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Histone H1 in *Arabidopsis thaliana*

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Histone H1 in *Arabidopsis thaliana*

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Report

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Histone H1 in *Arabidopsis thaliana*

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Histone H1, or linker histone, are unique histones that bind to the nucleosome to facilitate higher order chromatin structure. The linker histones, when compared to the core histones that make up the nucleosome, are poorly understood especially in plants. Linker histones are vital for plant development as well as for cell cycle regulation, sharing many qualities with animal linker histones. In this report, the first two parts introduce the current literature of H1, including result from non-plant systems, and the third section is a research proposal describing a research project to elucidate the roles of linker histones on the regulation of *FLOWERING LOCUS C* (*FLC*) in *Arabidopsis thaliana.*

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Chapter One: Introduction to Histone H1

Nucleosome Structure

The nucleosome is a structural unit of DNA compaction involving about 146 base pairs of DNA wrapped around a core group of histone proteins. These histones are evolutionarily conserved among eukaryotes and are required for not only the compaction of DNA into the 30-nanometer fiber (Thoma et al. 1979, Meyer et al. 2011), but also some aspects of gene regulation. Each nucleosome core is an octamer made up with two tetramers of four different histone subunits. Histone H2A and H2B form a heterodimer through hydrophobic interactions, with two of these heterodimers forming one of the base tetramers. Histone H3 and H4 form another heterodimer and together these form the second base tetramer (Oudet et al. 1975, Kornberg 1979). It binds to the histone octamer and linker DNA, the DNA between two nucleosome cores, in order to facilitate higher order chromosome structure by promoting the folding of the DNA into the 30-nanometer fiber (Robinson et al. 2006, Wong et al. 2007, Syed et al. 2010, Meyer et al. 2011). Linker histones tend to be lysine-rich which provides an overall positive charge allowing the histones to bind to DNA and neutralize the negative charge along with the core histones (Clark and Kimura 1990).

Histone H1 Structure and Evolution

Unlike the core histones that make up the nucleosome, histone H1 is not as evolutionarily conserved among Eukaryotes when compared with the canonical histones (Harshman et al. 2013). All linker histones among the higher Eukaryotes are lysine-rich proteins with a conserved central globular domain (GH1), which binds to DNA (Harshman et al. 2013).

They consist of a tripartite structure with the globular domain flanked by a variable N- and C- terminal chain. Unlike many animal H1 histones, the *Arabidopsis* histones contain highly variable flanking regions. These variable-flanking regions suggest that there can be different functions for these histones such as differential gene expression or developmental regulation (Harshman et al. 2013). These differences might also be involved in regulating the affinity for histone-chromosome binding to modulate chromatin condensation (Hendzel et al. 2004).

In *Arabidopsis*, there are three variants of histone H1: H1-1 (At1g06760), H1-2 (At2g30620), and H1-3 (At218050). Histone H1-3 is structurally divergent compared to the other *Arabidopsis* H1 proteins (Figure 1) and its expression is induced during drought stress (Ascenzi and Gantt 1997, Ascenzi and Gantt 1999). This linker histone variant is upregulated during drought-stress, but is not required for the expression of drought-resistance genes (Ascenzi and Gantt 1999). Although some plants share a similar drought induced linker histone (Scippa et al. 2004), this type of histone is not always associated with drought and so may have other functions in development (Over and Michaels 2013) (Figure 1).

