



Published in final edited form as:

Epigenetics. 2009 August ; 4(6): 366.

H3K27me1 is E(z) in animals, but not in plants

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Abstract

Post-translational modifications of histones play key roles in the regulation of gene expression and chromatin structure in eukaryotes. Methylation of histone 3 on lysine 27 (H3K27) is one of the most common and well-studied histone post-translational modifications. The vast majority of research on this histone residue, however, has focused on the trimethylated form (H3K27me3). Despite occurring at higher levels than H3K27me3 in animals and plants, the monomethylated form of H3K27 (H3K27me1) remains relatively poorly characterized. The absence of information concerning H3K27me1 is due in large part to the fact that the enzymes catalyzing this epigenetic mark were only recently identified. In this article, we highlight new findings concerning H3K27me1, including the identification of two plant-specific H3K27 monomethyltransferases that are required for gene silencing and heterochromatin condensation. We also discuss the emerging similarities and differences in H3K27 methylation in plant and animal systems.

Keywords

ATXR5; ATXR6; H3K27me1; heterochromatin; gene silencing; chromatin condensation

Introduction

Almost a decade ago, the SET domains of the human and mouse orthologs of SUPPRESSOR OF VARIATION 3-9 HOMOLOG 1 were found to catalyze the methylation of histone 3 lysine 9 (H3K9) ¹. This discovery had a major impact on the field of epigenetics, as it led to the identification of a large number of methyltransferases that establish and maintain various post-translational modifications (PTM) on histones in eukaryotes ². Today, the functional analysis of a particular histone PTM still frequently begins with the identification of the enzyme (s) responsible for it. Removal of the enzyme, and hence the PMT, often leads to molecular, cellular, and developmental phenotypes, which can be analyzed to understand the role of the specific PTM. An alternative approach to study the significance of specific histone modifications is to eliminate “reading” proteins, which carry domains capable of specifically interacting with particular PTMs ³. Both approaches, however, are often complicated by genetic redundancy, a lack of knowledge regarding the proteins involved, and/or absence of mutant alleles for these proteins.

Among histone PMTs, histone 3 lysine 27 monomethylation (H3K27me1) can be considered a quintessential “orphan” mark. Although H3K27me1 is found at high levels in most complex eukaryotic systems ⁴, relatively little is known about the proteins that catalyze and bind H3K27me1 or the biological role of this epigenetic mark. The recent identification of enzymes responsible for H3K27me1 in plants and mammals has yielded a first glimpse into the functions

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of this histone PTM^{5, 6}. In this article, we present the new findings concerning H3K27me1 in Arabidopsis and compare these findings with what is known about this epigenetic mark in animals. In addition, we speculate on how H3K27me1 might be maintained during DNA replication in Arabidopsis and its possible biochemical function.

Identification of novel H3K27 monomethyltransferases in Arabidopsis

Sequence analysis of the paralogous proteins ARABIDOPSIS TRITHORAX-RELATED PROTEIN 5 (ATXR5) and ATXR6 shows that they contain divergent SET domains. Although most SET domains function to methylate specific lysine residues of H3¹, some have been shown to catalyze the methylation of non-histone substrates⁷⁻¹⁰. Thus, it remained unclear for some time whether ATXR5 and ATXR6 actually play a role in histone methylation, as phylogenetic analysis did not indicate strong similarity with functionally-characterized SET domains known to methylate H3¹¹. The *in vitro* characterization of the enzymatic activity of ATXR5 and ATXR6 by our group has shown that these enzymes are in fact H3K27 methyltransferases⁵. More specifically, ATXR5 and ATXR6 are H3K27 monomethyltransferases, meaning that they convert H3K27me0 to H3K27me1, but not to H3K27me2 or H3K27me3 (Fig. 1). The Arabidopsis genome encodes three other H3K27 methyltransferases: CURLY LEAF (CLF), SWINGER (SWN), and MEDEA (MEA)¹². These three enzymes are homologs of the Drosophila protein ENHANCER OF ZESTE (E(z)). Like E(z), CLF, SWN, and MEA are the catalytic subunits of Polycomb Repressive Complex 2 (PRC2)-like complexes in plants (Fig. 1), which are responsible for the deposition of H3K27me3¹³. The presence of a new class of H3K27 methyltransferases in Arabidopsis was somewhat expected, as previous experiments had shown that CLF, SWN, and MEA were not required for the establishment of H3K27me1¹⁴. ATXR5 and ATXR6 are the first eukaryotic H3K27 methyltransferases to be identified that are not orthologous to E(z). It should be noted, however, that the viral protein vSET from the *Paramecium bursaria* chlorella virus 1 is also a non-E(z)-type H3K27 methyltransferase^{15, 16}.

