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Non-coding regulation in seasonal flowering control – Insights from *FLC*

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As sessile organisms, plants must adapt to fluctuating environmental conditions, with temperature serving as a key driver of developmental transitions. The ability to accurately perceive and respond to seasonal temperature fluctuations is critical for plant survival and reproductive success. In many species, prolonged exposure to the low temperatures of autumn and winter triggers vernalization, enabling flowering to occur under favourable spring conditions. This process has been extensively characterized in *Arabidopsis thaliana*, particularly through studies of the floral repressor *FLOWERING LOCUS C* (*FLC*). In this mini review, we summarize recent advances in understanding the genetic basis of vernalization, focusing on how non-coding polymorphisms influence *FLC* transcript accumulation and expression of long non-coding RNAs, thereby altering vernalization requirement and efficiency. Variation in the quantitative expression of *FLC* and its homologs has shaped the evolution of diverse life-history strategies of Arabidopsis relatives within the Brassicaceae family. Dissecting how naturally occurring non-coding variants reconfigure the cis-regulatory landscape of *FLC*-like genes will be key to understanding the molecular basis of phenological diversity. Such insights not only illuminate the evolutionary dynamics of flowering time control but also holds promise to provide targets for crop improvement under changing climatic conditions.

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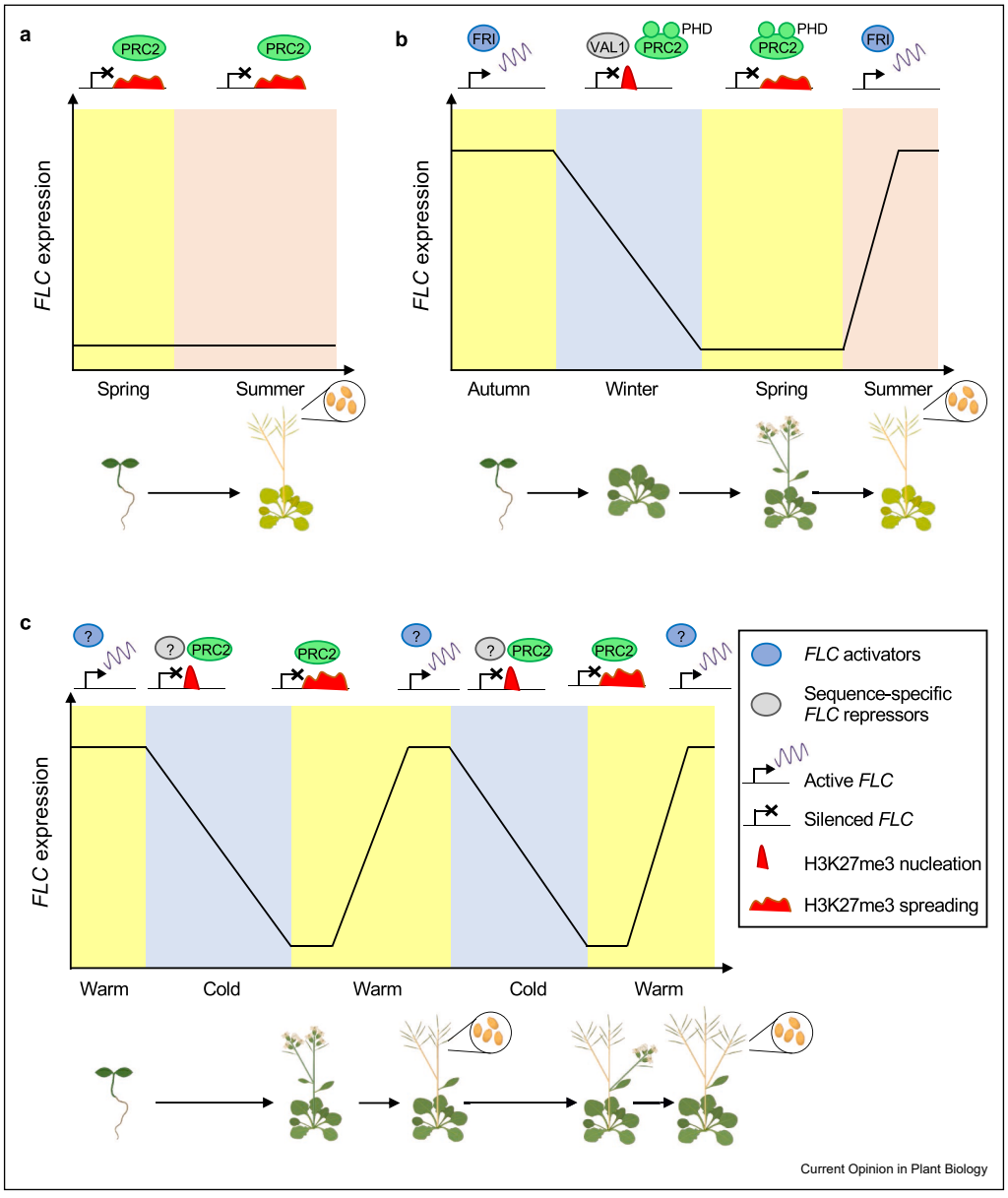
Introduction