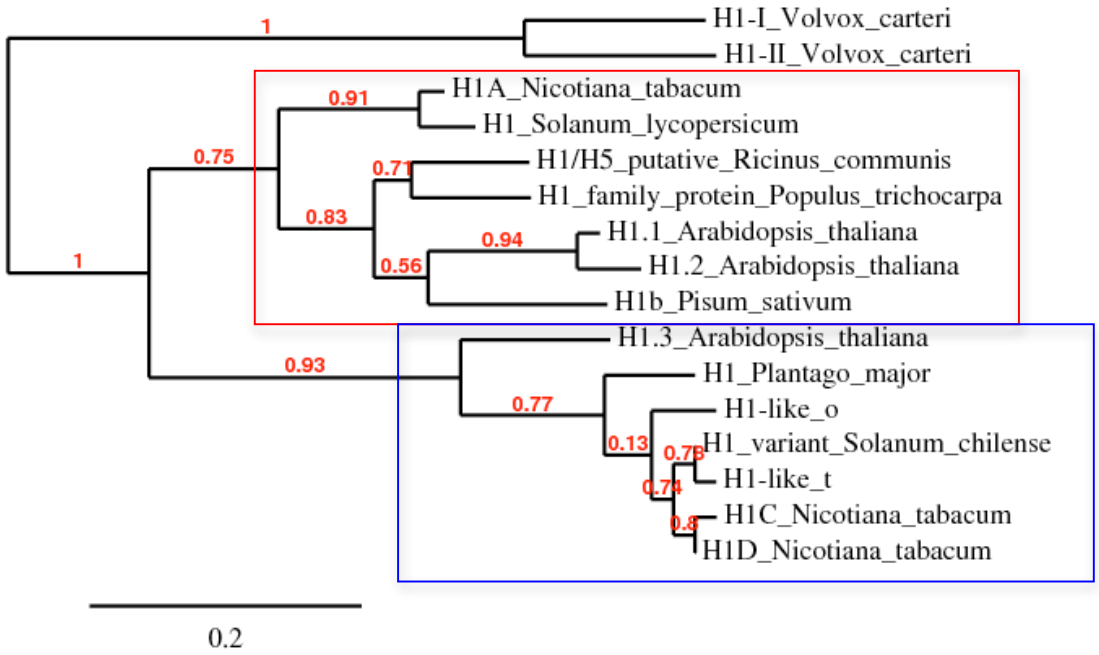

Figure 1. Phylogenetic analysis of H1 in plants and algae. There are two distinct clades. The blue clade contains the *Arabidopsis* drought-inducible linker histone. This variant is in a clade with the *Nicotiana tabacum* drought linker histone. The red clade contains the main histone variants for *Arabidopsis* as well as for other plants. The algae are the outliers. (Castresana 2000, Guindon and Gascuel 2003, Edgar 2004, Anisimova and Gascuel 2006, Chevenet et al. 2006, Dereeper et al. 2008, Dereeper et al. 2010)

Effects on Plant Development

Like in animals, there are substitutions among histone variants and different concentrations of H1 are found during cellular differentiation in plants (Over and Michaels 2013). There have been many studies to elucidate the role of H1 during the germination, but it is not yet known whether the levels of H1 change when chromatin compaction and de-condensation occur (Baluška and Kubica 1992, Dicorato et al. 1995, Alatzas et al. 2008). It was found that during cell differentiation there was an overall reduction of H1 and subsequent chromatin relaxation (She et al. 2013).

Changes in H1 concentration can also lead to visible phenotypes in plants. In Wierzbicki and Jerzmanowki 2005, a knockdown of all three H1 variants in *Arabidopsis* cause stochastic changes in DNA methylation, which lead to a stochastic phenotype. Plants that had the greatest change in DNA methylation patterns from wild-type had more severe phenotypes that ranged from small stature and late flowering to homeotic disruptions. It is also interesting to note that these defects were heritable suggesting an epigenetic role for linker histones. Overexpression of linker histones could cause developmental issues as well. In tobacco plants overexpressing *Arabidopsis* H1, there were also stochastic changes affecting growth and development of the tobacco ranging from a mild to severe phenotype affecting morphology and flowering time (Prymakowska-Bosak et al. 1996). Although that overexpressing linker histones could potentially have a developmental effect, it has to be taken into consideration that the study above expressed a non-native protein which could be the main cause of the developmental phenotypes.

H1 and the Cell Cycle

Plant linker histones, like animal linker histones, can have sites within their C-terminal domain available for Cyclin-dependent Kinase-mediated (CDK) phosphorylation (Ascenzi and Gantt 1997, Slaninova et al. 2003). CDK-mediated phosphorylation of histone H1 has been found in tobacco, rice, corn, and algae (Sauter et al. 1995, Zhang et al. 1996, Zhao and Grafi 2000, Slaninova et al. 2003). Plant linker histones can also vary in the number of CDK phosphorylation sites indicating that some variants may be more vital in regulating cell cycle progression (Over and Michaels 2013). Plants may use H1 variants that lack necessary a CDK-mediated phosphorylation site in order to suppress or slow cell cycle division. *Arabidopsis* H1.3, the drought inducible linker histone, does not have obvious CDK-mediated phosphorylation site indicating that it may replace other H1 variants to prevent cell cycle progression (Over and Michaels 2013). Although this could be a factor influencing cell-cycle progression, H1.3 was not the most abundant variant within non-dividing cells indicating there are probably more important factors and mechanisms, such as phytohormones, used to inhibit cell division in plants (Ascenzi and Gantt 1999).