Further work on ATXR5 and ATXR6 has revealed that these enzymes are responsible for the enrichment of H3K27me1 at chromocenters (constitutive heterochromatin) in Arabidopsis⁵. Also, ATXR5 and ATXR6 appear to function redundantly in maintaining the condensed and transcriptionally-silent state of heterochromatin in Arabidopsis, as *atxr5 atxr6* double mutants show both chromatin decondensation and loss of gene silencing. Interestingly, a reduction in H3K27me1 does not affect H3K27me2/3. This last result strongly suggests that the phenotypes observed in *atxr5 atxr6* mutants are due specifically to lower levels of H3K27me1 and that H3K27me2/3 does not depend on H3K27me1 as an intermediate for their catalysis.

Many questions arise from the discovery of the enzymes responsible for H3K27me1 in Arabidopsis. For example, what is the mechanism by which the spatial distribution of H3K27me1 is regulated in Arabidopsis? Apart from their SET domains, ATXR5 and ATXR6 also contain a plant homeodomain (PHD) domain. Because some PHD domains have been shown to bind specific PTMs on H3 proteins^{17, 18}, one possibility would be that the PHD domains of ATXR5 and ATXR6 could control the distribution of H3K27me1 by interacting only with certain forms of H3. Analysis of the spatial distribution of H3K27me1 in Arabidopsis shows that it is enriched at the chromocenters^{14, 19}. If the binding and catalytic activity of ATXR5 and ATXR6 are linked, we can hypothesize that the PHD domains of these proteins might interact with an epigenetic mark that is also enriched in heterochromatin.

Different mechanisms are involved in the deposition of H3K27me1 in plants and animals

The analysis of histone PTMs in a broad spectrum of eukaryotes has revealed both conservation and divergence in their spatial distributions and biological roles^{4, 20}. For H3K27me1, one clear difference between animals and plants is the molecular mechanism by which this PTM is deposited onto histones. Homologs of ATXR5 and ATXR6 are present in land plants, but not in metazoans. Thus, different proteins are used to monomethylate H3K27 in animals. In *Drosophila*, H3K27me1 is completely dependent on E(z)²¹. Similarly, two homologs of E(z), ENHANCER OF ZESTE HOMOLOG 1 (EZH1) and EZH2, function redundantly in mammals to control global levels of H3K27me1⁶. The mammalian PRC2 subunit EMBRYONIC ECTODERM DEVELOPMENT (EED) was also demonstrated to be required for H3K27me1²². Taken together, these results suggest that PRC2-type complexes are required for H3K27me1 in mammals (Fig. 1).

E(z) and its homologs have been implicated in both H3K27 mono- and trimethylation. How these enzymes regulate the level of H3K27 methylation, however, remains to be determined. The answer to this question may lie in the particular protein composition of the different PRC2-like complexes. For example, SUPPRESSOR OF ZESTE 12 (SUZ12) has been found to be essential for H3K27me3, but dispensable for H3K27me1 (Fig.1)²³. In the same manner, the mammalian protein PHD FINGER PROTEIN 1 (PHF1), a homologue of the *Drosophila* protein POLYCOMBLIKE (PCL), was shown to associate with PRC2 components and stimulate formation of H3K27me3, but not H3K27me1 or H3K27me2, *in vitro* and *in vivo*²⁴⁻²⁶. Although the *Arabidopsis* genome contains three SUZ12-related proteins (VERNALIZATION2 (VRN2), EMBRYONIC FLOWER2 (EMF2)), and FERTILIZATION-INDEPENDENT SEED 2 (FIS2)¹³, no PHF1/PCL homologs are found in *Arabidopsis*. This suggests that both conserved and plant/animal-specific mechanisms may regulate the histone methyltransferase activity of E(z)/PRC2-like complexes. It remains to be determined whether the plant E(z) homologs also contribute to H3K27me1. Only a 22 percent decrease in H3K27me1 was observed in *atxr5 atxr6* mutants, which suggests that other H3K27 methyltransferases can also catalyze this epigenetic mark⁵. However, a definitive conclusion still cannot be drawn, as the *atxr6* allele used in the study is not a null.

Aside from being catalyzed by non-homologous enzymes, there are striking similarities between the global distribution of H3K27me1 in plants and animals. Immunostaining experiments have shown centromeric and pericentromeric enrichments for H3K27me1 in mammals and *Arabidopsis*^{14, 19, 27}. More precise mapping of this histone PTM in mammals has revealed that H3K27me1 is actually widely distributed in the genome, being only excluded from the region surrounding the promoter of active genes²⁸. Similarly, the distribution of H3K27me1 was found recently to be largely homogenous over the entire length of the chromosomes in two gymnosperms and maize^{29, 30}. It should be noted that the compact nature of the constitutive heterochromatin in both plants and animals may have the effect of enhancing the apparent heterochromatic enrichment of H3K27me1 in immunolocalization experiments. The similar genomic distributions in plants and animals suggests that the role of H3K27me1 might be at least partially conserved in eukaryotes.

