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In Brief

Chromatin gatekeeping at cooler temperature: VIL1 modulates CBF-driven growth in Arabidopsis

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Plant growth and development are greatly influenced by the environment. Temperature stands out as a critical factor that intricately shapes plant growth and productivity. While the molecular basis of plant response to rises in temperature (or heat) (Park et al 2021; Delker et al 2022; Perrella et al 2022) and cold stress, including cold acclimation and vernalization (Shi et al 2018; Ding and Yang 2022), are well established, mechanisms underlying growth retardation at suboptimal temperatures remain obscure. These suboptimal temperatures, around 12 °C for the model plant *Arabidopsis thaliana*, do not cause cellular injury, yet they reliably suppress growth. Whether this slowdown in growth reflects passive metabolic constraints or an actively regulated developmental program has remained an open research question.

In recent work, Junghyun Kim, Rachel E. Jeong, and Sibum Sung (Kim et al 2026) provide compelling evidence that growth retardation at low ambient temperature is not merely a biochemical consequence of cold but a chromatin-encoded regulatory response. Their study identifies VERNALIZATION INSENSITIVE 3 -LIKE 1 (VIL1), a Polycomb-associated protein known for its role in vernalization and histone H3 Lys-27 (H3K27me3) deposition via Polycomb Repressive Complex 2 (PRC2), as the main genetic determinant of restricted growth at 12 °C. Through a combination of transcriptomics, genetics, and chromatin conformation analyses, the authors reveal that VIL1 fine-tunes the expression of the *C-REPEAT BINDING FACTOR* (*CBF*) gene cluster by modulating chromatin accessibility and loop formation, thereby controlling growth under low ambient temperature (Fig. 1a).

The authors first confirmed that Arabidopsis plants grown at 12 °C develop smaller rosettes and accumulate less biomass than those grown at 22 °C (optimal temperature). Screening loss-of-function *vil1* mutants implicated in temperature signaling revealed that *vil1* plants were unusually insensitive to growth reduction at 12 °C. These plants maintained larger rosettes and higher biomass than wild type. This phenotype is specific to low ambient temperature, as *vil1* resembled wild type at 17 °C and 22 °C. Transcriptome profiling provided a molecular explanation for this enhanced growth. In wild-type plants grown at 12 °C, upregulated genes revealed activation of translation machinery as a mechanism to compensate for the slow rate of translation under low temperature. Meanwhile, downregulated genes suggested induction of mechanisms to conserve energy and resources and maintain basic metabolic functions. To decipher the molecular mechanisms underpinning low ambient temperature growth retardation, the team performed hierarchical clustering of transcriptomes from wild-type and *vil1* plants. This revealed that VIL1 fine-tunes the expression of the *CBF* gene cluster, a trio of transcription factors canonically associated with cold acclimation. Surprisingly, all 3 *CBF*s, including *CBF1*, *CBF2*, and *CBF3*, were induced in *vil1* at 12 °C. Even more interestingly, the normally repressive histone marker H3K27me3 accumulated at the *CBF* cluster during low ambient temperature exposure. This counterintuitive increase in the repressive marker at induced genes suggested the VIL1 helps prevent excessive activation to maintain balanced transcriptional response. Using genetic analyses, Kim and colleagues clarified the functional contribution of each *CBF* gene. The *cbf1* mutant exhibited stunted growth at a low ambient temperature of 12 °C, which was phenocopied in the *cbf123* triple mutant, thus establishing CBF1 is the principal growth-promoting factor under low ambient temperature. Overexpression of CBF1 increased biomass at the low ambient temperature of 12 °C, reinforcing its positive role in growth. These findings reveal a previously unrecognized function of CBF1 not just for freezing tolerance but also supporting growth when temperatures are moderately low.