Chapter Two: Histone H1 and its role in DNA methylation

Histone H1 is known to stabilize chromatin structure, but little else is known about the biological function of these linker histones especially in plants. In lower eukaryotes, there was no visible phenotype or effect on specimen viability in knockouts of H1 (Shen et al. 1995, Ramon et al. 2000). However in higher Eukaryotes, there are many isoforms of histone H1, which make knockout experiments problematic. The study of histone H1 especially when compared with the core histones is a relatively untouched subject particularly in the plant system. Nonetheless, there have been a few studies that have uncovered a connection between histone H1 and DNA methylation. Particularly, studies involving transposable element silencing and imprinting have shown that the linker histones have a role in both the methylation and demethylation of DNA in plants.

H1 mutants involved in stochastic DNA methylation changes

Histone H1 is associated with DNA methylation. In Wierzbicki and Jerzmanowski 2005, they used double-stranded RNA (dsRNA) of all three *Arabidopsis* histone H1 genes to produce plants with about a 90% reduction in H1 expression. These plants did not have genome-wide changes on DNA methylation, neither hypo- nor hyper methylation, but there were changes on the DNA methylation patterns on some single copy genes and repetitive sequences. The changes did not occur consistently among the transgenic plants; instead they showed stochastic alteration in DNA methylation patterns (Wierzbicki and Jerzmanowski 2005). To observe the effects of reduced levels of histone H1 on constitutive heterochromatin and ribosomal DNA, a restriction enzyme analysis was used and it was found that CpG methylation in constitutive heterochromatin was not reduced

in transformants but rDNA methylation in the CpG and CpNpG contexts were stochastic in manner with transformants sometimes exhibiting increased or decreased methylation (Wierzbicki and Jerzmanowski 2005).

Transposable Element Silencing

Although it is known that H1 knockdown in *Arabidopsis* can cause stochastic changes in DNA methylation in different contexts (Wierzbicki and Jerzmanowski 2005), the connection between H1 and DNA methylation is becoming better understood through the use of DEFICIENT IN DNA METHYLATION 1 (DDM1) defective plants. In ddm1 mutants, there is a strong reduction in DNA methylation (Jeddeloh et al. 1999), but the requirement for DDM1 was not yet known. In H1 knockdown plants, the DNA methylation patterns were properly restored in the *ddm1* mutant especially within the methylation contexts affected by the RNA-directed DNA methylation (RdDM pathway) (Zemach et al. 2013). DDM1 is a nucleosome remodeler and without the H1 methylation machinery was still able to properly methylate the DNA. This suggests that the linker histones act as a physical block preventing DNA methylation and requiring DDM1 to remodel the chromatin by removing the linker histones (Figure 2).

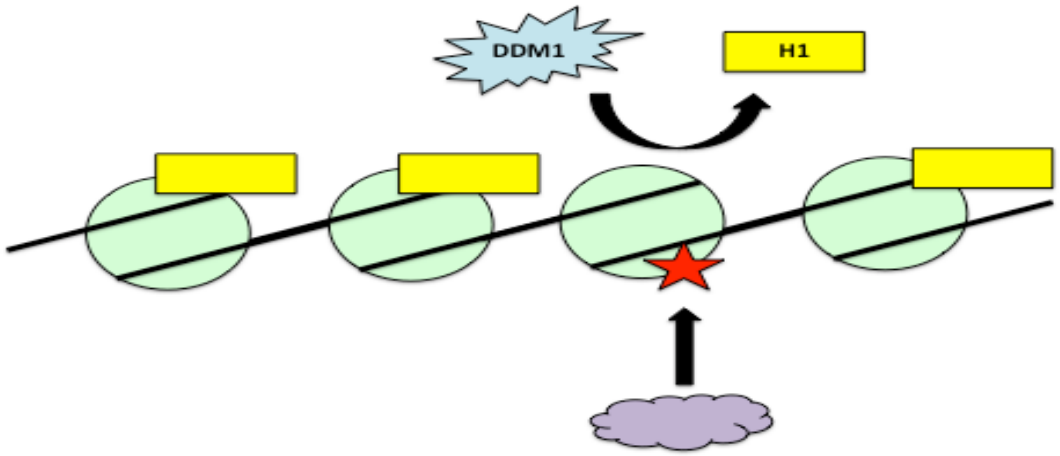

Figure 2. Schematic of H1 and Transposable element silencing. DDM1 removes the linker histone by remodeling the nucleosome. This allows the methylase/methyltransferase (represented by purple) to methylate the DNA (red star).

