In plants, the CG and CHG methylation during cell division are methylation in a promoter region of a gene often causes gene silencing and thus maintain genome stability and integrity. In addition, cytosine transposons, repetitive elements and other invasive DNA elements, CG context, whereas in plants it also occurs in CHG and CHH (H=A, T or C) in addition to CG. DNA methylation is critical to silence transposons, repetitive elements and other invasive DNA elements, and thus maintain genome stability and integrity. In addition, cytosine methylation in a promoter region of a gene often causes gene silencing. A histone protein is composed of a structural core and an unstructured N-terminal tail that is subject to various modifications. Depending on the position of a modified residue and the modification nature, histone modifications may lead to promotion or prevention of gene expression. For instance, histone acetylation, histone H3 lysine-4 (H3K4) methylation, H3K36 methylation and H2B monoubiquitination are associated with active gene expression, whereas histone deacetylation, H3K9 dimethylation (H3K9me2), H3K27 trimethylation (H3K27me3) and H2A monoubiquitination are often linked to gene silencing. Histone-modifying enzymes function either as a ‘writer’ or an ‘eraser’ to introduce or remove a modification, respectively [1]. A chromatin mark is typically recognized and bound specifically by a ‘reader’ and further interpreted by an ‘effector’ protein or protein complex [1], leading to a change in the chromatin state of a particular locus, which may cause gene activation or repression.

Cytosine DNA methylation occurs in a wide variety of eukaryotes. In animals such as mammals, cytosine methylation primarily is in the CG context, whereas in plants it also occurs in CHG and CHH (H=A, T or C) in addition to CG. DNA methylation is critical to silence transposons, repetitive elements and other invasive DNA elements, and thus maintain genome stability and integrity. In addition, cytosine methylation in a promoter region of a gene often causes gene silencing. In plants, the CG and CHG methylation during cell division are maintained by the highly-conserved DNA Methyltransferase 1 (MET1) and the plant-specific chromomethylase 3 (CMT3), respectively, and the maintenance of CHH methylation requires small RNAs (for target selection) and Domains-Rearranged Methyltransferase 2 (DRM2) [2].

The non-coding small RNAs (siRNAs) trigger de novo CHH methylation through two pathways in plants, including the well-characterized siRNA-directed DNA methylation (RdDM) pathway and the NERD pathway (NERD for 'Needed for RDR2-independent DNA methylation') [3,4]. In RdDM, RDR2 (for RNA-Dependent RNA Polymerase 2)-dependent 24-nt siRNAs are loaded to an ARGONAUTE 4 (AGO4)-containing effector complex, which associates with DRM2 and is guided to target loci by the siRNAs, resulting in cytosine methylation. In the NERD pathway, RDR1 or RDR6-dependent siRNAs such as 21-nt siRNAs, are loaded to an AGO2 effector complex; NERD, a GW repeat- and PHD finger-containing protein, directly interacts with AGO2 and target chromatin as well, and conceivably works together with the siRNAs to guide the effector complex to target loci, leading to cytosine methylation [3]. In addition to CHH methylation, the NERD pathway is also involved in CG and CHG methylation in a subset of non-conserved loci, as revealed in a study using the model plant Arabidopsis thaliana [3].

At the RdDM target loci, there are not only silencing cytosine methylations, but also repressive chromatin modifications such as histone deacetylation and H3K9me2, and these modifications together establish a repressive chromatin environment at a target locus for its silencing [4], in which cytosine methylations promote or facilitate repressive histone modifications, and vice versa. In addition, at the NERD target loci, the NERD protein not only mediates cytosine methylation, but also recognizes and binds to un-methylated H3K4, and maintains lower levels of H3K4 methylation (note that elevated levels of this mark are associated with gene expression), to enforce silencing of its target loci [3]. Repressive histone/chromatin modifications are known to be required for DNA methylation. A few chromatin modifiers have been shown to be required for de novo and/or maintenance of cytosine methylation, among which are the ATP-dependent chromatin remodeler DDM1, the H3K9 methyltransferase KYP, Histone Deacetylase 6, and an H2B deubiquitinase called SUP32 [4]; in addition, several H3K4 demethylases known as LDL1, LDL2 and JM14/PKDM7B are also partly required for cytosine methylation [5-7]. Obviously, siRNA-triggered silencing of the RdDM and NERD target loci involves not only DNA methylation, but also repressive histone modifications. In addition to siRNAs, long non-coding RNAs (lncRNAs; >200 nts) can also trigger chromatin silencing at their target loci. These chromatin-silencing mechanisms are referred hereafter as to non-coding RNA-mediated Chromatin Silencing (RmCS).

In the lncRNA-triggered RmCS, the RNA trigger causes repressive histone modifications such as H3K27me3 (catalyzed by Polycomb repressive complex 2; PRC2) for silencing, which may not involve cytosine methylation. For instance, in Arabidopsis the COLDAIR lncRNA-triggered chromatin silencing of a central floral repressor known as FLC, involves H3K27me3, but not cytosine methylation [8]. In response to a prolonged cold exposure, COLDAIR expression is induced from the first intron of FLC, and subsequently, this lncRNA recruits a PRC2-like complex to FLC chromatin to deposit H3K27me3, leading to FLC silencing. A similar silencing mechanism has been observed in mammals: the mammalian HOTAIR lncRNAs spark X chromosome silencing via the recruitment of a PRC2 complex to the target chromosome, leading to its silencing or inactivation [9].

Genome-wide transcriptional analyses have identified thousands of lncRNAs in human and the Arabidopsis plant as well [10,11]. Some of these lncRNAs are likely to be involved in chromatin silencing.

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RmCS triggered by long (lncRNAs) or short (siRNAs) non-coding RNAs, is emerging as an important chromatin mechanism underlying eukaryotic gene regulation.

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