Throughout their life cycle, plants must adapt to seasonal environmental shifts. Among these, temperature is a key factor shaping growth, development, and distribution. To align their development with seasonal cues, plants sense daily and annual temperature fluctuations alongside changes in photoperiod length [1]. The circadian clock integrates these signals, allowing plants to fine-tune the timing of critical developmental events [2,3]. One such event, flowering, is pivotal for reproductive success, as its precise timing ensures optimal conditions for seed production.

To cope with varying seasonal environments, plants have evolved different reproductive strategies [4,5]. Certain species exhibit a rapid-cycling strategy (summer-annual) that does not require cold exposure (Figure 1a), allowing them to produce multiple generations within a single growing season when conditions are favourable. In contrast, the overwintering ability of many species depends on exposure to winter cold, which enables them to survive unfavourable conditions and resume development in spring (Figure 1b). Unlike rapid-cycling species, this strategy typically restricts plants to a single generation per year, while helping them avoid mortality during stressful summer conditions.

The overwintering requirement of plants is fulfilled through vernalization, a process by which prolonged exposure to cold stimulates flowering in spring - i.e., when temperatures rise and day length increases [6]. Molecular insights into how plants register noisy temperature cues have emerged from the dissection of vernalization in *Arabidopsis thaliana* [7]. Vernalization is a process involving the multiphase, cold-dependent silencing of the major floral repressor *FLOWERING LOCUS C* (*FLC*) [8,9]. Previous studies have addressed the epigenetic mechanisms underlying vernalization, the key contribution of mathematical modelling to better understand its dynamics, as well as the contrast between laboratory and field studies in capturing environmentally relevant responses. These different aspects of the vernalization mechanism have been extensively reviewed in the scientific literature, including in recent years [7,10–12]. Here, we aim to highlight the crucial role of non-coding regulation in modulating *FLC* expression in *A. thaliana* and *Brassicaceae* relatives, a process that profoundly influences both the

Figure 1



Reproductive strategies are conditioned by the dynamic expression of *FLC* and its orthologs in both annual and perennial Brassicaceae species. (a) In rapid-cycling accessions lacking a functional *FRI*, *FLC* is repressed, resulting in early flowering without the requirement of vernalization. **(b)** In winter-annual plants, FRI-upregulated *FLC* is counteracted by cold through the vernalization process. Prolonged exposure to cold down-regulates *FLC*, and this repression is stably maintained upon return to warmth. During cold, VAL1 protein assists the assembly of PHD-PRC2 complex to promote H3K27me3 deposition at the *FLC* nucleation region. On return to the warm, H3K27me3 spreads along the entire locus. *FLC* is reset during embryogenesis. **(c)** In perennial plants, flowering is induced in some meristems through seasonal *FLC* repression, while its programmed reactivation maintains vegetative development in other meristems. Unlike winter-annual plants, *FLC* expression is not stably repressed by vernalization and increases again upon return to warm conditions. Despite the conservation of H3K27me3 dynamics over *FLC* locus, the molecular components triggering the recruitment of PRC2 during vernalization, as well as the composition of the vernalization-specific PRC2 complex, remain less characterized in perennial plants.

requirement for and efficiency of vernalization. In particular, we draw attention to how natural variation in non-coding genomic regions, which affects the dosage of *FLC* transcripts produced by the plant, has played a major role in winter adaptation and in the evolution of life history strategies across the *Brassicaceae* family. Furthermore, the production of long non-coding RNA (lncRNA) transcripts at the *FLC* locus contributes an

additional regulatory layer, modulating both transcriptional and epigenetic activity. Unless otherwise stated, Arabidopsis hereafter refers to the winter-annual accession of *A. thaliana*.