H1 and its Role in Imprinting

In *Arabidopsis*, DEMETER (DME) regulates genomic imprinting by excising 5'-methylcytosine from DNA. This DNA glycosylase demethylates the MEDEA (MEA) promoter of the maternal allele allowing gene expression in the endosperm. For the paternal allele, MEA remains methylated and gene expression is repressed. Although it was unknown whether DME interacted with other proteins, it was recently discovered that DME could interact with H1.2 (Rea et al. 2012). This data comes from a yeast two-hybrid screen so the *in vivo* interaction has not yet been shown (Rea et al. 2012). Although the direct interaction between DME and histone H1 has not been shown in vivo, the genetic analysis of H1 and the imprinted genes *MEA*, *FWA*, and *FIS2* did show that H1 is required for DME regulation.

In Rea et al., they used a triple mutant, h1.1-1 h1.2-1 h1.3-1, which was a knockout for H1.1 and a knockdown for both H1.2 and H1.3. Although the RNAi approach mentioned above from Wierzbicki and Jerzmanowski 2005 also created an h1 triple knockdown, this allele is dominant and not useful for imprinting studies. Based on the data presented in Rea et al. is Figure 3, it shows that a wild-type maternal allele is necessary for the expression of the imprinted genes *FWA* and *FIS2*. When the maternal allele was the h1 triple mutant, the expression of the imprinted genes was drastically reduced when compared with the wild-type and reciprocal crosses suggesting the H1 might play a role in DME-mediated DNA methylation at imprinted loci.

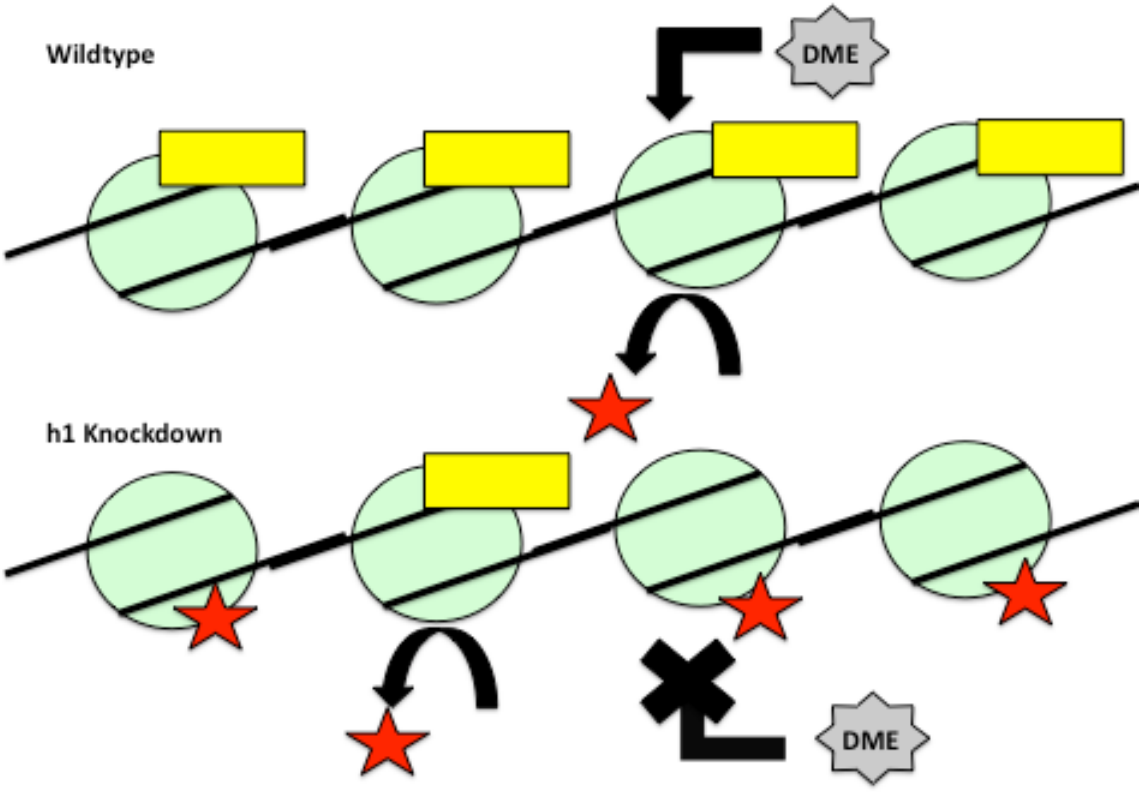

Figure 3. DME-mediated demethylation at imprinted loci. The nucleosomes represent the maternal allele of the imprinted gene *FWA*. With wildtype H1 (yellow rectangle), DME demethylates the DNA allowing the expression of the imprinted gene. Conversely, in the h1 knockdown, without the presence of H1 DME cannot mediate DNA demethylation causing reduced expression of *FWA*.