To uncover how low ambient temperature activates the *CBF* genes, the authors examined the chromatin dynamics at the *CBF* locus. They found 2 major changes induced by temperature: (i) nucleosome depletion and chromatin opening and (ii) transient chromatin loop formation (Fig. 1b and c). The chromatin loops connect the *CBF1* promoter and *CBF3* gene body to a central intergenic region enriched in H3K27me3. Loop formation occurred rapidly within 1 hour after the shift to 12 °C and disappeared by 8 h, suggesting a tightly regulated mechanism that enables coordinated *CBF* activation. Given that VIL1 promotes H3K27me3 deposition and maintains chromatin compaction, the authors investigated how VIL1 influences these chromatin dynamics. In *vil1* mutants, chromatin opening occurred prematurely, nucleosome

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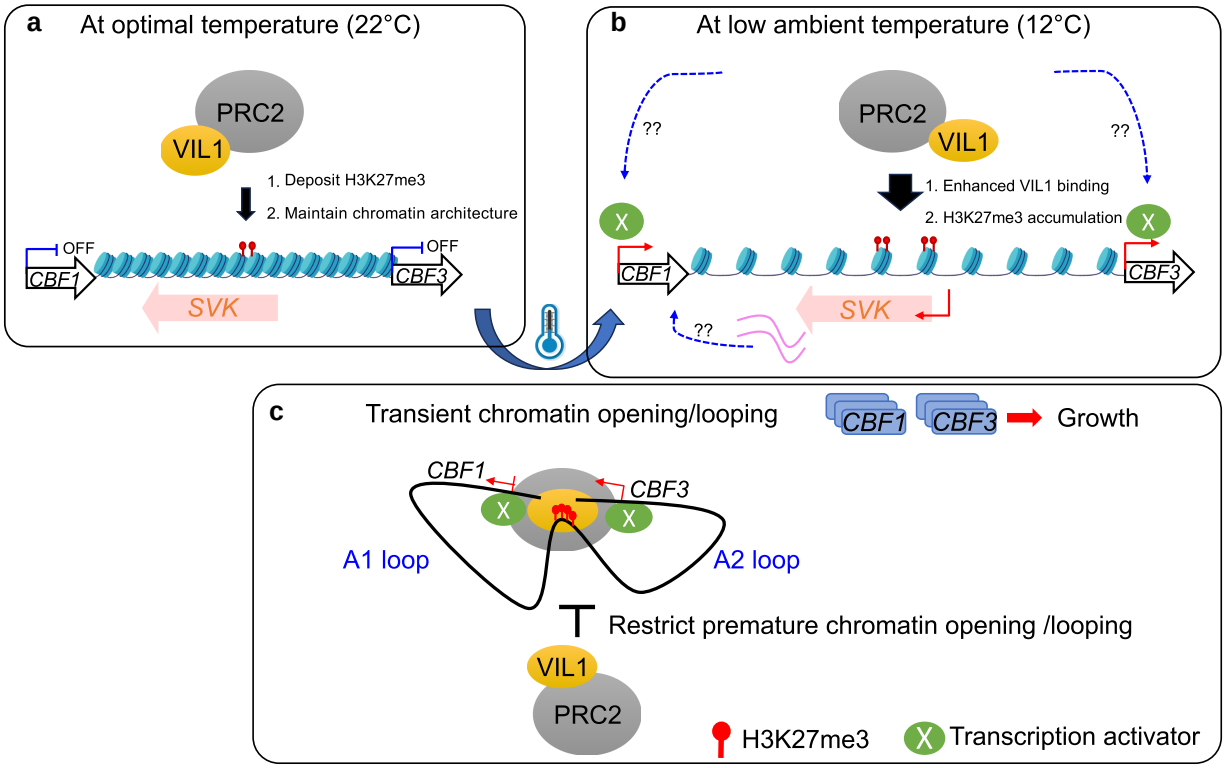