Molecular basis of vernalization: cis-regulatory elements and long non-coding RNAs

In Arabidopsis, Brassica crops and cereals, prolonged exposure to cold in winter stimulates flowering in spring through vernalization [13]. Prior to vernalization, the MADS-box transcription factor (TF) FLC blocks the activation of *FLOWERING LOCUS T* (*FT*) and *SUPPRESSOR OF OVEREXPRESSION OF CONSTANS1* (*SOC1*), two key floral integrators, thereby delaying Arabidopsis flowering until after winter [9]. In winter-annual Arabidopsis accessions, *FLC* expression is induced by the upstream transcriptional activator FRIGIDA (FRI) [14].

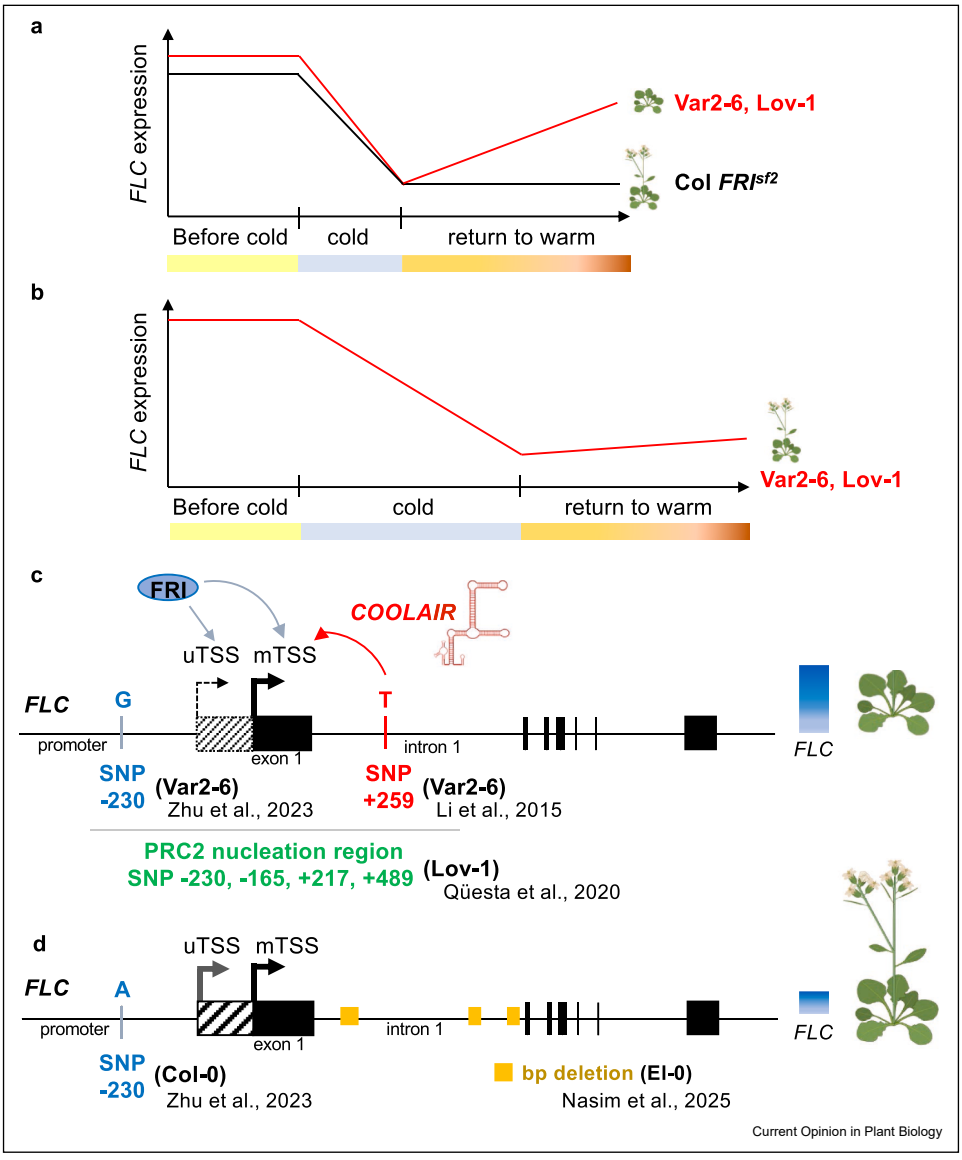
Short cold exposure in autumn/winter leads to the transcriptional repression of *FLC*, a process involving the rapid activation of a set of cold-induced antisense long non-coding RNA (lncRNA) transcripts collectively referred to as *COLD INDUCED LONG ANTISENSE INTRAGENIC RNA* (*COOLAIR*) [15–18]. Despite some controversy on its function [19,20], *COOLAIR* is conserved throughout the *Brassicaceae* [21–24]. Several cold-inducible TFs are known to activate the expression of *COOLAIR*, including NTM1-LIKE8 (NTL8), C-repeat/dehydration-responsive element binding factors (CRT/DRE)-binding factors and WRKY63 [20,25,26]. One distal *COOLAIR* isoform promotes the formation of nuclear condensates that sequester FRI away from the *FLC* promoter [17]. *COOLAIR* adopts multiple secondary structures with distinct conformational dynamics, which are influenced by temperature, affecting both *FLC* expression and flowering time [27]. These findings suggest that *COOLAIR* may act as a "local thermosensor", enabling plants to fine-tune *FLC* expression in response to environmental temperature changes.