Epigenetic inheritance of H3K27me1 in plants

One of the most interesting questions concerning epigenetic marks like H3K27me1 is the mechanism by which they are maintained through DNA replication. Although DNA replication is a challenge for chromatin homeostasis, it also provides the cell with a window of opportunity for chromatin remodeling^{31, 32}. Transcription factors and other bound proteins that might otherwise restrict access to chromatin are displaced, allowing passage of the replication fork.

In addition, the newly-synthesized histones incorporated on both daughter strands can be given new PTMs that can alter the epigenetic status of the associated locus. A few SET-domain proteins in animals have already been shown to function during DNA replication^{33, 34}.

Results from previous work indicate that ATXR5 and ATXR6 may be involved in chromatin modification during DNA synthesis. ATXR5 and ATXR6 are expressed at highest levels during DNA synthesis and physically interact with PROLIFERATING CELL NUCLEAR ANTIGEN (PCNA) through a PCNA-interacting protein box motif³⁵. These two findings indicate that ATXR5 and ATXR6 are well positioned to modify histones during DNA replication. Newly-synthesized histones could be a substrate for monomethylation by ATXR5 and ATXR6, before and/or after deposition on the chromatin. Modification of these histones would prevent dilution of H3K27me1 by restoring pre-replication levels of this mark. Interestingly, it has been speculated that H3K27me1 might also be the default state of newly assembled chromatin following DNA replication in mammals²⁸. PCNA seems to remain associated with the newly-replicated daughter strands, at least in animal cells, for approximately 20 minutes³⁶. If this also happens in plants, it would represent a temporal window for PCNA-binding factors, like ATXR5 and ATXR6, to perform their activity.

What is the role of H3K27me1?

In addition to increasing our understanding of the mechanisms that maintain or modify chromatin during DNA replication, further studies of ATXR5 and ATXR6 may also shed light on the biochemical function of H3K27me1. Our work strongly suggests that H3K27me1 plays a critical role in maintaining heterochromatic chromatin condensation and gene silencing in Arabidopsis⁵. It remains unknown, however, how this histone modification signals such events. One possibility is that H3K27me1 simply acts as a recruitment signal for an H3K27me1-binding protein. Alternatively, monomethylation of H3K27 (and possibly H3K27me2 and H3K27me3) may act in a negative manner. This could occur by either preventing H3K27me0-interacting proteins from binding chromatin or by blocking alternative histone modifications from being made on H3K27.

If an H3K27me1-binding protein exists, what might we expect its function to be? Based on our results, this protein would likely promote chromatin condensation, either directly or as part of a complex. Chromatin-remodeling enzymes are good candidates for this activity, as they use the energy stored in ATP to promote nucleosome sliding, remodeling, and dissociation³⁷. DECREASE IN DNA METHYLATION1 (DDM1) is a chromatin-remodeling protein in Arabidopsis that could potentially be linked to H3K27me1. Similar to *atxr5 atxr6* double mutants, *ddm1* plants show chromatin decondensation and reactivation of repressed heterochromatic elements (Fransz 2006). These effects of *ddm1* mutations, however, are associated with decreased levels of DNA methylation, and our work clearly shows that *atxr5 atxr6* has no impact on DNA methylation^{5, 38}. Another chromatin-remodeling protein that could be part of an H3K27me1 pathway is MORPHEUS' MOLECULE 1 (MOM1)³⁹. The MOM1 protein is responsible for silencing heterochromatic elements, and it does so, like ATXR5 and ATXR6, without affecting DNA methylation³⁹⁻⁴². However, unlike *atxr5 atxr6* mutants, compaction of the heterochromatin is unaffected in *mom1*⁴⁰. More work will have to be done in order to identify the factor(s) working downstream of H3K27me1.

Concluding Remarks

For various reasons, our understanding of H3K27me1 has lagged behind that of other epigenetic marks. With the identification of the enzymes responsible for this mark in animals and plants, however, the near future should reveal many exciting discoveries about the function of this mark in eukaryotic cells. It will be particularly interesting to see the degree of similarity

and divergence that exists between plants and animals regarding the deposition of H3K27me1 and its biological function.

Acknowledgments

This work was supported by a grants to S.D.M. from the NIH (GM075060) and to Y.J. from NATEQ (Fonds québécois de recherche sur la nature et les technologies).

