruitment of HIRA via FRI [41]*. To understand where and how histone variants will exert their function, it is important to consider the different modes and machineries of deposition and exchange. Furthermore, post-translational modifications of histone variants may modulate the effect of a histone variant on nucleosome stability or further modify the activity and read-out of the histone variant. Finally, selective incorporation of the 11 different H2B variants [12] into the nucleosome adds another level of complexity as they likely impact nucleosome properties or are substrates for specific reader proteins. The study of multiple closely related histone variants with potential compensatory roles has been tedious, requiring combinations of mutants or RNAi lines. The CRISPR/Cas9 technology is now allowing to create multiple variant-specific mutations as recently done for histone H4 and H3.1 [44,56]. This will certainly help to advance our knowledge on histone variants both in model and crop plants with more complex, transposon-rich genomes.

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I apologize to all colleagues whose work I was not able to discuss because of space constraints.

Credit Authors Statement:

A. Probst: Writing – original draft; Writing – review & editing.

Figures and legends:

Figure 1:

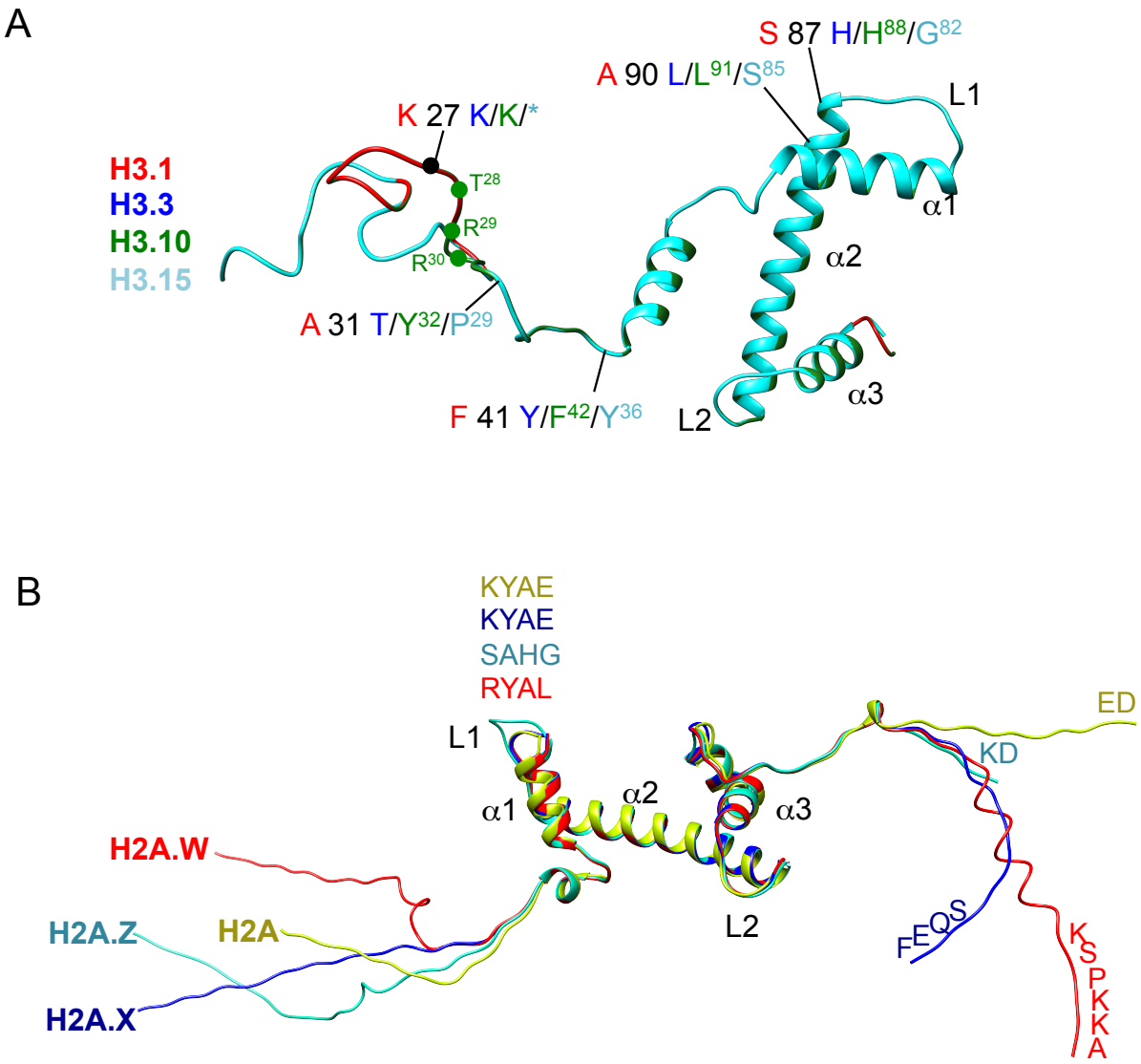

Figure 1: Representation of structural differences between selected H3 and H2A variants