In parallel, prolonged exposure to cold leads to the gradual epigenetic silencing of *FLC*, which requires the action of the conserved POLYCOMB REPRESSIVE COMPLEX 2 (PRC2) as well as members of PLANT HOMEODOMAIN (PHD) protein subfamily, including VERNALIZATION INSENSITIVE 3 (*VIN3)* [28–30]. Like *COOLAIR*, induction of *VIN3* is also dependent on NTL8 protein, whose concentration increases in response to low temperature and slow growth [18,31]. NTL8 acts as a "cellular thermosensor", slowly accumulating in the cold, leading to the upregulation of *VIN3* [25]. VIN3 is an accessory protein to the core PRC2 complex, essential for the gradual accumulation of the histone repressive mark H3K27me3 at the 5′end of *FLC* locus (nucleation region) during winter cold [28].

While *COOLAIR* takes the lead in the rapid transcriptional shutdown of *FLC* in response to cold, a separate yet interconnected pathway results in the slow cold-induced PRC2-dependent silencing [18]. Thus, one important aspect is what triggers *FLC* epigenetic silencing in response to cooling temperatures. Initially, the lncRNA *COLD ASSISTED INTRONIC NONCODING RNA* (*COLDAIR*), transcribed from *FLC* intronic region, was proposed to assist the recruitment of PRC2 during cold [32]. However, the limited follow-up on *COLDAIR* in recent years, together with the controversy surrounding PRC2 recruitment by lncRNAs in mammals [33] raise questions about the precise function of *COLDAIR* in *FLC* regulation. Instead, two parallel studies demonstrated that binding of the sequence-specific transcriptional repressors of the VIVIPAROUS1/ABI3-LIKE1 (VAL) subfamily (VAL1 and VAL2) to *FLC* chromatin is a necessary step for *FLC* epigenetic silencing during winter [34,35]. A point mutation disrupting the sequence of a SphI/RY cis-regulatory element (CRE) located within *FLC* intronic sequence fully abolishes PRC2 activity at *FLC* chromatin [34], providing the first evidence of an epigenetic process driven by a genetic sequence in plants. Additional studies further demonstrated that transcriptional repressors belonging to different TF families enable the assembly of PRC complexes at multiple genomic loci controlling Arabidopsis development [36]. Remarkably, recent work has shown that the DNA methylation machinery is also guided by TFs to specific genomic sites in plant reproductive tissues [37]. This evidence demonstrates that both histone- and DNA methylation-based epigenetic silencing strategies require genetic determinants for target recognition.