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Abbreviations

ATXR5	ARABIDOPSIS TRITHORAX-RELATED PROTEIN 5
ATXR6	ARABIDOPSIS TRITHORAX-RELATED PROTEIN 6
CLF	CURLY LEAF
DDM1	DECREASE IN DNA METHYLATION1
EED	EMBRYONIC ECTODERM DEVELOPMENT
EMF2	EMBRYONIC FLOWER2
E(z)	ENHANCER OF ZESTE
EZH1	ENHANCER OF ZESTE HOMOLOG 1
EZH2	ENHANCER OF ZESTE HOMOLOG 2
FIS2	FERTILIZATION-INDEPENDENT SEED 2
H3	histone 3
K	lysine
MEA	MEDEA
me	methyl
MOM1	MORPHEUS' MOLECULE 1
PHF1	PHD FINGER PROTEIN 1
PHD	plant homeodomain
PRC2	Polycomb Repressive Complex 2
PCL	POLYCOMBLIKE
PTM	post-translational modifications
PCNA	PROLIFERATING CELL NUCLEAR ANTIGEN
SUZ12	SUPPRESSOR OF ZESTE 12
SWN	SWINGER
VRN2	VERNALIZATION2

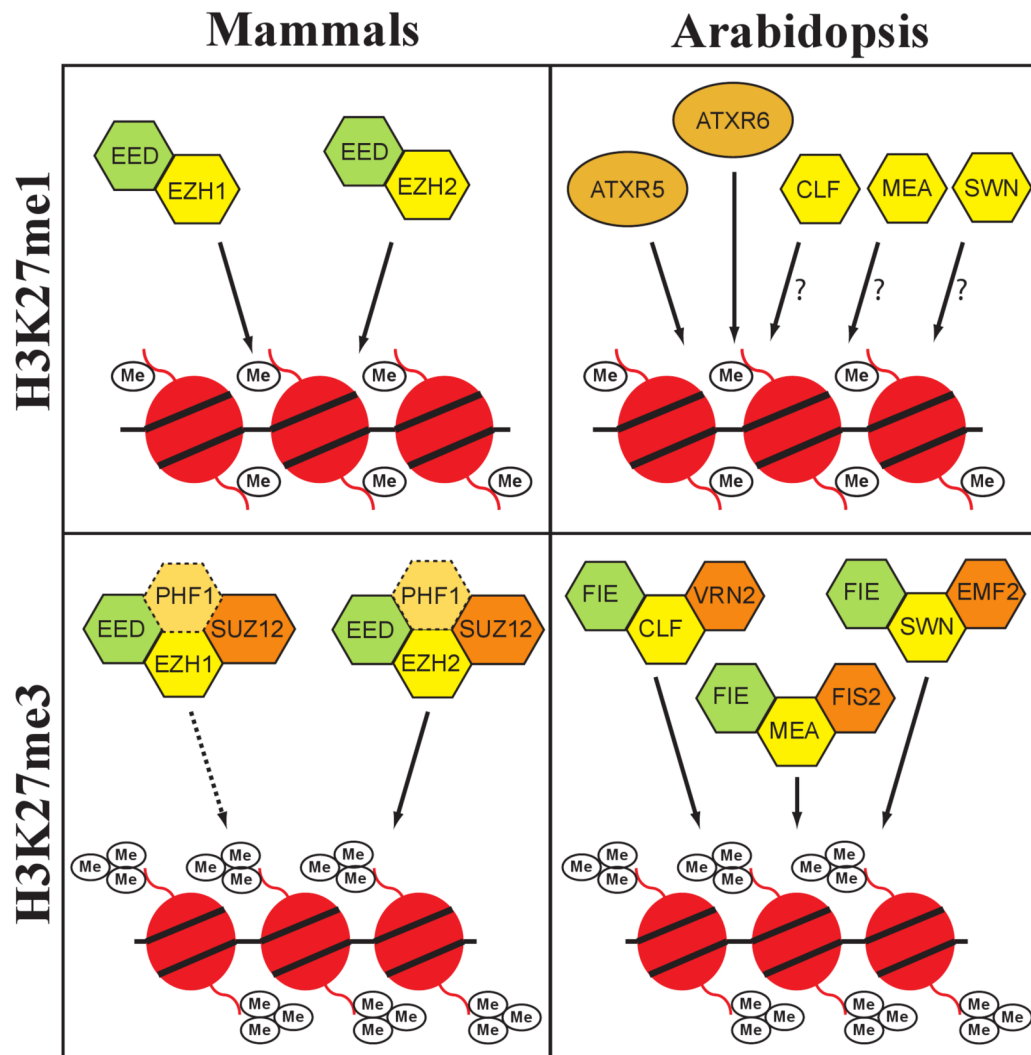


Figure 1. Proteins involved in mono- and trimethylation of H3K27 in mammals and Arabidopsis. PRC2 core components and other associated proteins are shown in hexagons bounded by solid lines and dashed lines, respectively. Non-PRC2 proteins are shown in ovals. Homologous proteins are color-coded.