(A) Models of Arabidopsis histone H3.1 (red), H3.3 (blue), H3.10 (green) and H3.15 (cyan). The position of the four amino acids at position 31, 41, 87 and 90 that distinguish H3.1 from H3.3 are indicated together with the amino acid changes observed in H3.10 and H3.15 in the respective colors. Positions 31 and 41 are situated in the histone tail, and positions 87 and 90 in the HFD, which consists of three alpha helices (α1, α2 and α3) and two loops (L1, L2). Lack of Lysine 27 (K27, black circle) in H3.15 is indicated by an asterisk. Green circles illustrate the three amino acid differences between H3.1/3 and H3.10 (TRR) downstream of K27, which negatively affect K27 histone lysine methyltransferase activity [52].

(B) Models of Arabidopsis histone H2A (H2A.2, brown), H2A.Z (H2A.Z.8, cyan), H2A.X (H2A.X.3, blue) and H2A.W (H2A.W.7, red). The four types of H2A histone variants differ in the length of their N- and C-terminal tails and in the loop 1 (L1) region connecting the first and second alpha helix of the HFD and involved in H2A self-interaction within the nucleosome. Loop1 sequences in the 4 variant types are indicated in the respective color. H2A.W and H2A.X are characterized by specific KSPKKA and SQEF motifs in the C-terminal tails, respectively.

3D models were obtained with PHYRE2 [57] for H3 and AlphaFold [58] for H2A and were aligned and colored with Chimera.

Figure 2

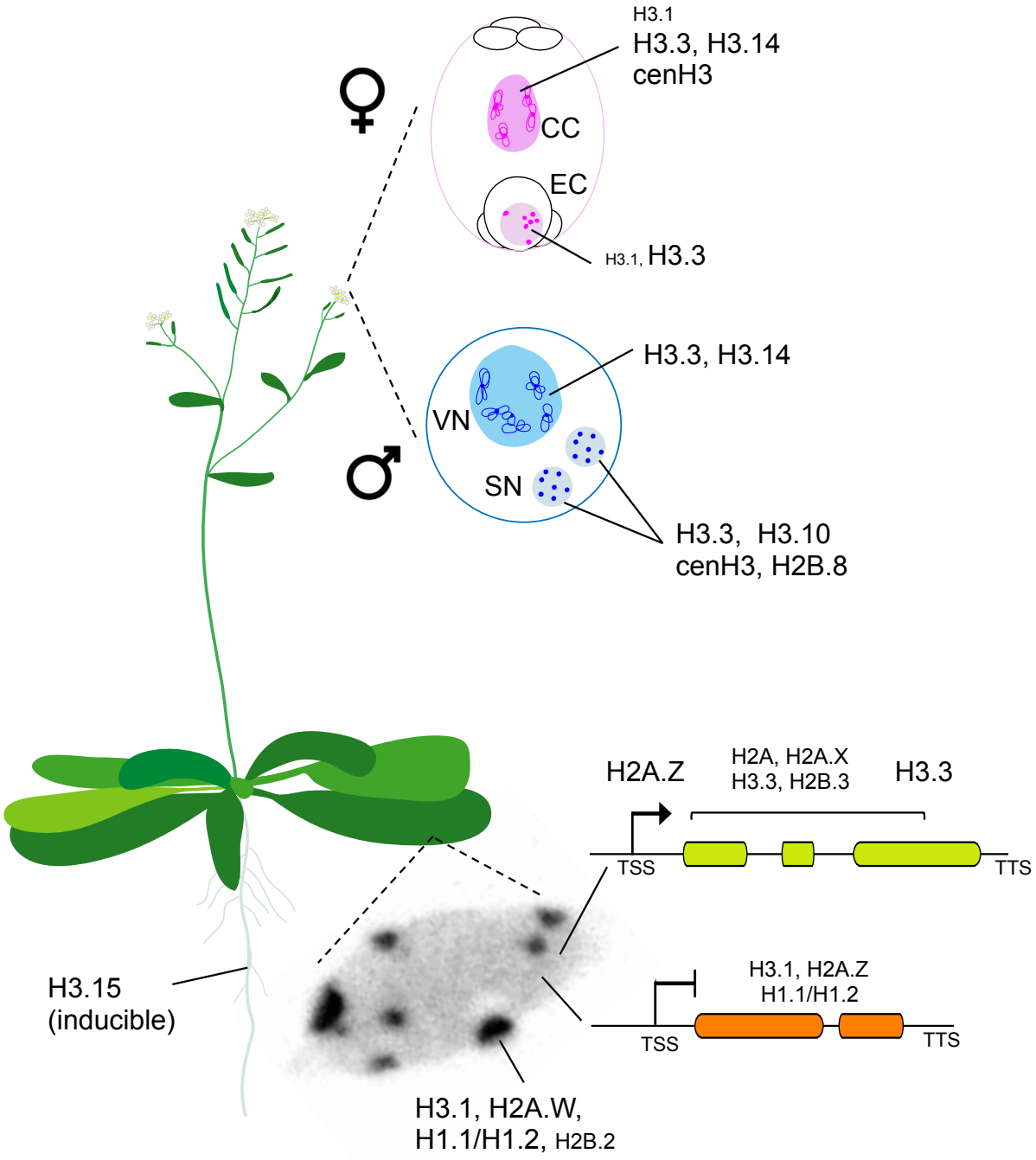
H3.1
H3.3, H3.14
cenH3
CC
EC
H3.1, H3.3
H3.3, H3.14
VN
SN
H3.3, H3.10
cenH3, H2B.8
H2A.Z
H2A, H2A.X
H3.3, H2B.3
H3.3
TSS
TTS
H3.1, H2A.Z
H1.1/H1.2
TSS
TTS
H3.15
(inducible)
H3.1, H2A.W,
H1.1/H1.2, H2B.2