Upon return of warm temperatures in spring, the PHD-PRC2 complex spreads across *FLC* genomic sequence, resulting in the accumulation of H3K27me3 over the entire locus to maintain long-term silencing [29]. *COLD OF WINTER-INDUCED NONCODING RNA FROM THE PROMOTER* (*COLDWRAP*), a third cold-induced lncRNA arising from the *FLC* locus, has been proposed to mediate PRC2 spreading [38], although its precise mechanism remains elusive. Self-reinforcing loops of H3K27me3 readers and writers likely enable the propagation of silenced *FLC* over cell divisions [39]. Taken together, vernalization is a highly complex, multi-step process that engages numerous components across all cellular levels, encompassing seasonal temperature sensing, precise transcriptional downregulation, and stable epigenetic silencing. In this context, *FLC* has served as a unique platform for uncovering how non-coding regulatory sequences and thermosensory lncRNAs mediate epigenetic regulation in plants.

Figure 2



Non-coding polymorphisms modulate *FLC* expression, thereby affecting the vernalization requirement in natural Arabidopsis accessions. (a) Before cold, the two Swedish accessions Var2-6 and Lov-1 exhibit higher levels of *FLC* expression compared to the vernalization-requirement control genotype Col FRI^{sf2}. A 4-week cold treatment is sufficient for the stable epigenetic silencing of *FLC* in FRI^{sf2}, and plants can transition to flower under the inductive conditions of spring. However, Var2-6 and Lov-1 show unstable *FLC* silencing upon return to warm temperatures. Post-cold *FLC* reactivation prevents the floral transition in these accessions. **(b)** Longer cold exposure (e.g. 8-week cold treatment) is required for the stable *FLC* silencing in Var2-6 and Lov-1. *FLC* transcript levels are maintained at low levels enabling the floral transition. **(c)** Natural variation in this quantitative vernalization response is primarily driven by non-coding polymorphisms at the *FLC* locus, representing a well-established case of cis-regulatory control of an adaptive trait in winter annual plants. In the slow vernalizer Var2−6 haplotype, a single noncoding T-to-G mutation in *FLC* intron 1 alters *COOLAIR* splicing, leading to increased *FLC* expression and a longer cold requirement for full vernalization [45]. Moreover, a G at position −230 found in most Arabidopsis accessions reduce uTSS usage, resulting in higher *FLC* expression and late flowering. In contrast, a less common G-to-A substitution increases uTSS usage and reduces *FLC* expression, leading to early flowering. Additionally, SNP+259 further elevates *FLC* expression and delays flowering in Var2-6 [47]. In Lov-1, four non-coding SNPs widespread at the 5′ end of the *FLC* genomic region, overlapping the PRC2 entry site (nucleation region), underlie adaptation to the extreme winters of northern latitudes. The synergistic interaction of the 4 SNPs impairs the long-term maintenance of the H3K27me3 histone mark at *FLC* [46], resulting in strong *FLC* reactivation when the winter season is too short. **(d)** In the rapid-cycling accession El-0, multiple small deletions (1-6 bp) at the first intron of *FLC* result in low *FLC* transcript levels and early flowering phenotype [42]. Several rapid-cycling accessions like Col-0 carry an A variant at position - 230 [47]. TSS, Transcriptional Start Site. uTSS, upstream TSS, mTSS, main TSS. SNP positions are shown as distance to ATG start codon. Graded blue boxes depict *FLC* expression levels.

Non-coding polymorphisms modulate *FLC* expression dynamics

In line with the role of non-coding genetic sequences on *FLC* regulation, vernalization is strongly influenced by non-coding genetic variation (Figure 2a–b), in particular single nucleotide polymorphisms (SNPs) within the promoter and intronic sequences of *FLC* gene. This is not surprising since mutations affecting *FLC* coding sequence as well as strong or null alleles of *FLC* lead to severe flowering phenotypes. Instead, the non-coding SNPs quantitatively fine-tune *FLC* expression levels under both laboratory and natural field conditions, thereby determining the duration and temperature threshold required for vernalization [40–43]. Five major *FLC* haplotypes have been described in the global *A. thaliana* population, each characterized by distinct non-coding polymorphisms that modulate *FLC* transcriptional activity and the efficiency of its epigenetic silencing during cold exposure [44]. While virtually all FLC protein sequences remain intact, non-coding SNPs affect temperature perception at *FLC* through distinct mechanisms that act synergistically [45–47]. Conceptually, this provides a robust strategy for mediating adaptation to diverse climates.