Chapter Three: *FLC* repression and Histone H1

Due to the sessile nature of plants, plants have developed many mechanisms to adapt to their environment. In particular, cold acclimation in plants is often necessary for many epigenetic changes. *FLC* is perhaps one of the most well studied environmentally effected genes. *FLC* is a major component of the vernalization pathway in *Arabidopsis* acting as the primary repressor. It encodes a MADS-domain transcription factor, which represses the transcription of *SUPRESSOR OF CONSTANS 1* (*SOC1*) and *FLOWERING LOCUS T* (*FT*), two important floral integrators responsible for promoting the transition from a vegetative state to a reproductive state (Hepworth et al. 2002). During the prolonged cold exposure, levels of VERNALIZATION INSENSITIVE 3 (VIN3) increase and form a complex with the POLYCOMB REPRESSIVE COMPLEX 2 (PRC2) (Wood et al. 2006, De Lucia et al. 2008).

Over the course of vernalization, cold treatment, PRC2 mediates the deposition of trimethylated H3 at lysine 27 causing FLC repression. In addition to histone modification, the lncRNA *COLDAIR* is induced during the cold to associate with PRC2 acting as a scaffolding mechanism (Heo and Sung 2011). Subsequently, after the repression of FLC, the plant is now able to transition from the vegetative state to floral state. In order for the plant to flower, FLC must remain in a repressed state. This repressive state is mitotically stable and serves as a plant "memory" for winter (Henderson and Jacobsen 2007). However, it is not meiotically stable and FLC returns to its active state in the next generation (Trevaskis et al. 2007) (Figure 4).

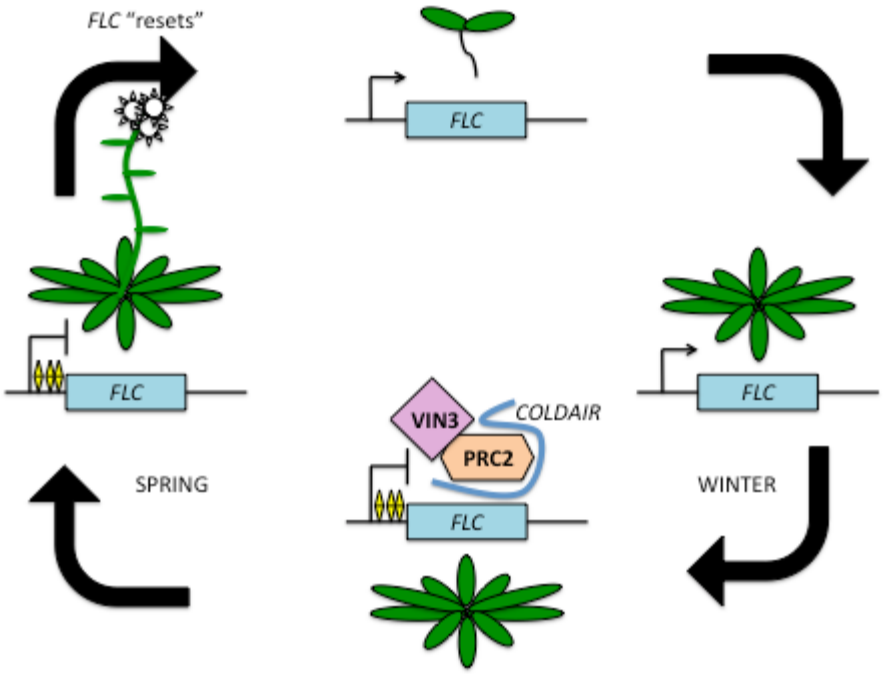

Figure 4. The vernalization pathway in *Arabidopsis*. *FLC* is actively expressed in plants before winter. During vernalization, PRC2 deposits H3K27me3 (represented by the diamond) at the *FLC* promoter region with the lncRNA *COLDAIR* acting as a scaffold. This represses *FLC* expression allowing the plant to flower after returning to warmer temperatures in the spring. *FLC* then returns to its active state in the next generation.