Figure 2: Differential enrichment of histone variants along the genome during plant development

The Arabidopsis histone variant repertoire includes several H3 variants (centromere-specific cenH3, H3.1, H3.3, and atypical H3 variants), 13 H2A variants (four of the H2A, two of the H2A.X, three of the H2A.W and three of the H2A.Z type not including the potential pseudogene *HTA4*), eleven H2B variants and three H1 variants [5]. *In somatic nuclei*, exemplified by the DAPI-stained mesophyll leaf nucleus, histone variants are differentially enriched along the genome, contributing to the establishment of different functional chromatin states permissive or repressive for transcription. The highly condensed heterochromatic regions (strongly DAPI-stained, black) are enriched in H3.1 [10], H1.1 / H1.2 [59], H2A.W [11], which contributes to chromatin condensation through chromatin fiber-fiber interactions, and show some enrichment for the H2B.2 variant [12]. H1 and H2A.W jointly contribute to chromatin accessibility in these regions [45]. In euchromatin (faintly stained by DAPI, grey), the transcriptionally active state of genes is reflected by enrichment in the histone variant H3.3 in particular at the 3' end, while H3.1 marks silent genes [10]. Enrichment of H2A, H2A.X [11] and H2B.3 [12] also correlates positively with gene expression level and H2B.3 is enriched in H3.3 containing nucleosomes [12]. More complex are the occupancy patterns for H2A.Z, which is enriched at transcriptional start sites for highly transcribed genes [60], while it localizes along gene bodies at different sets of genes for example thermo-responsive genes [33] where H2A.Z especially in its mono-ubiquitinated form contributes to transcriptional repression [61].

During reproductive development, specific histone variants are expressed in certain cell types only, where they contribute to reprogramming of histone modifications. The mature pollen contains a large vegetative cell and two sperm cells, neither expressing H3.1 [55]. Sperm cell nuclei (SN), in which heterochromatic regions form condensed structures, express cenH3 [55], H3.10 [52] and H2B.8 [12,54], while vegetative nuclei (VN), which show decondensed heterochromatin, express the H3.14 variant [55]. H3.10 enrichment in sperm cells helps to reprogram H3K27me3 [52], while H2B.8 is thought to contribute to chromatin compaction [54]. Central and egg cells (EC) in the female gametophyte also show differential enrichment in histone variants with H3.14 enriched in the central cell (CC). The role of H3.14 in the VN and CC is still unknown, but correlates with decondensed heterochromatin. After fertilization, the histone variant repertoire of EC, CC and SN is rapidly modulated [55]. Finally, upon wounding of roots, the H3.15 variant is specifically induced, facilitating H3K27me3 resetting at wound-responsive genes [51].

TSS, Transcriptional Start Site; TTS, Transcriptional Termination Site.