One example of a non-coding polymorphism affecting *FLC* autumnal expression levels is a SNP located at the position −230 upstream of the ATG start codon (at *FLC* promoter), which alters transcriptional start site (TSS) selection [47] (Figure 2c). While the majority of *Brassicaceae FLC*s carry a G in position −230, a rare G to A substitution is only found in rapid-cycling Arabidopsis variants. Although the mechanism remains unclear, the A SNP leads to lower *FLC* mRNA accumulation during autumn, thus shortening the cold duration required for vernalization. The −230 SNP may alter the binding of *FLC* upstream regulators such as FRI [48]. It could also interfere a "ACGCAA" CRE, a core binding site for NAC TFs [49], although it remains to be determined whether NAC TFs mediate *FLC* activation. Besides, given that cold-induced *COOLAIR* splice variants are essential for *FLC* repression [17], it is plausible that natural variation within the *COOLAIR* sequence contributes to differences in *FLC* expression among genotypes. Indeed, *COOLAIR* splicing variants due to the natural intronic +259 SNP in Var2-6 accession correlate with increased *FLC* transcript levels [45]. Experimental alterations in RNA conformation through the introduction of specific mutations on *COOLAIR* sequence affect the association of *COOLAIR* with *FLC* chromatin, sense *FLC* expression levels and flowering time [27]. Very likely, an important proportion of the natural variation in autumnal *FLC* expression levels within the collection of *A. thaliana* accessions may be the result of non-coding SNPs modulating *COOLAIR* structure dynamics. Interestingly, in accessions adapted to the long winters of high latitudes, a G at position −230 enhancing FRI activity at *FLC* coexists with the *COOLAIR* variants generated by SNP+259 [47]. The interplay between these cis- and trans-acting determinants ensures a quantitatively high *FLC* dosage, maintaining repression of flowering until the prolonged winter period has passed.

Other *FLC* polymorphic variants modulate epigenetic activity at the locus. The northern Swedish accession Lov-1 requires prolonged cold for vernalization due to the unstable silencing of its *FLC* allele following weeks of winter cold exposure [46]. This instability has been attributed to a combination of four non-coding SNPs widespread within the 5′end region of *FLC* (from the promoter to the first intron; Figure 2c). Lov-1 SNPs impair the long-term maintenance of the fully spread H3K27me3 mark over *FLC* when plants return to grow at warm temperature during spring [46]. Therefore, another adaptive strategy for Arabidopsis to survive long and extreme winters is to carry an *FLC* allele that by default reactivates its transcription on return to spring conditions. If the winter is long enough, *FLC* mRNA levels become very low, and any spring reactivation would be negligible to prevent the initiation of the floral transition. Although not directly linked to vernalization, the rapid-cycling accession El-0 carries several base pair deletions dispersed along *FLC* intron 1, resulting in low *FLC* expression levels and early flowering phenotype (Figure 2d; [42]). Further analyses will be required to elucidate the exact mechanisms of *FLC* regulation that are either interfered or enhanced by the Lov-1 and El-0 SNPs.

Beyond Arabidopsis, variation in the expression of *FLC* paralogues also contributes to flowering time change in polyploid *Brassica* crops [50]. *Brassica rapa* and *B. oleracea* carry at least four and five *FLC* copies, respectively, and their hybrid *B. napus* has at least nine. In *B. oleracea*, the comparison of two functional alleles of *BoFLC.C2* revealed that both confer a vernalization requirement but display distinct expression dynamics during cold exposure due to sequence variation within non-coding regions of the gene [51]. A combination of transcriptome time series and modelling highlighted that total *FLC* expression dynamics better explain differences in vernalization requirement between cultivars than the expression of individual *FLC* paralogues in *B. napus* [50]. In vitro structural analyses revealed *COOLAIR* structure conservation in different *Brassicaceae* species, despite very low nucleotide sequence identity [23]. Unlike *COOLAIR*, there is no evidence of the conservation of *COLDAIR* and *COLDWRAP* lncRNAs in the Brassica *FLCs* [21]. An intriguing question is how much of this variability in total *FLC* expression in complex Brassica genomes could be explained by different *COOLAIR* structural conformations.