Although the mechanism for initial FLC repression is fairly well understood, the maintenance of the repressive chromatin mark is not. This maintenance of the repressive chromatin state could be because of the chromatin structure itself, suggesting that histones may be involved. Because of this we are going to look at histone H1 and if the occupancy of this histone changes over the course of vernalization. Histone H1 is a relatively unstudied protein, especially in plants, and its effects on *FLC* repression and the vernalization pathway are unknown. It is possible that histone H1 has a role in the repression and maintenance of *FLC* by facilitating higher order chromatin structure, thereby repressing *FLC* and promoting the floral transition.

Potential effects of histone H1 on *FLC* repression

Because relatively little is known about histone H1 in plants, the hypotheses in this report are based upon a chimeric model of organisms to help explain the potential effect of H1 on *FLC* repression and floral transition. In *Mus musculus*, depletion of histone H1 was found to have an effect on two key histone modifications. In an immunoblot of H1-null ES cells, it was found that there was about a four-fold change in H4K12Ac and about a two-fold decrease in H3K27me3 (Fan et al. 2005). Since PRC2 deposits H3K27me3 at *FLC* chromatin during prolonged cold exposure, this can suggest that H1 might have a role in facilitating and perhaps maintaining the compaction of chromatin through interaction of histone modifications or that histone modification will compensate for the loss of histone H1. It is important to note that in this study, reduction of the linker histones caused a decrease in both H4K12Ac and H3K25me3, active and repressive histone modifications respectively. Therefore reduced levels of histones can have antagonizing stochastic effects both potentially causing more or less chromatin compaction.

Experimental Design

Designing transgenic plants for Histone H1 is difficult because as shown above, an h1 triple knockdown cause stochastic changes in DNA methylation as well as developmental defects (Wierzbicki and Jerzmanowski 2005), making triple null knockout experiments unlikely. In addition to complications from making a triple null mutant, because H1 is not

commonly studied in plants, availability of an H1 antibody specific to each variant is not commercially available. But due to the variation found with the terminal tails, an antibody can likely be made to be specific to the linker histone variant although there is likely to be some non-specific binding. To this end, we propose to make C-terminal tagged H1, both overexpression lines and complimentary lines. The use of the overexpression line is to ensure the incorporation of the tagged histone to the chromatin, since the tag might change chromatin affinity preventing proper binding. Additionally, overexpression of H1 might cause unforeseen developmental defects due to increased chromatin compaction, although this is unlikely because H1 has been shown to compensate for altered histone H1 levels (Prymakowska-Bosak et al. 1996).

After the creation of the transgenic plants containing tagged overexpression and complimentary lines, the goal is to put these transgenes into the *FRIGIDA* (*FRI*) Columbia background. The *FRI* background imparts a need for the plant to undergo vernalization in order to flower. By putting these transgenes into this background, when can monitor the effects of H1 at *FLC* chromatin during and after vernalization. To determine how H1 levels change, a Chromatin Immunopreciptation (ChIP) assay will be used, utilizing antibodies for the tags, at specific time points. These time points include before vernalization, ten days of vernalization, twenty days of vernalization, forty days of vernalization, and ten days post vernalization to ensure that a complete picture for H1 can be made over the entire course of vernalization.

Three Hypotheses

Based upon the information stated above, histone H1 can affect *FLC* in one of three ways. The first hypothesis is that histone H1 is not required for *FLC* repression and maintenance and therefore has no effect on the vernalization pathway. In Fan et al. 2005, as stated above, it was shown that linker histones might be involved in the proper deposition of histone modifications, but this data could be taken that there is a compensation in histone modification instead of a dependence on H1. The second hypothesis is that during vernalization the levels of linker histones will increase to facilitate chromatin condensation, therefore promoting *FLC* repression. This hypothesis can be supported by the fact that the binding of linker histones is transient and compaction during vernalization is facilitated in the short-term by linker histones. This leads to the third hypothesis in which linker histone levels with increase after vernalization at *FLC* chromatin as a type of chromatin maintenance to maintain the repressive state. As stated above, linker histones are transient in binding, so the long-term maintenance of *FLC* repression by histone H1 is probably unlikely, but a possibility.

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