Figure 3:

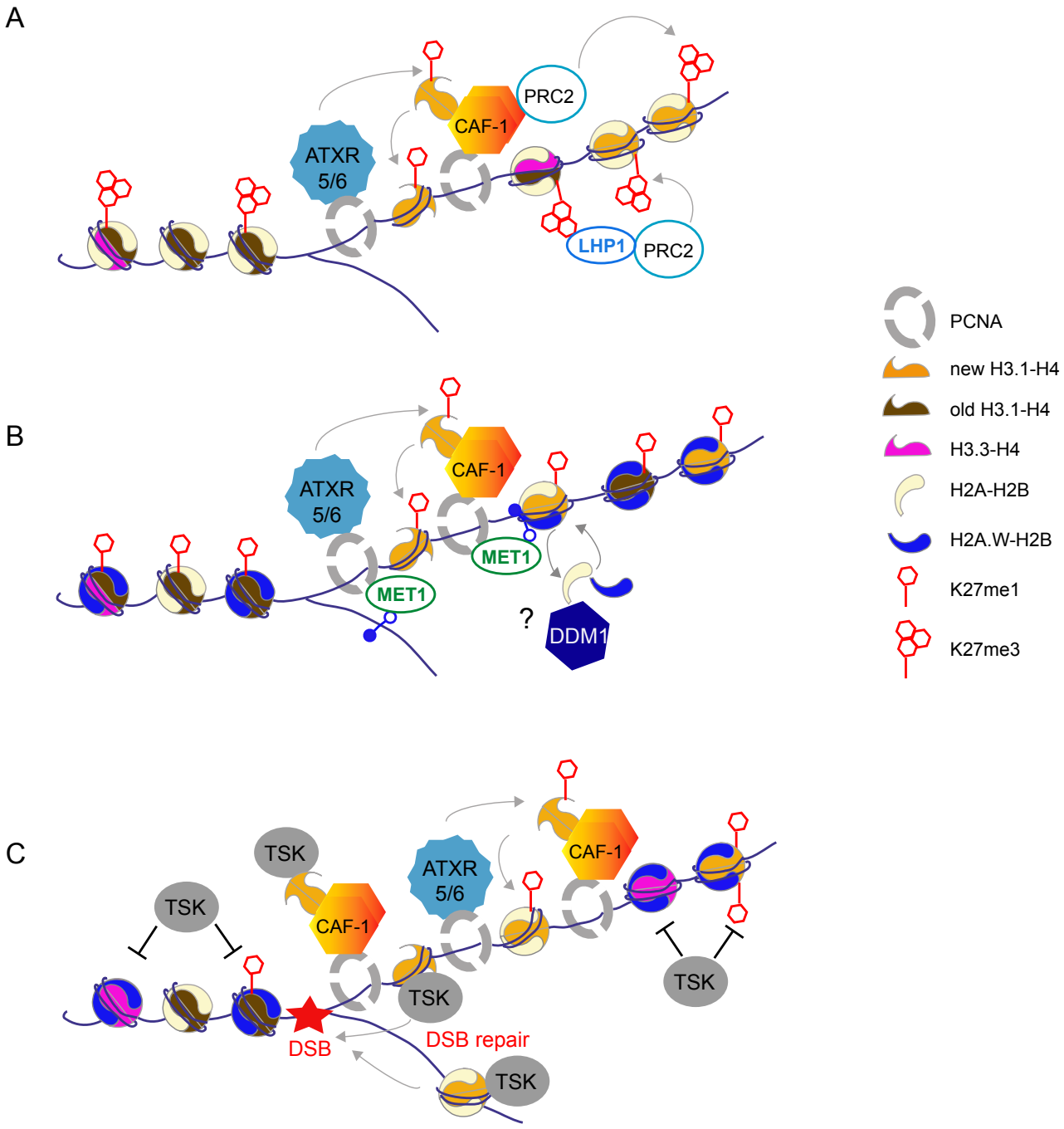
A
ATXR 5/6
CAF-1
PRC2
LHP1
PRC2
B
ATXR 5/6
CAF-1
MET1
MET1
?
DDM1
C
TSK
TSK
CAF-1
ATXR 5/6
CAF-1
TSK
TSK
DSB
DSB repair
TSK
PCNA
new H3.1-H4
old H3.1-H4
H3.3-H4
H2A-H2B
H2A.W-H2B
K27me1
K27me3

Figure 3: Models for the role of the H3.1 variant in maintenance of epigenetic marks and genome stability at the replication fork