Altogether, these findings show that non-coding *FLC* polymorphisms act through distinct and multilayered regulatory mechanisms - affecting both transcriptional outputs and chromatin-based memory - providing differences in vernalization requirement and efficiency, which have contributed to plant adaptation to distinct climates.

Vernalization variation drives divergence in plant life history strategies

As sessile organisms, plants have evolved sophisticated mechanisms to synchronize their reproductive development with seasonal environmental cues [52]. Flowering plants exhibit two main life-history strategies: annuals complete their life cycle within a single growing season (Figure 1a–b), while perennials live for multiple years and can thus, in most cases, flower multiple times (Figure 1c). Annual plants can be classified as summer annuals, winter annuals, or biennials, depending on whether vernalization is not required, accelerates flowering, or is strictly required for flowering, respectively [53]. These life history strategies are shaped not only by genetic programs of growth and development, but also by environmental cues that can modulate the timing of key transitions [54].

A breakthrough in understanding the link between vernalization and life-history strategy came from comparing *A. thaliana* with its close polycarpic perennial relative *Arabis alpina* [55,56]. Through screening of early-flowering *A. alpina* mutants lacking vernalization requirement, *PERPETUAL FLOWERING 1* (*PEP1*), an orthologue of the *A. thaliana* floral repressor *FLC*, was identified as a key regulator of perennial flowering. *FLC* and *PEP1* are both epigenetically silenced during prolonged cold exposure. However, *PEP1* silencing is transient, and quickly terminates after the end of vernalization, preventing the induction of *FT* [57]. *PEP1* reactivation upon warming allows some meristems to remain vegetative, requiring another period of cold to continue growth in the following year [55]. The parallel behaviours of *PEP1* and Lov-1 *FLC* regarding the instability of epigenetic silencing on return to warm suggest that cis-acting sequence variation may be responsible for *PEP1* seasonal expression patterns. In fact, introducing the *FLC* gene from the closely related annual species *Arabis montbretiana* into the *A. alpina* genetic background leads to stable silencing after vernalization [57,58]. This highlights how cis-regulatory variation contributes to differences in *FLC* regulation between annual and perennial species. Cold-induced *COOLAIR* has also been detected in *PEP1* locus (*AaCOOLAIR*), supporting its role in the regulation of *FLC* in perennial Brassicaceae [22]. Noteworthy, functional disruption of *PEP1* in *A. alpina* does not prevent the plant from behaving as a perennial [55]. *pep1* mutant still exhibits continuous flowering, suggesting that other factors contribute to perenniality.

To identify these additional factors, a very elegant recent study employed two pairs of species from two genera (*Crucihimalaya* and *Erysimum*) within the *Brassicaceae* family with contrasting life-history strategies [59]. The authors specifically took advantage of the natural differences between strongly polycarpic perennials (*Crucihimalaya himalaica* and *Erysimum nevadense*), a weakly polycarpic perennial/biennial (*Crucihimalaya wallichii*), and an annual species (*Erysimum cheiranthoides*). Strikingly, they showed that the conversion of annual or winter annual Brassicaceae plants into polycarpic perennials can be achieved by introducing the genomic sequence of a single MADS-box gene — *FLC* or its orthologs *FLM* and *MAF* - from perennial species. Conversely, knocking out these genes in polycarpic perennials can induce a shift toward an annual life cycle [59]. A high dosage of either *C. himalaica FLC* or *E. nevadense FLC* is adequate to convert the winter-annual *A. thaliana* into a polycarpic perennial flowering plant. Furthermore, H3K27me3 levels increase in *C. himalaica and E. nevadense FLCs* during cold in the heterologous background, and subsequently decrease upon return to warm conditions. These observations reinforce the notion that differences in *FLC* dosage and epigenetic silencing between annual and perennial *FLCs* are caused by regulatory non-coding polymorphisms. It remains to be determined which polymorphisms distinguish the different *FLC* genes in each perennial species, and whether these loci contain distinct CREs or produce different *COOLAIR* variants.