Figure 1 **a)** VIL1 associates with Polycomb Repressive Complex 2, and deposits histone H3 Lys-27 (H3K27me3) between CBF clusters. VIL1 restricts chromatin opening and prevents premature loop formation. **b)** Low temperature transiently opens chromatin, allowing CBF induction to support growth, while VIL1 fine-tunes the amplitude and duration of this response. **c)** Two short-range chromatin loops (A1 and A2) form between CBF cluster facilitating efficient transcription, and in VIL1 mutants they persist longer after exposure. (a and b) Loss of VIL1 causes exaggerated and prolonged CBF activation regardless of presence of SVK transcripts. (Adapted from Kim et al. (2026) Fig. 5D).

occupancy was reduced, even before the temperature shift, and chromatin loops formed earlier and persisted for longer. This persistent open chromatin state likely underlies the hyper-induction of *CBF* genes in *vil1*. These findings demonstrate that VIL1 prevents untimely chromatin opening and ensures proper timing of *CBF* induction, thereby modulating growth under low ambient temperature.

In summary, this study established VIL1 as a key regulator of growth retardation under low ambient temperature in *A. thaliana*. VIL1 operates by fine-tuning the transcription response at the *CBF* gene cluster by promoting H3K27me3 deposition, maintaining nucleosome occupancy, preventing premature chromatin opening, and ensuring timely formation and resolution of chromatin loops. Loss of *VIL1* disrupts this balance, leading to persistent chromatin accessibility, hyper-induction of *CBF* genes, and enhanced biomass accumulation. Genetic analyses highlight *CBF1* as the central growth-promoting factor, with partial redundancy. Overall, this study reveals that growth retardation at low ambient temperature is not merely a metabolic slowdown but a regulated developmental program encoded in chromatin architecture. By integrating histone modification, chromatin topology, and transcriptional networks, plants finely adjust growth to fluctuating environmental conditions. These insights advance our understanding of temperature adaptation and might inform strategies to improve crop performance in cooler climate or early-season planting.

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- Li et al. (2026). Integrated data on chromatin accessibility, histone modifications, and transcriptomics dynamics from Populus shoot meristems accross five key stages of the annual growth dormancy cycle. Overall, the study showed that deposition of repressive histone marker H3K27me3 by the Polycomb Repressive Complex 2 plays a vital role in regulating growth dormancy transitions.

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Conflicts of interest

None declared.

Data availability

No new data were generated or analyzed in support of this research.

References

Delker C, Quint M, Wigge PA. 2022. Recent advances in understanding thermomorphogenesis signaling. Curr Opin Plant Biol. 68:102231. https://doi.org/10.1016/j.pbi.2022.102231.

Ding Y, Yang S. 2022. Surviving and thriving: how plants perceive and respond to temperature stress. Dev Cell. 57:947–958. https://doi.org/10.1016/j.devcel.2022.03.010.

Kim J, Jeong RE, Sung S. 2026. Chromatin remodeling at the C-REPEAT BINDING FACTOR cluster controls growth retardation at low ambient temperature. Plant Cell. 38:koag007. https://doi.org/10.1093/plcell/koag007.

Li Y et al. Epigenetic reprogramming drives the annual growth-dormancy cycle in Populus. Plant Cell. 2026:koag018. https://doi.org/10.1093/plcell/koag018

Park YJ, Kim JY, Lee JH, Han SH, Park CM. 2021. External and internal reshaping of plant thermomorphogenesis. Trends Plant Sci. 26:810–821. https://doi.org/10.1016/j.tplants.2021.01.002.

Perrella G, Bäurle I, van Zanten M. 2022. Epigenetic regulation of thermomorphogenesis and heat stress tolerance. New Phytol. 234:1144–1160. https://doi.org/10.1111/nph.17970.

Shi Y, Ding Y, Yang S. 2018. Molecular regulation of CBF signaling in cold acclimation. Trends Plant Sci. 23:623–637. https://doi.org/10.1016/j.tplants.2018.04.002.