A. Maintenance of H3K27me3 at the replication fork. Genes regulated by Polycomb are marked by H3K27me3 enrichment. During replication, parental histone tetramers (here containing H3.1 (brown) and H3.3 (pink)) are recycled together with their post-translational modifications on the newly replicated DNA strand. To restore nucleosomal density, CAF-1 complexes recruited via PCNA deposit H3.1-H4 tetramers on the newly replicated DNA strand. To reestablish H3K27me3, PRC2 complexes are recruited via interaction with CAF-1 subunits or via the H3K27me3 reader LIKE HETEROCHROMATIN PROTEIN1 (LHP1) that binds to parental H3K27me3 [16]. PRC2 action can be facilitated by pre-marking new H3.1 (potentially in complex with CAF-1) before or during deposition with K27me1 by ATXR5 and 6 also recruited via PCNA.

B. Maintenance of heterochromatin characteristics at the replication fork. During replication of heterochromatin that is enriched in DNA methylation, H2A.W (blue) and H3K27me1, these different characteristics could be propagated directly at the replication fork or shortly after within newly assembled chromatin. Parental H3-H4 tetramers are recycled and similar to H3K27me3 enriched genes, ATXR5 and 6 propagate the H3K27me1 mark by methylating newly synthesized H3.1 (orange) [16]. METHYLTRANSFERASE 1 (MET1) possibly recruited via PCNA methylates cytosines in hemimethylated free DNA, but potentially also within newly assembled chromatin where it requires the chromatin remodeling factor DDM1 to gain access to DNA [38]. In a process coupled or uncoupled from DNA replication, DDM1 then exchanges H2A to H2A.W [36]*.

C. Restricting TSK-mediated double-strand break repair to the replication fork. TONSOKU (TSK) binds specifically to the H3.1 histone tail in its unmethylated form [44]* and is therefore absent from mature chromatin containing either H3.3 (pink) or methylated, parental H3.1 histone tails (brown). During DNA replication TSK binds to newly deposited H3.1 (orange) either in complex with CAF-1 or after its deposition onto newly replicated DNA. In case of a DNA double strand breaks (DSBs) encountered by the replication fork TSK can initiate homologous recombination mediated DNA repair. During post-replicative maturation of chromatin through methylation of K27me1 by ATXR5 and 6 or other lysines of the H3.1 tail, TSK looses affinity for the H3.1 tail [44]* thereby its action is restricted to the replication fork.

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Highlighted References

34, Xue, Mol Plant 2021:*
The authors investigated the mode of H2A.Z removal from chromatin and identified a role for the chromatin remodeling complex INO80 in H2A.Z eviction. Recruited by PIF4, INO80 removes H2A.Z from thermoresponsive genes thereby cooperating with transcription elongation factors SPT4 and SPT5 suggesting a tight correlation between transcription and H2A.Z removal.

36, Osakabe, Nat Cell Biology, 2021:*
This article investigated the deposition mode for the H2A.W histone variants highly enriched in Arabidopsis heterochromatin. The authors show that H2A.W interacts with the chromatin-remodeling factor DDM1, which is required for H2A.W deposition at potential mobile TE coding regions and for their silencing.

41 Zhao, Plant Physiol, 2021:*
This study revealed a requirement for H3.3 in *FLC* expression. The authors show that the *FLC* activator FRIGIDA directly interacts with the histone chaperone HIRA and thereby promotes H3.3 deposition, which in turn facilitates loop formation at the *FLC* locus and stimulates deposition of active histone modifications.

44, Davarinejad, Science, 2022:*
This study reveals how initiation of double strand repair by TONSOKU (TSK) is restricted to replication forks in plants relying on specific H3.1 variants and deposition modes. TSK preferentially binds to non-modified H3.1 and H3.3 histone tails allowing its recruitment to the replication fork. During post-replicative chromatin maturation, H3.1 tails become methylated, which leads to release of TSK from chromatin.

46, Rutowicz, Genome Biology, 2019:*
This paper reports a careful analysis of the developmental defects and structural changes in chromatin organization caused by lack of linker histone H1. Within heterochromatin, loss of H1 leads to decondensation of repetitive sequences, without releasing silencing of the majority of transposable elements. Within euchromatin, H1 reduces nucleosome mobility and controls the arrangement of compact chromatin nanodomains.

52, Borg, Nature Cell Biology, 2020:*
This study addressed the longstanding question on the function of the sperm cell specific H3.10 variant. Being a poor substrate for histone methyltransferases due to amino acid differences surrounding lysine 27 in the histone tail, H3.10 is part of a multi-layer mechanism leading to H3K27me3 reprogramming in the male sperm cells.