Studies conducted on the perennial species *Arabidopsis halleri* have provided valuable insights into the seasonal dynamics of *FLC* expression under natural field conditions [60]. These investigations revealed that the key steps of the vernalization mechanism - nucleation, spreading, and resetting of the H3K27me3 silencing mark - originally characterized in *A. thaliana* under controlled laboratory settings, also operate in perennial *FLC* loci during natural seasonal cycles [11,24,61]. Moreover, antisense *COOLAIR* transcripts have been detected at the *A. halleri FLC* locus, although their functional relevance remains unexplored [24]. Future efforts to map cis-regulatory variation within the *A. halleri FLC* region will be essential to elucidate the regulatory features controlling its expression dynamics in perennial contexts.

In sum, these findings underscore the importance of non-coding regulation in enabling plant adaptation to

diverse habitats. Subtle changes in the non-coding genomic sequences of a key floral repressor - affecting core processes such as transcriptional activity and the stability of epigenetic silencing - have driven the evolution of diverse life-history strategies within the Brassicaceae. Despite a continued emphasis on protein-coding genes, we would like to highlight the significance of exploring the non-coding genome to fully understand fundamental developmental mechanisms in plants.

Current challenges and future perspectives

Due to global climate change, the low temperatures typically experienced during winter are rising significantly. These milder cold seasons pose a major challenge for the cultivation of various winter crops, including oilseed species that require vernalization. Over evolutionary time, the selection of different combinations of non-coding variants within the *FLC* loci of *Brassicaceae* species has driven adaptations to both mild and harsh winter conditions. In this context, a key question emerges: can we strategically modify non-coding regulatory elements within genomic sequences of *FLC*-like genes to achieve effective and climate-resilient flowering responses?

Detailed mechanistic analyses of Arabidopsis *FLC* have been instrumental in revealing the remarkable extent to which non-coding regulation fine-tunes the activity of a single gene locus. These seminal studies are particularly relevant today, as research on cis-regulatory modules - such as promoters, enhancers, and silencers - is gaining renewed attention for their role as major drivers of genetic innovation during evolution and adaptation across different plant species [62]. Furthermore, as numerous cases of crop domestication have been linked to the selection of non-coding regulatory variants [63], there is increasing interest in manipulating non-coding regulatory sequences for precision genome editing. However, findings from Arabidopsis *FLC* indicate that individual non-coding SNPs rarely produce measurable effects on their own. Instead, synergistic interactions among multiple SNPs spanning relatively large genomic regions appear necessary to modulate gene expression and flowering time effectively [46,47]. Beyond *FLC*, a comparative case study of the duplicated genes *BLADE ON PETIOLE 1* (*BOP1*) and *BOP2* in *A. thaliana* and *Capsella rubella* further demonstrated that the interactions between promoter cis-regulatory regions, rather than the activities of individual cis-regulatory regions, account for the divergence in gene expression patterns and redundancy in each species [64]. Likewise, redundant interactions between CREs fine-tune the expression of the conserved stem cell regulator *CLAVATA 3* (*CLV3*) both in tomato and *A. thaliana* [65]. Together, these examples highlight the complexity of non-coding variation and the challenges it poses for targeted genome editing. Apart from cis-regulatory sequences, it remains to be tested whether mutations disrupting *COOLAIR* structure could serve as mechanisms for adapting flowering responses to warmer seasons, given that this antisense RNA is conserved across Brassicas *FLC* loci. Further studies will be essential to disentangle these multilayered interactions and guide precise genome editing approaches.

The contrast between annual and perennial life histories reflects a balance between rapid reproduction and long-term survival. Annuals, with their single-season life-cycle, can complete reproduction quickly but often rely on high environmental inputs, whereas perennials invest in longevity and resilience, coping with interannual climatic variability yet requiring careful management over multiple seasons. Variation in *FLC* regulation, including non-coding sequence differences, represents a key molecular axis along which *Brassicaceae* species navigate this trade-off: robust and stable *FLC* repression drives rapid flowering in annuals, while flexible or attenuated responses support perennial strategies. Linking these ecological strategies to underlying genetic and epigenetic mechanisms illuminates how life-history diversity and vernalization pathways shape both evolution and ecological adaptation.

Author contributions

M.O. and J.I.Q wrote and edited the article.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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Data availability

No data was used for the research described in the article.

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* of special interest
** of outstanding interest

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